Primary Health Care Booklet

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PRIMARY HEALTH CARE BOOKLET

UPPER RESPIRATORY TRACT INFECTIONS

كتابة: عبدالله الوهيبي ، يوسف المانع / مراجعة وتعديل: يوسف المانع

Pharyngitis (Sore throat)

Causes:

1. Viral (most common >50%)
    a. Rhinovirus, coronavirus and parainfluenza virus (causes of the common cold) are the most common (25%)
    b. Other viral causes are: Influenza virus, adenovirus, herpes simplex virus type 1, EBV (causes infectious mononucleosis, causes splenomegaly) and HSV.
2. Bacterial
    a. The most important and most common cause of bacterial pharyngitis is group A beta haemolytic streptococcus (GABHS) (5-20% in adults, 15-30% in children).
    b. There are other rare causes e.g. haemophilus influenzae, Neisseria gonorrhoeae and others. .

Rhinovirus, coronavirus and parainfluenza virus (causes of the common cold) are the most common cause of viral pharyngitis (25%)

Due to the possibility of rheumatic fever, group A β-hemolytic streptococcus is the main concern when diagnosing for sore throat

Clinical Features (not always present!)

1. Painful swallowing
2. Fever (more common in bacterial)
3. Chills (more common in bacterial)
4. Cough
5. Nausea, vomiting and abdominal pain
6. Headache
7. Erythema and edema of the pharynx, uvula and tonsils, with hypertrophy of the tonsils.
8. Gray-white membrane or exudates (more common in bacterial)
9. Petechiae on soft palate (in bacterial)
10. Scarlatiniform rash (in bacterial)
11. Swollen and tender anterior cervical lymph nodes (more common in bacterial

Differential diagnosis

1. Peritonsillar abscess (quinsy).
2. Kawasaki disease (children younger than 5).
3. Epiglottitis (if suspected do not examine throat, examination may lead to airway obstruction).
4. Diphteria (adherent gray membrane over tonsils and pharynx)
5. Non infectious causes (GERD, acute thyroiditis, chronic cough, post nasal drip, irritation by tobacco smoke, alcohol or NG tube)
6. Tumor

Diagnosis

1. Diagnostic tests are not used routinely.
2. Rapid antigen detection test (RADT): detect the presence of GABHS antigen in throat swab in 10-15 minutes, >90% specificity and 59%-95% sensitivity (higher in newer kits).
3. Throat culture: though it's more accurate, yet negative culture does not rule out the diagnosis of streptococcal infection

Management:

1. Reassure the patient that the illness is self limiting and rarely causes complications, educate patients about viral nature of most infections, and how antibiotics are not indicated.
2. Encourage adequate fluid intake to avoid dehydration.
3. Paracetamol or ibuprofen to relieve symptoms.
4. Majority of sore throats are caused by viruses hence they are self-limiting. Antibiotics do not significantly relieve symptoms or reduce duration of illness in most patients and though complications of streptococcal infection are rare antibiotics are proved to have little benefit in preventing them:
    a. Use Centor criteria to assess the need of antibiotics
        - Centor criteria is clinically based 4 points criteria:
            - Absence of cough
            - History of fever
            - Exudates on tonsils or pharynx
            - Tender anterior cervical lymphadenopathy.
        - Interpretation:
            - (0-1) do not prescribe.
            - (2) Consider RAST or culture.
            - (3-4) immediate prescription
    b. Antibiotics recommended (Penicillin, if allergic use Macrolides) however Ampicillin and Amoxicillin are not recommended as they cause rash in infectious mononucleosis.
5. Safety netting (if patient do not require immediate prescription, use safety netting i.e. "if your symptom persist for >3 days, or it get worse, visit for antibiotic prescription")

Group A β-hemolytic streptococcus (GABHS) infection complications

1. Suppurative complications: parapharyngeal, retropharyngeal and peritonsillar abscess, sinusitis, cervical lymphadenitis, otitis media, and mastoiditis.
2. Non suppurative complications: Acute rheumatic fever and Poststreptococcal glomerulonephritis

Rhinosinusitis

Definition

1. Acute rhinosinusitis (ARS): < 4 weeks.
2. Subacute rhinosinusitis: 4 to 12 weeks.
3. Chronic rhinosinusitis: > 12 weeks.
4. Recurrent acute rhinosinusitis: four or more episodes of ARS per year.

Acute rhinosinusitis

Causes:

Table .2/Viral VS. Bacterial rhinosinusitis:

	Viral	Bacterial
Nasal discharge	Bilateral, clear	Unilateral predominance, purulent
Pain	Bilateral	Unilateral predominance
Fever	<38 c° (low grade fever)	>38c° (high grade fever)
Course	Mild symptoms	Acute deterioration After mild symptomc
Period	Resolves in 7 to 10 days	May persist beyond 7 days
Organisms	1.rhinovirus 2.influenza virus 3.parainfluenza virus	1.Strept. pneumoniae 2.Haemophilus influenzae.

Clinical features

1. Nasal congestion and obstruction
2. Purulent nasal discharge
3. Sinus pain (mostly maxillary and could mimic pain of dental caries)
4. Headache or facial pain that worsens with percussion or bending head down
5. Hyposmia or anosmia (loss of smell)
6. Fever
7. Cough
8. Ear pressure or fullness (mostly in children, due to Eustachian tube blokage)

Diagnosis

The diagnosis of acute rhinosinusitis is based on clinical signs and symptoms thus physical examination and diagnostic testing (culture, endoscopy and radiology) have limited role in the diagnosis unless signs suggesting complicated disease appeared.

Differential diagnosis

- Common cold
- Allergic rhinitis
- Vasomotor rhinitis
- Tension headache, migraine and cluster headache

Management

1. **Indications for urgent admission**
   A. Signs of sever systemic infection.
   B. Signs of orbital involvement: Displaced globe, diplopia, ophthalmoplegia, or reduced visual acuity (orbital cellulitis)
   C. Signs of intracranial involvement: Severe frontal headache, frontal swelling, altered mental status, symptoms or signs of meningitis, or focal neurological signs (Intracranial involvement).
2. **Measurements of treatment**
   A. Reassurance: reassure the patient that the illness is self limiting, taking 2.5 weeks on average, educate patients about viral nature of most infections, and how antibiotics are usually **not indicated!**, encourage adequate fluid intake to avoid dehydration.
   B. Symptomatic relief
      a. Relieve pain and headache with warm packs and/or giving (Paracetamol or ibuprofen)
      b. Treat nasal congestion with: normal saline irrigation or/and intranasal decongestant (Pseudoephedrine or Oxymetazoline) and emphasize on limited usage <7days.
      c. For prolonged or severe symptoms consider prescribing an intranasal corticosteroid.
      d. Avoid prescribing antihistamines unless there is co-existing allergic rhinitis.
   C. Antibiotics: if symptoms suggestive of bacterial cause (see above)
      a. 1st line: amoxicillin, if allergic doxycycline or macrolides (Clarythromycin and erythromycin)
      b. 2nd line: amoxicillin/clavulinic acid, if allergic azithromycin
   D. Safety netting.

Treatments that are not recommended include:

1. Steam inhalation
2. Oral corticosteroids
3. Antihistaminesamines
4. Complementary and alternative medicine (as the benefits have not been proven).

Safety netting: if patient do not require immediate prescription, advise him/her to visit for antibiotic prescription if symptom persist beyond 3 days, or it get worse.

Box .1

Rhinitis medicamentosa (RM)
This condition is due to the prolonged use of nasal decongestants leading to medication tolerance, whereas the use of nasal decongestants become of a very little effect on the patient making him feels the need to increase the dose to get the desired effect.

Allergic rhinitis

1. Allergic rhinitis is associated with the development of allergic conjunctivitis, eczema, and asthma (atopy).
2. 10-30% of adults, and up to 40% in children worldwide.
3. Classification: e.g. (intermittent moderate-severe allergic rhinitis) or (intermittent mild …)
4. Intermittent (<4 days per week or <4 weeks), persistent (>4 days per week or >4 weeks)
5. Severity of symptoms (mild, moderate-severe) by its affect to quality of life

Common allergens:

House dust mite, pollens, pets' hair or occupational (like: latex gloves, flour, and wood dust).

Clinical features

1. Sneezing
2. Itching (pathognomonic)
3. Watery rhinorrhea
4. Nasal obstruction
5. Associated symptoms (fatigue, poor sleep, snoring, headache, and symptoms of atopy)
6. Edematous and pale nasal mucosa on anterior rhinoscopy.

Differential diagnosis

1. Infective Rhinitis (acute onset, cough, fever, lymphadenopathy, and purulent discharge)
2. Irritant rhinitis (with exercise, humidity, temperature change, or chemical exposure)
3. Other non-allergic causes of rhinitis:
    a. Vasomotor rhinitis
    b. Hormonal rhinitis

c. Drug induces rhinitis (ACEi, Beta blockers, Aspirin chlorpromazine, NSAIDs, and cocaine) or rhinitis medicamentosa (caused by topical decongestants).

Diagnosis is made clinically if:

1. Infective and irritant rhinitis are unlikely (see above).
2. Personal or family history of atopy.
3. Associated with exposure to known allergen.
4. Nasal itching.
5. If unclear Refer to immunologist for skin prick test and send blood for serum total and specific IgE, if it is negative consider other non-allergic causes.

Management

1. Allergens avoidance.
2. Pharmacotherapy is complicated but there is some important facts
    a. Antihistamines are used for occasional symptoms "as required".
    b. Intranasal corticosteroids are used as maintenance therapy regardless the symptoms; use maximal dose then taper to the lowest effective dose
    c. Starting on intranasal corticosteroid is preferable, intranasal antihistamine may be added.
    d. Antihistamines are more effective on itching and sneezing, not much on congestion.
    e. Oral antihistamine are preferable over intranasal when conjunctivitis is present.
    f. Take patient preferences into consideration, oral or intranasal, corticosteroids or not.
    g. Other measurements for refractive cases are oral corticosteroid and montelukast

Otitis Media

Types

1. Acute otitis media (AOM)
2. Otitis media with effusion (OME)
3. Chronic suppurative otitis media
4. Adhesive otitis media

Investigations

1. Otoscope

2. Tympanometry
3. Pure Tone Aoudiometry

Risk factors

1. Passive tobacco smoke
2. Group daycare attendance
3. Seasonality
4. Supine bottle feeding

Supine bottle feeding results in a drainage of some of the feeding contents into the Eustachian tube reaching the middle ear and causing infection

Table .1/ Symptoms of different types of Otitis Media:

	AOM	OME	CSOM
	1.Otalgia,	1.Otalgia,	1. Otorhhea,
	2.Otorrhea,	2.hearing loss,	2. hearing loss,
	3.Fever,	3.tinnitus,	3. tinnitus
	4.vomiting,	4.vertigo	4. Otorhhea,

Acute otitis media (AOM)

Management

1. Treat pain and fever
2. No Abx (or delayed Abx if it persists >4days)
3. < 3 months children could need immediate Abx, admission or referral to a specialist
4. Referral or admission ?

Otitis media with effusion (OME)

Management

1. It is a self-limiting illness and 90% of children will have complete resolution within 1 year, so reassure the parents.
2. There is no proven benefit from treatment with any medications or any complementary or alternative therapies.
3. For acute cases, a period of active observation over 6–12 weeks is appropriate, as spontaneous resolution is common (during this period, do not prescribe antibiotics, steroids, antihistamines, decongestants, or mucolytics).
4. If signs and symptoms persist, refer the child for a hearing test or/and to the specialist.
5. Children with Down's syndrome or cleft palate who are suspected to have OME, require immediate referral for specialist assessment.

Chronic suppurative otitis media

Management

1. Admit people with signs of infection beyond the ear, (e.g. postauricular swelling or tenderness, headache, facial paralysis, or vertigo).
2. Refer to ENT specialist for the treatment and follow up.

PRIMARY HEALTH CARE BOOKLET

Osteoporosis & Vitamin D Deficiency

كتابة: أحمد الغامدي ، طارق الفريان / مراجعة وتعديل: يوسف المانع

Osteoporosis

Definition

Osteoporosis is a progressive systemic skeletal disease characterized by low Trabecular (spongy) bone mass and micro-architectural deterioration of skeletal tissue, despite the normal mineralization.

Prevalence

1. **Worldwide:** According to a study done in 2006, it's been estimated that over 200 million people worldwide have osteoporosis, and the number is yet increasing. [1]
2. **Saudi Arabia:** A study was done in 2012 found that approximately 36.6% of the female ranged from 50 to 79 were osteopenic and 34.0% were osteoporotic. In three other studies on males, the prevalence of osteopenia was 46.3% and osteoporosis 30.7%. [2]

Risk factors

1. **Modifiable:**
   A. Decrease physical activity.
   B. Hyperthyroidism.
   C. Vitamin D deficiency.
   D. Smoking.
   E. Alcohol abuse.
   F. Glucocorticoid use.
   G. Calcium deficiency.
   H. Low BMI.
   I. Prolonged immobility.
2. **Non-modifiable:**
   A. Age.
   B. Female gender.
   C. Family history.
   D. Post menopausal state.

Symptoms

It is asymptomatic disease so the patient can present with the Complications or we can detect it early by screening.

Complications

1- Fracture.
2- Loss of height over time.

3- Deformity.

Screening

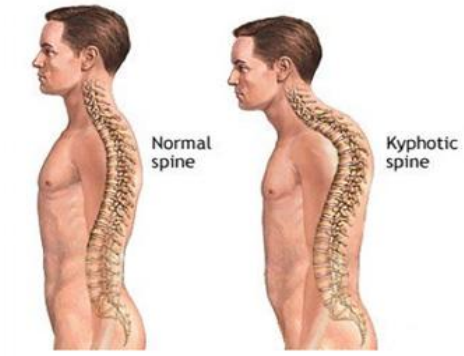
Screening is by dual-energy x-ray absorptiometry (DXA).

Diagnosis

Osteoporosis is a silent disease, the disease is diagnosed by screening methods (will be explained) or the patient may come late to the clinic with a vertebral fracture, at that time when you do screening for the patient, you may find that the patient has osteoporosis, and vertebral fracture is the result of it.

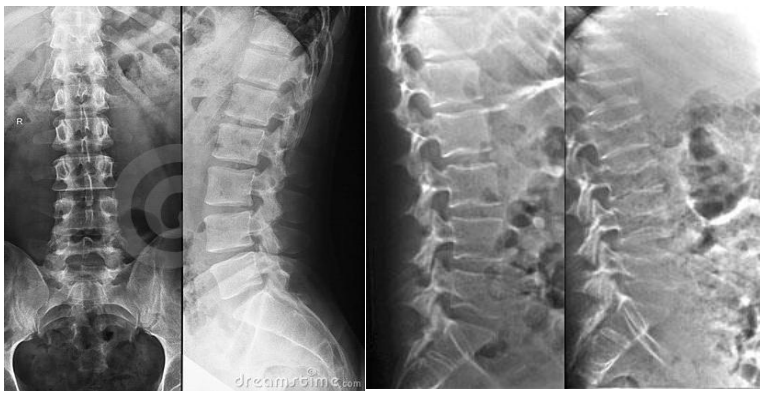
Physical Examination:

We have 4 main steps for examination: look, feel, move and special test, and it depends on the patient presentation, If it's a fracture then there is decrease in the range of motion, tenderness , swelling and sometimes deformities.



Investigations

1. X Ray



1 **2**

1. Normal Spine X Ray. 2. Two views of the lumbar spine taken 1 year apart demonstrate rapidly developing osteoporosis and multiple compression fractures in this patient on exogenous steroids.

2. Dual energy x-ray absorptiometry (DXA)

Population eligible for screening:

1. Women aged 65 years and older.
2. Women under 65 years whose 10-year fracture risk is greater than or equal to that of a 65-year-old women without additional risk factors based on the FRAX tool (9.3).
3. Women and men of any age who had suffered a low impact fracture.
4. Women and men of any age who are at increased risk as a result of selected medical conditions or treatment with specific medications.

Table .1/ Interpretation of bone density test results:

T score	Interpretation
≥ (-1)	Normal
Between (-1) and (-2.5)	Osteopenia (low bone density).
≤ (-2.5)	Osteoporosis

3. Biochemistry:

a) Complete blood count (CPC)
b) Serum chemistry levels (Calcium, Phosphate and Alkaline phosphatase), their levels will be normal in case of osteoporosis.

Management

The goals of osteoporosis treatment are to:

A. Prevent bone fractures with medicines that strengthen bone.
B. Slow down or stop bone loss.
C. Minimize the risk of falls that might cause fractures.

1. lifestyle modification

General practitioners should recommend the following important lifestyle choices for all postmenopausal women and older men:

- adequate but safe exposure to sunlight as a source of vitamin D
- maintenance of a healthy weight and BMI
- cessation of smoking
- avoidance of excessive alcohol consumption.

2. Non Pharmacological Intervention:

a) **Exercise**
   1)High intensity strength training.
   2)Low impact weight bearing exercise.
b) **Calcium supplementation (Table.2)**
c) **Vitamin D supplementation (Table.3)**

Table .2/ Recommended Calcium Intake Vs. Age:

Age	Recommended Calcium Intake
1. Men age 50-70	1000 mg per day
2. Women age 51 or older & Men age 71 or older	1200 mg per day

Table .3/ Recommended Vit.D (IU) Vs. Age:

Age	Recommended Calcium Intake
1. 50 or older	800-1000 IU per day*
2. 51- 70 year old	600 IU per day
3. 71 or older	800 IU per day

* Many patients, including those with malabsorption, will need more than the recommended 800-1,000 IU per day.

Box .1

Calcium-rich Food
Food like: milk, plain yogurt, cottage cheese, cheddar cheese, vanilla ice cream, orange juice.

3. Pharmacological Treatment

Who Should Be Considered for Treatment?

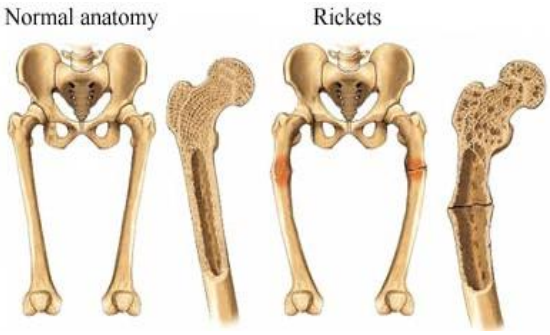
Postmenopausal women and men age 50 and older presenting with the following should be considered for treatment:

1)A hip or vertebral fracture.
2)T-score ≤ 2.5 at the femoral neck, total hip or lumbar spine.
3)Low bone mass (T-score between -1.0 and -2.5 at the femoral neck or lumbar spine).

1. **Bisphosphonate (Alendronate Sodium)**
   Is used for prevention (5 mg daily), for treatment (10 mg daily), it reduces the incidence of spine and hip fractures by about 50 percent over three years in patients with a prior vertebral fracture, also reduces the incidence of vertebral fractures by about 48 percent over three years in patients without a prior vertebral fracture.
2. **Strontium ranelate**
   It reduces the risk of both spine and non-vertebral fractures.

Rickets and Osteomalacia

Bone disease characterized by bone demineralization due to deficiency or impaired metabolism of vitamin D or phosphates, called rickets in children, osteoporosis in adult.



*Highlights on vitamin D deficiency (*According to NICE guidelines*)*

Vitamin D is essential for skeletal growth and bone health. Dietary sources are limited. The major natural source of vitamin D is from skin synthesis following exposure to sunlight. Severe vitamin D deficiency can result in rickets and osteomalacia. It has also been associated with some diseases and long-term conditions, such as osteoporosis, diabetes and some cancers. Vitamin D deficiency can occur at any age but is more likely during periods of rapid growth (for example, during childhood), during pregnancy and while breastfeeding. A newborn baby's vitamin D status is largely determined by the mother's level of vitamin D.

Prevalence

1. **United state:** A cross sectional study was done in University of Pennsylvania (N=4495) in 2011 estimated that the overall prevalence rate of vitamin D deficiency is 41.6% [1]
2. **Saudi Arabia:** A cross sectional study was done in King Abdul-Aziz University (N=834 male aged 20-74 years living in Jeddah area) in 2012 and the prevalence of vitamin D deficiency was 87.8% were [2]

Risk factors

1. **Modifiable**
   A. Less sun expose
   B. Low vitamin D supplements.
   C. Obesity
2. **Non-Modifiable**
   A. Age

B. Female gender
C. Malabsorption
D. Non-white race
E. Antiepileptic therapy
F. Burns

Symptoms

1. **Osteomalacia:**
   a) Muscle weakness.
   b) Bone pain
   c) Fracture.
2. **Rickets:**
   a) Muscle weakness.
   b) Bone pain.
   c) Fracture.
   d) Skeletal deformity.
   e) Poor growth.

Diagnosis

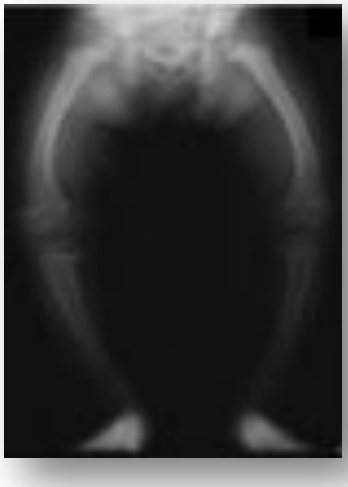
1. Physical Examination

Rickets Signs:

A. Pigeon chest deformity.
B. Rickety rosary.
C. Craniotabes.
D. Genu varium or genu valgum.

2. Investigations

A. X-Ray



B. Biochemistry (Table.4)

Treatment

1. For people age 1-18 suggest to treat with 2000IU/d for at least 6 weeks, followed by maintenance therapy of 600-1,000 IU/day.
2. Suggest that all adults who are vitamin D deficient be treated with 50,000 IU of vitamin D once a week for eight weeks, followed by maintenance therapy of 1,500-2,000 IU/day.
3. In obese patients, patients with malabsorption syndromes, and patients on medications affecting vitamin D metabolism, we suggest a higher dose (two to three times higher; at least 6,000-10,000 IU/day) of vitamin D.

Take home message

1. Osteoporosis is a silent disease and cannot be diagnosed clinically
2. Osteopenia differs from osteoporosis, in which osteopenia is an early stage of osteoporosis.
3. Patients with osteoporosis are diagnosed mainly either if they have a fracture (late) or based on DXA if they meet the criteria for screening.
4. The most common risk factor for developing
5. Osteomalacia is lack of sun exposure.
6. Nowadays rickets is not that common as before, because of availability of vitamin D in most of the child food.

Table .4/ Biochemistry findings in Osteomalacia/Rickets:

	Level in the blood
Vitamin D	Low
Phosphate	Low
Calcium	Low
PTH	High
Alkaline Phosphatase	High

PRIMARY HEALTH CARE BOOKLET

Hypertension

كتابة: سامر الباشا ، عبدالعزيز الزهراني ، رائد الموسى / **مراجعة وتعديل**: عبدالعزيز الزهراني

Hypertension

Epidemiology in Saudi Arabia

1. Developed and developing countries are alike; Essential Hypertension affects 25-35% of the adult population.
2. The prevalence of hypertension among attendants of primary health care centers in Al-Qassim region Saudi Arabia was 30%.
3. It was more among obese patients than non-obese.

Risk factors

1. Smoking.
2. Dyslipidemia.
3. Diabetes Mellitus.
4. Obesity.
5. Age older than 60 years.
6. Sex (men or postmenopausal women).
7. Family history of cardiovascular disease.

Diagnosis of hypertension

Two or more elevated readings are obtained on at least two visits over a period of one to several weeks. If the clinic blood pressure is 140/90 mmHg or higher, offer ambulatory blood pressure monitoring (ABPM) to confirm the diagnosis of hypertension.

When using the following to confirm diagnosis, ensure:

1. **ABPM**: At least two measurements per hour during the person's usual waking hours, average of at least 14 measurements to confirm diagnosis.
2. **HBPM:** Two consecutive seated measurements, at least 1 minute apart.

Blood pressure is recorded twice a day for at least 4 days and preferably for a week. Measurements on the first day are discarded average value of all remaining is used.

In the Framingham Heart Study:

1. Those below Age of 55 diastolic BP are the strongest predictor of cardiovascular risk.
2. Above 55 years, diastolic BP was negatively related to the risk of coronary events, so the pulse pressure became superior predictor to the systolic Bp.

What happens to blood pressure with aging?

1-) Systolic pressure increases with age.

2-) Diastolic pressure increases with age but peaks between 55 and 60 years then starts to decrease.

3-) Arterial stiffness: cause of elevated systolic and lower diastolic pressure with aging.

Category	Systolic blood pressure (mmHg)	Diastolic blood pressure (mmHg)
Optimal blood pressure	<120	<80
Normal blood pressure	<130	<85
Pre-hypertension	130-139	85-89
Grade 1 hypertension (mild)	140-159	90-99
Grade 2 hypertension (moderate)	160-179	100-109
Grade 3 hypertension (severe)	≥ 180	≥ 110
Isolated systolic hypertension Grade 1	140-159	<90
Isolated systolic hypertension Grade 2	160-179	<90
Isolated systolic hypertension Grade 3	≥180	<90

Box .1

White-Coat Hypertension: the risk of cardiovascular disease is lower in patients with white-coat hypertension.

Raised clinic blood pressure in the presence of a normal daytime ambulatory blood pressure.

Stages of hypertention (Update in NICE 2011)

1. **Stage 1 hypertension:** Clinic blood pressure (BP) is 140/90 mmHg or higher and ABPM or HBPM average is 135/85 mmHg or higher.
2. **Stage 2 hypertension:** Clinic blood pressure (BP) is 160/100 mmHg or higher and ABPM or HBPM daytime average is 150/95 mmHg or higher.
3. **Severe hypertension:** Clinic BP is 180 mmHg or higher or Clinic diastolic BP is 110 mmHg or higher.

Why we have to control hypertension:

1. Increased blood pressure is the leading risk for death and disability globally according to the Global Burden of Disease.
2. Hypertension is a major risk factor for CHD and CVA.
3. Increase blood pressure is symptomless until it causes organ damage.
4. The benefits of lowering blood pressure are as follow: reduction in STROKE by (35 – 40 %), MI by (20 – 25 %), and HEART FAILURE by > 50%

Each increment of 20 mm Hg in systolic blood pressure or 10 mm Hg in diastolic blood pressure doubles the risk of cardiovascular disease events independent of other factors.

Measures of prevention:

Lifestyle modifications to prevent and manage hypertension	Approximate SBP Reduction
Weight reduction Maintain normal body weight (body mass index 18.5–24.9 kg/m2).	5–20 mmHg/10kg
Adopt DASH eating plan Consume a diet rich in fruits, vegetables, and low fat dairy products with a reduced content of saturated and total fat.	8–14 mmHg
Dietary sodium reduction Reduce dietary sodium intake to no more than 100 mmol per day (2.4 g sodium or 6 g sodium chloride).	2–8 mmHg
Physical activity Engage in regular aerobic physical activity such as brisk walking (at least 30 min per day, most days of the week).	4–9 mmHg

DASH, Dietary Approaches to Stop Hypertension; SBP, systolic blood pressure
For overall cardiovascular risk reduction, stop smoking.

How to approach a patient with Hypertension

Medical History, Physical Examination, Routine Laboratory Tests, Optional Tests, Non-Pharmacological Treatment, Drug Treatment.

Smaller size cuff lead to over estimation of BP.

Larger size cuff lead to under estimation of BP.

Patient Evaluation:

Evaluation of patients with documented HTN has three objectives:

1. Assess lifestyle and identify other CV risk factors or concomitant disorders that affects prognosis and guides treatment.
2. Reveal identifiable causes of high BP.
3. Assess the presence or absence of Target Organ Damage and CVD.

MEDICAL HISTORY:

- Patient History of Cardiovascular Disease
- Current and Previous Medications
- Smoking
- Lifestyle Factors
- Family History

PHYSICAL EXAMINATION:

- Blood Pressure (2 Readings)
- Height, Weight and Pulse
- Exam. Of Neck, Heart, Lungs, Abdomen and Extremities
- Funduscopic Examination (Arterial narrowing "copper wiring", A-V nipping, Flame shaped hemorrhages, Soft

Investigations:

Routine

1. 1.CBC
2. Urine Analysis and Microalbuminuria
3. Urea , Creatinine, Electrolytes, Uric Acid and Calcium
4. Fasting Plasma Glucose
5. Lipid Profile (T.ch, Trig, LDL and HDL)
6. ECG
7. Chest X-ray?

Optional

1. 24-hour Urinary Protein
2. Creatinine Clearance
3. Echocardiography
4. Ultrasonography
5. Thyroid Stimulating Hormone
6. 24-hour Urinary Vanyl Mandelic Acid
7. 24-hour Urinary Catechleamines
8. 24-hour Urinary Free Hydrocortisol

Target Organ Damage:

- **Heart:** Left ventricular hypertrophy, Angina or prior myocardial infarction, Heart failure
- **Brain:** Stroke or transient ischemic attack
- Chronic kidney disease
- Peripheral arterial disease
- Retinopathy

Management of HTN:

What is the goal of management of hypertension?

1. Treating (Non-Diabetic) SBP and DBP to targets that are < 140 / 90 is associated with decrease in CVD Complications.
2. < 140/80 mmHg for people with diabetes.
3. For diabetic patients with proteinuria systolic blood pressure < 130 mmHg.
4. Limited data suggest possible worsening of both renal and CVD outcomes if systolic blood pressure is lowered to < 110 mmHg.

Non-pharmacological: Offer to all hypertensive and those with family history of increased BP

For pre hypertensive patients give life style modification. No need for drugs.

1. See table above "Measures of prevention"
2. Offer smoking cessation.
3. Don't offer Ca2+, Mg2+, or K+ supplements as a method to reduce BP.

Pharmacological:

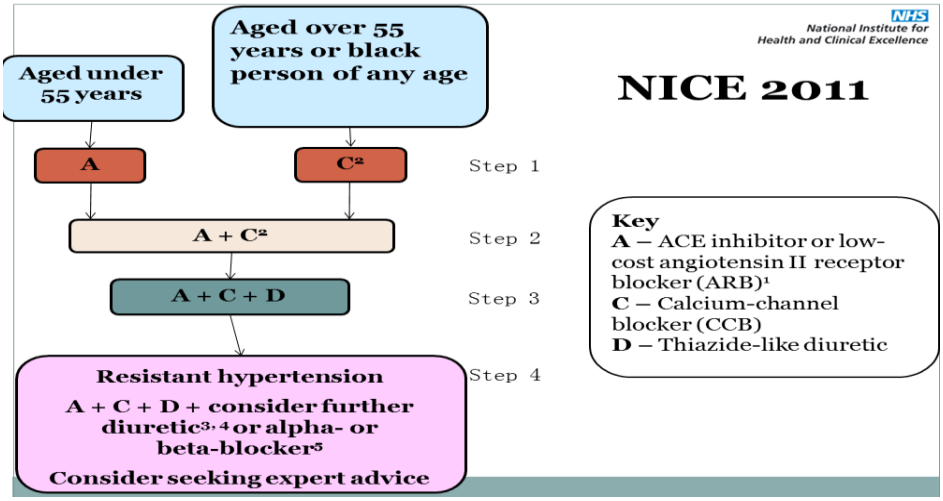
1. Offer antihypertensive drug treatment to people aged under 80 years with stage 1 hypertension who have one or more of the following:
    a. Target organ damage
    b. Established cardiovascular disease
    c. Renal disease
    d. Diabetes
    e. A 10-year cardiovascular risk equivalent to 20% or greater.
2. Offer antihypertensive drug treatment to people of any age with stage 2 hypertension.

	Drug name
β BLOCKERS	Atenolol, Bisoprolol, Carvedilol
ACE Inhibitors	Captopril, Lisinopril, and Enalapril
Angiotensin II Receptor Blocker	Losartan, Candesartan, Valsartan, Irbesartan
Ca+ Blockers (Long Acting**)**	Nifedipine Retard, Amlodipine, Felodipine
Diuretics	Thiazides, Indapamide SR
Vasodilators	Hydralazine (for gestational hypertension)

Box .2

Angiotensin-receptor blocker:

ARB therapy may cut the risk of Alzheimer's disease (AD) by reducing amyloid deposition in the brain. (*Archives of Neurology*, September 13, 2012)
890 hypertensive patients with available brain autopsy data.
The risk for AD was 24% lower in those prescribed ACE inhibitor.



More explanation for the above figure:

1. In hypertensive patients aged 55 or older or black patients of any age: The first choice for initial therapy should be either a calcium-channel blocker or a Thiazide-type diuretic. If a third drug is needed an ACE inhibitor or ARB is a choice.
2. In hypertensive patients younger than 55, the first choice for initial therapy should be: An ACE inhibitor (or an ARB if an

ACE inhibitor is not tolerated). Adding an ACE inhibitor to a calcium-channel blocker or a diuretic (or vice versa are logical combinations).

3. Beta-blockers may be considered in younger people, particularly: Those with an intolerance or contraindication to ACE inhibitors and ARB or Childbearing potential or People with evidence of increased sympathetic drive.
4. If therapy is initiated with a beta-blocker and a second drug is required, add a calcium-channel blocker rather than a Thiazide-type diuretic to reduce the patient's risk of developing Diabetes.

Beta-blockers and HTN:

B blockers are associated with an increased risk for new-onset DM by 22%.

They are no longer recommended as a first-line drug.

1. There is a paucity of data or an absence of evidence to support the use of beta-blockers as Monotherapy or as First-line agents in uncomplicated HTN.
2. There is strong evidence to use it in post MI patient or heart failure.

Exception: B-blockers may be considered as first line treatment for

a. Younger women of child-bearing potential or
b. Patients with hypertension and evidence of increase sympathetic drive or
c. Patients intolerant of/with contraindication to ACE inhibitors/ARBs.

DIURETICS:

Meta-analysis of all RCTs supports diuretics as first line agent.

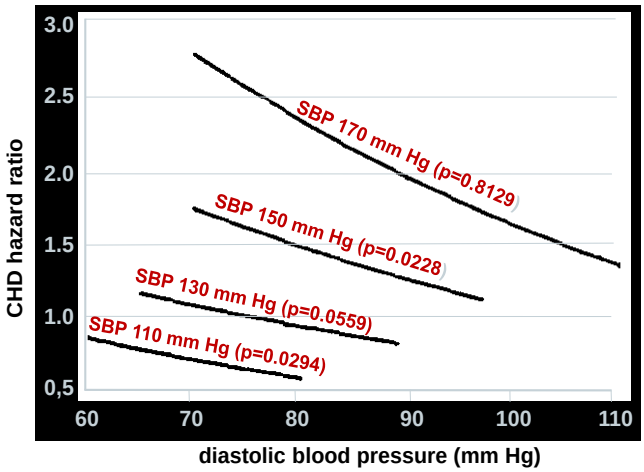
B.P. and DIABETES MELLITUS:

1. Diabetic patients with BP > 140/80 are candidate for antihypertensive treatment.
2. Patients should be checked to confirm the presence of hypertension.

3. Behavioral Approach / Lifestyle Modification
4. Drug Treatment:
    a. ACE Inhibitors
    b. Angiotensin II Receptor blockers
5. In Micro-albuminuria and Nephropathy (Renal damage) lower BP to ≤ 130/80

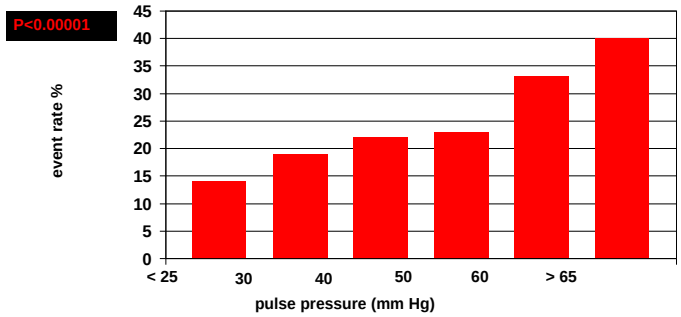
The following 2 figures were explained by the Doctor himself after the seminar:

Pulse Pressure and Coronary Risk



Franklin, S.S. et al., Circulation 1999;

Pulse Pressure and Total Mortality



Mitchell, G.F. & Pfeffer, M.A., Curr Opin Cardiol 1999; 14: 361-9

PRIMARY HEALTH CARE BOOKLET

Headache

كتابة: محمد الريشان ، إبراهيم المزروع / **مراجعة وتعديل**: عبدالله الوهيبي

Headache

Epidemiology

1. Globally, it has been estimated that prevalence among adults of current headache disorder is 47%. (WHO)
2. Half to three quarters of the adults aged 18–65 years in the world have had headache in the last year and among those individuals, more than 10% have reported migraine.
3. Despite regional variations, headache disorders are a worldwide problem, affecting people of all ages, races, income levels and geographical areas.
4. Saudi Arabia: Al Jumah M (2013) found that among 2,421 respondents, the prevalence of all headaches was 63%, of migraine 32%, of tension type headache 27% and of medication-overuse headache 2.7%.

How to approach patients with headache

1. History:
    a. History of headache (analyzing pain)
    b. Previous or recurrent headaches, the previous diagnosis (if any) and whether the current headache is similar or different.
    c. For recurrent headaches, age at onset, frequency of episodes, temporal pattern and response to treatments.
    d. Assess risk factors for headache, including exposure to drugs, substances (particularly caffeine), and toxins.
    e. Review of systems should seek symptoms suggesting a cause.
    f. Past medical history

2. Physical examination:

    a. Vital signs.
    b. General appearance.
    c. General examination, with a focus on the head and neck, Palpate (skull base, TMJs, temporal arteries, upper cervical facets, pericranial muscles, paranasal sinuses).
    d. Full neurological examination.

3. Investigation

    Most patients with headache are diagnosed without investigations. However, when suspecting serious causes we consider further investigations (Box.1).

Box .1

CT or MRI should be done in patients with any of the following findings:

- Severe, sudden-onset headache (thunderclap headache)
- Altered mental status
- Meningism
- Papilledema
- Signs of sepsis (rash, shock)
- Acute focal neurologic deficit
- Severe hypertension (systolic blood pressure > 220 mm Hg or diastolic pressure > 120 mm Hg on consecutive readings).

Thunderclap headache suggest subarachnoid hemorrhage

Classification

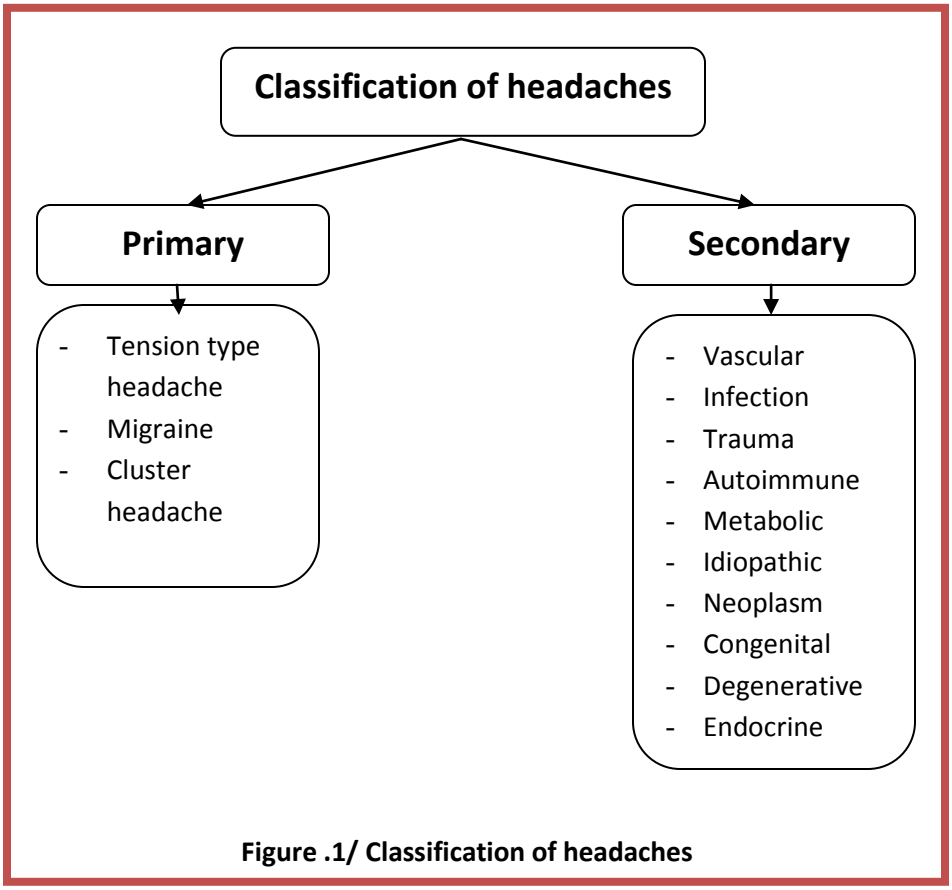


Figure .1/ Classification of headaches

Mnemonic: VITAMIN CDE

Primary Headaches

1. **Tension-type headache (TTH)**
    a. It is the most common type of headaches among adults and adolescents
    b. Causing mild to moderate pain and come and go over a prolonged period of time.
    c. The most intense pressure at the temples or over the eyebrows.
    d. The pain occurs sporadically (infrequently and without a pattern) but can occur frequently and even daily in some people.
    e. The pain allows most people to function normally.
    f. Unknown cause; could be caused by skull muscles contractions.
    g. Related to stress, depression, anxiety, head injury, or holding your head and neck in an abnormal position.
    h. Management
        i. Aspirin, paracetamol or NSAIDs.
        ii. If recurrent, amitriptyline and acupuncture are used for prophylaxis.
        iii. In chronic headache, consider relaxation training.

There is no aura in TTH

Table .1/ Diagnosis of tension-type headache :

	Tension Type Headache	
Pain location	Bilateral	
Pain quality	Pressing/Tightening (not pulsating)	
Pain Intensity	Mild to moderate	
Effect on activities	Not aggravated by daily activities	
Other symptoms or signs	None	
Duration of headache	30 minutes – continuous	
Frequency of headache	<15 days per month	> 15 days per month
Diagnosis	Episodic tension- type headache	Chronic tension-type headache

2. **Migraine**

The exact cause of migraine is not known, but there are some triggers like: Stress, Menstruation, Visual stimuli, weather changes, Nitrates, Fasting, Alcohol, sleep disturbances, and aspartame.

A. *Presentation of migraine (figure.2)*

Prodrome	Aura	Headache	Postdrome
1-2 days before headache - Euphoria - Depression - Irritability - food cravings - Constipation - neck stiffness - increased yawning	Can occur during or before headache - Visual aura: vision loss, and seeing shapes and flashes - Sensory aura: tingling then numbness. - Dysphasia or aphasia	Throbbing, unilateral moderate to severe headache lasting 4-72 hours, associated with photophobia, phonophobia, nausea, and/or vomiting.	Exhaustion or sometimes slight euphoria

Figure .2/ Stages of Migraine

Not all patients experience all stages

B. *Risk Factors:*

a. Family history: Up to 90 percent of people with migraines have a family history.
b. Age: can begin at any age, but most people experience their first migraine during adolescence.
c. Sex: Women are three times more likely to have migraines.
d. Hormonal changes: before or after menstrual cycle.

C. *Diagnosis (table.2)*

D. *Management*

a. Acute management:
   i. Oral triptan (serotonin receptor agonist)
   ii. NSAIDs
   iii. Paracetamol
   iv. Combination of triptans with NSAIDs or paracetamol
   v. Nasal triptan (in young)

Do not offer opioids or ergots

vi. Antiemitic (even in the absence of nausea/vomiting)

b. Prophylaxis
   i. B-blockers (propranolol)
   ii. Anticonvulsants (topiramate)
   iii. Others (Ca-channel blockers, valproate, antidepressants, gabapentin, and others)

Topiramate is teratogenic and reduce contraception effectiveness.

Table .2/ Diagnosis of Migraine :

	Migraine	
Pain location	Unilateral or bilateral	
Pain quality	Pulsating	
Pain Intensity	Moderate to severe	
Effect on activities	Aggravated by, or causes avoidance of daily activities	
Other symptoms or signs	Photophobia, phonophobia, nausea, and/or vomiting. Aura	
Duration of headache	4-72 hours in adults 1-72 hours in people aged 12-17	
Frequency of headache	<15 days per month	> 15 days per month for more than 3 months
Diagnosis	Episodic migraine (with or without aura)	Chronic migraine (with or without aura)

3. Cluster headache

1. Cluster headaches are so named because they tend to occur daily for periods of a week or more with long periods of time, months to years, with no headache symptoms. They occur at the same time of day, often waking the patient in the middle of the night.
2. The cause is unknown, but it tend to runs in families, so there is a role of genetics, it is triggered by sleep disturbance, and some medications (nitroglycerine).
3. Diagnosis (table.3)

Cluster headache is rare, unlike TTH and migraine

4. Management

1. Acute treatment: Oxygen and/or a subcutaneous or nasal triptan.
2. Prophylaxis: Verapamil (Ca-Channel blocker).

Table .3/ Diagnosis of cluster headache :

	Migraine	
Pain location	Unilateral (around the eye)	
Pain quality	Variable (can be sharp, burning, dull, thropping, or tightening)	
Pain Intensity	Severe to very severe	
Effect on activities	Causes restlessness and agitation	
Other symptoms or signs	Ipsilateral red watery eye, nasal congestion, swollen eyelid, sweating, miosis, and ptosis.	
Duration of headache	15-180 minutes	
Frequency of headache	1 every other day to 8 per day, with remission >1 month	1 every other day to 8 per day, with continuous remission < 1 month in a 12 months period
Diagnosis	Episodic cluster headache	Chronic cluster headache

Examples of Secondary Headaches

1. **Idiopathic intracranial hypertension**
    a. Increased intracranial pressure in the absence of a tumor or other diseases.
    b. Most cases occur in young women who are obese. Patients with higher BMIs and recent weight gain are at increased risk.
    c. Symptoms
        i. Diffuse headache, worse in the morning.
        ii. Aggravating by coughing and sneezing.
        iii. Horizontal diplopia
        iv. Pulsatile tinnitus
    d. Management
        i. Lumbar puncture
        ii. Patients without visual loss: carbonic anhydrase inhibitor (eg, acetazolamide).
        iii. Patients with severe symptoms, early visual field loss high-dose corticosteroids.
        iv. diuretics
        v. If the medication is not useful refer to neurosurgeon.
2. **Temporal arteritis (Giant cell arteritis)**
    a. It's a form of vasculitis that affects medium and large arteries especially external carotid artery and its branches.
    b. Risk Factors
        i. Age: >50
        ii. Sex: Women
        iii. Polymyalgia rheumatica

c. **Symptoms**
   i. Sudden onset headache, localized to the temporal region.
   ii. Tenderness and sensitivity on the scalp.
   iii. Jaw claudication
   iv. Unilateral visual loss or occasionally diplopia.
   v. Constitutional symptoms.

d. **Investigations:** arterial biopsy and ESR.
e. **Diagnosis:** (at least 3 out of 5 criteria must be present Box.2):
f. **Management:** high dose corticosteroids as soon as possible to prevent blindness. Refer to ophthalmologist and rheumatologist.

Box .2

Criteria for diagnosing temporal arteritis
• Age of onset >50 • New-onset headache or localized head pain • Temporal artery tenderness to palpation or reduced pulsation • ESR > 50 mm/h • Abnormal arterial biopsy

3. Space occupying lesions

a. Usually due to malignancy but it can be caused by other pathology such as an abscess, cysts and hematoma.
b. Headache tends to be quite a late feature, and it is usually very severe, waking patients from sleep.
c. **Presentation**
   i. Papilledema.
   ii. Vomiting.
   iii. Focal neurological symptoms, or non-focal neurological symptoms
d. **Investigations:** CRP, ESR and brain CT or MRI.
e. **Management:** (refer to specialist)

Lumbar puncture is contraindicated when suspecting space occupying lesion

Red Flags (Box.3)

Box .3

Red flags (**SNOOP**)
2. **S**ystemic symptoms (weight loss, fever) or **S**econdary risk factor (HIV infection or Cancer) 3. **N**eurological symptoms 4. **O**nset (abrupt) 5. **O**lder age of onset 6. **P**revious history of headache (if first headache or different from usual headache in terms of severity, frequency and feature)

PRIMARY HEALTH CARE BOOKLET

Changes in bowel movement

كتابة: باسل الصقير **/ مراجعة وتعديل**: عبدالله الشعلان ، حسام المراد

Change in bowel movement

- **Normal bowel movement**

Definition:

Body wastes passed through the rectum and anus.

Normally: consist of brown stools, not too hard, not too loose

Frequency of bowel movements

There is no rule for frequency of bowel movements, but the general range is from 3 times a day to 3 times a week. "each person has his own pattern"

- **Abnormal bowel movement (*Constipation* & *Diarrhea*)**

1. ***Constipation***
   a. **Definition:**
      i. Infrequent bowel movements (less than three times per week)
      ii. g defecation (straining and a sensation of hard stools that are difficult or painful to pass)
   b. **Causes:**
      i. Insufficient dietary fiber intake or a diet high in fats.
      ii. Inadequate fluid intake or dehydration.
      iii. Decreased physical activity.
      iv. Side effects of medications :
         1. Pain medications (especially narcotics)
         2. Blood pressure medications (calcium channel blockers).
   c. **Alarm symptoms (Red flags)**

      Especially in patients over the age of 50:
      1. Anemia.
      2. Weight loss.
      3. Rectal bleeding.
      4. Positive occult blood test.
      5. Recommended test: **colonoscopy.**
   d. **Management:**
      i. Treat the cause (Hypothyroidism, drug side effect)
      ii. Lifestyle and diet changes (Patient Education):
         1. Increase intake of water and fibers in diet
         2. Exercise is recommended for better overall health
      iii. Consider drugs only if above measures fail (laxatives)
   e. **When to refer to specialist?**
      i. Constipation which last for three weeks or more.
      ii. Never been constipated before, especially 50 years and above.
      iii. Severe abdominal pain.
      iv. Noticed blood in stool.
      v. Unintentional weight loss.

2. *Diarrhea*

a. **Definition:** frequent bowel movements (three times or more per day) loose or watery stools
   i. **Osmotic diarrhea:** means that something in the bowel is drawing water from the body into the bowel, such as excessive sugar or excessive salt. (ex: in malabsorption)
   ii. **Secretory diarrhea:** occurs when the body is releasing water into the bowel when it's not supposed to or if there is an inhibition of absorption. (ex: bacterial toxin: cholera & E. coli)
   iii. **Exudative diarrhea:** refers to the presence of blood and pus in the stool. This occurs with inflammatory bowel diseases

b. **Alarming symptoms (Red flags):**
   i. Rectal bleeding.
   ii. Nocturnal or progressive abdominal pain.
   iii. Weight loss.
   iv. Laboratory abnormalities such as anemia, elevated inflammatory markers, or electrolyte disturbances.

	Acute Diarrhea	Chronic Diarrhea
Duration	Less than 14 days	More than 4 weeks
Causes:	**Infections** Bacterial: Campylobacter Viral: Rotavirus Parasites	Irritable bowel syndrome, Inflammatory bowel disease
	Allergies to certain food	Mal-absorption syndromes
	Reaction to certain medications like antibiotics.	Chronic infections

Irritable bowel syndrome:

(the main & most important topic of this lecture !)

Definition:

It is a gastrointestinal syndrome characterized by chronic abdominal pain and altered bowel habits in the absence of any organic causes; it is the most commonly diagnosed gastrointestinal condition, and the point prevalence worldwide is 10-20%.

Etiology:

The causes of IBS are not well understood, a combination of physical and mental health problems can predispose to IBS and the possible causes of IBS include the following:

a. Brain-gut signal problems (thalamic activity).
b. Mental health problems (such as anxiety and depression).
c. Gastrointestinal infections.

Clinical Manifestations:

1. Chronic abdominal pain or discomfort.
2. Altered bowel habits (diarrhea and constipation).
3. Other gastrointestinal symptoms (as Nausea).
4. Abdominal bloating or distention.

Diagnostic Approach:

1. (NICE guidelines)

Consider assessment for IBS if the person reports having had any of the following symptoms for at least 6 months:

1. **A**bdominal pain or discomfort
2. **B**loating
3. **C**hange in bowel habit.

2. Rome III diagnostic criteria: *(Important)*

Recurrent abdominal pain or discomfort for at least 3 days per month in the last 3 months, plus 2 or more of the following:

1. Improvement by defecation.
2. Onset associated with change in stool frequency.
3. Onset associated with change in form (appearance) of the stool.

Symptoms Support The Diagnosis.

Altered stool passage (straining, incomplete evacuation "tenesmus")

Symptoms made worse by eating

Passage of mucus.

Diagnostic tests:

In people who meet the IBS diagnostic criteria, the following tests should be undertaken **to exclude other diagnoses**:

1. Complete blood count (CBC)
2. erythrocyte sedimentation rate (ESR)
3. c-reactive protein (CRP)
4. antibody testing for coeliac disease (endomysial antibodies [EMA] or tissue transglutaminase [TTG]).

Table .2/ Red flags: (should be referred to secondary care for further investigation if any are present)

	History	Clinically
	Unintentional and unexplained weight loss	Anemia
	Rectal bleeding	Abdominal masses
	Rectal masses	Rectal masses
		Markers for IBD.

Management:

The main goal of treatment is to decrease the severity of the symptoms and improve quality of life.

1. **Non pharmacological**
    a. **Patient education:**
        i. People with IBS should be given information about the importance of lifestyle and dietary modifications in effectively managing their IBS.
        ii. Healthcare professionals should encourage people with IBS to create relaxation time and to avoid stressful situations.
    b. Physical activity: Give people with low activity levels brief advice and counseling to increase their activity.
    c. Dietary modification:
        i. Have regular meals and take time to eat. Avoid missing meals or leaving long gaps between eating.
        ii. Drink at least eight cups of fluid per day, especially water.
        iii. Reduce intake of alcohol and soft drinks.
        iv. Possible precipitating substances, such as caffeine, lactose, or fructose, may need to be eliminated from the diet. Symptom monitoring can be helpful to identify precipitating substances and factors.
2. **First-line pharmacological treatment**

    Choose single or combination medication based on the predominant symptom(s):

    a. Consider offering antispasmodic agents (Mebeverine).
    b. Consider offering laxatives for constipation.
    c. Offer loperamide as the first choice of antimotility agent for diarrhea.
3. **Second-line pharmacological treatment**

    Tricyclic antidepressant (TCAs) and Selective serotonin re-uptake inhibitors (SSRIs)

4. **Psychological Interventions**

    Should be considered for people with IBS who do not respond to pharmacological treatments after 12 months:

    a. Cognitive behavioral therapy
    b. Hypnotherapy

Follow-up:

Follow-up should be agreed based on the response of the patient's symptoms to interventions. The emergence of any 'red flag' symptoms

during management and follow-up should prompt further investigation and/or referral to secondary care.

Referral to secondary care: in case of red flag, when the symptoms persist and if the diagnosis is uncertain.

Prognosis:

Patients with IBS have an excellent prognosis in the sense that they have a normal life expectancy, and there are no long-term complications of their disease.

PRIMARY HEALTH CARE BOOKLET

Urinary tract infections

كتابة: أحمد نقشبندي ، فهد الموسى ، يزيد العيد / مراجعة وتعديل: سعود الرميح

Approach to patient with UTI

Definition

1. A urinary tract infection (is an infection in any part of your urinary system, most infections involve the bladder and the urethra.
2. Urinary tract infections (UTIs) include:
    a. Cystitis: Infection of the bladder (Lower UTI).
    b. Pyelonephritis: infection of the kidney (Upper UTI).
    c. Urethritis: infection of the urethra.

Classification

Symptoms	Symptomatic	Asymptomatic
Recurrence	Sporadic (≤ 1 UTI/ 6m)	Recurrent (> 1 UTI/ 6m)
Complicating factors	Uncomplicated	Complicated

1. Uncomplicated UTI: Infection in a structurally and neurologically normal urinary tract.
2. Complicated UTI: Infection in a urinary tract with functional or structural abnormalities.

Acute cystitis: refers to infection of the bladder (lower urinary tract); it can occur alone or in conjunction with pyelonephritis (infection of the kidney – the upper urinary tract).

1. It is less common in men; this is due to longer urethral length and antibacterial substances in prostatic fluid.
2. However, it may indicate underlying diseases such as prostatitis or malignancy in the prostate or the bladder.
3. Women tend to get them more often because their urethra is shorter and closer to the anus than in men.

Acute pyelonephritis: is a potentially organ- and/or life-threatening infection that often leads to renal scarring.

1. Bacteria usually reach the kidney by ascending from the lower urinary tract.
2. Bacteria may also reach the kidney via the bloodstream.
3. Timely diagnosis and management of acute pyelonephritis has a significant impact on patient outcomes.

A complicated urinary tract infection, whether localized to the lower or upper tract, is associated with an underlying condition that increases the risk of failing therapy, including the following:

a. Diabetes
b. Pregnancy
c. Symptoms for seven or more days before seeking care
d. Hospital acquired infection
e. Renal failure
f. Urinary tract obstruction
g. Recent urinary tract instrumentation
h. Functional or anatomic abnormality of the urinary tract
i. History of urinary tract infection in childhood
j. Renal transplantation
k. Immunosuppression
l. Infection with an uropathogen with broad-spectrum antimicrobial resistance is also considered complicated.

Causes

1. Urinary tract infections typically occur when bacteria enter the urinary tract through the urethra and begin to multiply in the bladder.
2. Although the urinary system is designed to keep out such microscopic invaders, these defenses sometimes fail.
3. When that happens, bacteria may take hold and grow into a full-blown infection in the urinary tract.

Risk factors

In men:

1. Obstruction from any cause is a major risk factor for the development of UTI, such as catheterization.
2. In males older than 50 years, prostatic hypertrophy with partial obstruction is the main contributor to the increase in UTI.
3. Homosexual behavior with anal intercourse
4. Intercourse with a female infected or colonized with a uropathogen
5. Lack of circumcision
6. Human immunodeficiency virus (HIV) infection
7. Prostatitis, epididymitis, orchitis

In women:

1. Most episodes of cystitis and pyelonephritis are generally considered to be uncomplicated in otherwise healthy nonpregnant adult women.
2. Short urethra
3. Sexually active women

4. Postmenopausal
5. Spermicide use

Common Organisms:

Table .1/ Common Urinalysis Dipstick Findings:

The most common organism causes UTI is E.coli

Bacteria	%Unomplicated	%Complicated
Gram negative		
Escherichia coli	***70-95***	***21-54***
Proteus mirabilis	***1-2***	***1-10***
Klebsiella spp	***1-2***	***2-17***
Citrobacter spp	<1	5
Enterobacter spp	<1	2-10
Pseudomonas aeruginosa	<1	2-19
Gram positive		
Coagulase-negative staphylococci	5-10	1-4
Enterococci	1-2	1-23
Group B streptococci	<1	1-4

Clinical feature

A UTI can present with a range of symptoms, or may be totally asymptomatic and diagnosed only on routine dip testing.

1. **Bladder (cystitis)**
   a. Dysuria
   b. Frequency and urgency
   c. Suprapubic pain
   d. Hematuria
   e. Sensation of incomplete voiding
2. **Kidneys (pyelonephritis)**
   a. Upper back and side (flank) pain
   b. High fever
   c. Shaking and chills
   d. Nausea and vomiting
   e. Fatigue and night sweating

Constitutional symptoms usually comes with pyelonephritis

f. Hematuria

Diagnosis of Urinary Tract Infection:

1. In straightforward cases, a diagnosis may be made and treatment given based on symptoms alone without further laboratory confirmation.
2. In complicated or questionable cases, it may be useful to confirm the diagnosis via midstream urine (MSU) sample. A midstream urine sample can be used for urinalysis (dipstick, microscopic) and urine culture.

Investigations

1. **Dipstick Urinalysis:** Leukocyte esterase, nitrates, protein, and blood are the important features in evaluating for UTI.

Table .1/ Common Urinalysis Dipstick Findings:

Finding	Significance
Color	Typically pale yellow to colorless
Clarity	Typically clear
Odor	Mild characteristic odor
Specific gravity (SG)	Dilute urine = SG ≤ 1.008 Concentrated urine = SG > 1.020
Leukocyte esterase (LE)	Test for enzyme present in white blood cell (WBC)
Nitrites	Surrogate marker for bacteriuria. Presence indicates bacterial reduction of dietary nitrates to nitrites by select Gram-negative uropathogens including *Escherichia coli, Proteus* spp. Normally absent in sterile urine and infection caused by enterococci, staphylococci.
Protein	Dipstick testing most sensitive for albumin
PH	Average pH = 5-6 Acid pH = 4.5-5.5 Alkaline pH = 6.5-8
Red blood cells (RBCs)	Low number of RBCs noted. Gross hematuria may occur in uncomplicated UTI but may be present in infection complicated by nephrolithiasis

2. **Microscopy Urinalysis**
    a. Look for the presence of red blood cells, white blood cells, or bacteria.
    b. When coupled with classic symptoms, a finding of **2-5 WBCs or ≥ 15 bacteria per hpf** in a centrifuged urine sediment is consistent with UTI. The presence of many epithelial cells usually indicates a contaminated specimen.
3. **Urine culture**
    a. Urine culture is important when diagnosis is not clear or UTI is recurrent.
    b. The presence of more than one organism may indicate a contaminated urine specimen and collection and testing should be repeated.
    c. The presence of **≥ 10^5 CFU/mL** of bacteria is the traditional diagnostic indicator for UTI. However, in the presence of dysuria and other symptoms for UTI, **10^2 CFU/mL** confirms the diagnosis.

When to start full investigations like U/S, CT scan, IVP, (Females and Males)?

1. Imaging and urologic intervention should be considered in the following patients:
    a. Patients with a history of kidney stones.
    b. Patients with diabetes.
    c. Patients with polycystic kidneys are prone to abscess formation.
    d. Patients with tuberculosis Have persistently not responded to treatment.
    e. Patients have a history of renal tract disease or anomaly.
    f. Patients have haematuria.
    g. Patients are women with more than three confirmed infections in the preceding year (two confirmed infections in the case of men) with no known contributing comorbidity
2. Imaging in the emergency department is typically not necessary unless concomitant obstructive uropathy is suspected, as this is an emergent condition that requires prompt intervention.
3. Modalities for this include ultrasonography, intravenous pyelography (IVP), contrasted computed tomography (CT) scanning, or helical CT scanning of the urinary system (currently preferred by most experts)

When to refer to specialist?

1. **Referral for assessment should be considered for men who have:**
    a. Symptoms of upper urinary tract infection (pyelonephritis).
    b. Failure to respond to appropriate antibiotic therapy.

c. Frequent episodes of urinary tract infection (UTI) - this is stated as two or more episodes in a 3-month period.
d. Features of urinary obstruction (e.g. in older men, enlarged prostate).
e. History of pyelonephritis, calculi, or previous genitourinary tract surgery.
f. Any age with painless macroscopic haematuria:
g. Recurrent or persistent UTI associated with haematuria, in a male aged 40 years or older.
h. Unexplained microscopic haematuria, in a male aged 50 years or older.
i. With an abdominal mass identified clinically or on imaging that is thought to arise from the urinary tract.

2. Specialist referral is recommended for investigation of women with:

a. Risk factors for complicated UTI.
b. Surgical correction of a cause of UTI.
c. When the diagnosis of recurrent uncomplicated UTI is uncertain.

Management

1. Cystitis in women:

•**Uncomplicated:** 1st is Trimethoprim-sulfamethoxazole then Nitrofurantoin.

If they were not available or resistant, use fluoroquinolone (ciprofloxacin).

Duration: 3- 5 days

•**Complicated:** orally fluoroquinolone (ciprofloxacin)

Duration: 5- 14 day

If the patient not tolerate orally or resistant organism start Parenteral therapy with levofloxacin (500 mg) or ceftriaxone (1 g) once daily until the patient improve then transition to oral antibiotic therapy.

2. Cystitis in men:

a. Uncomplicated:
1) 1st is Trimethoprim-sulfamethoxazole
2) If it was not available or resistant use fluoroquinolone (ciprofloxacin).
3) Duration: 7 - 14 days

b. Complicated:

1) orally fluoroquinolone (ciprofloxacin)
2) Duration: 5- 14 day
3) If the patient not tolerate orally or resistant organism start Parenteral therapy with levofloxacin (500 mg) or ceftriaxone (1 g) once daily until the patient improve then transition to oral antibiotic therapy.

3. **Pyelonephritis in both men and women:**
   a. **•Mild to moderate (uncomplicated):** treat as Outpatient with: fluoroquinolone (ciprofloxacin) for 7 days.
   b. **•Sever or with risk factor (complicated):** treat as Inpatient with: Intravenous therapy such a long acting parenteral as (ceftriaxone 1g) until patient improve then transition to oral antibiotic therapy.
4. **Prophylaxis of UTI:**
   a. The patient considered for prophylaxis if:
      1) infected ≥ 2 infections in 6 months.
      2) infected ≥ 3 infections in one year.
   b. Antimicrobial prophylaxis:

Table .2/ Continues VS. postcoital prophylaxis:

	Continues prophylaxis	postcoital prophylaxis
	Continuous prophylaxis decreases recurrences by up to 95% .	More efficient and acceptable prevention than continuous
	1. Trimethoprim 2. sulfamethoxazole	In women with UTIs temporally related to sexual intercourse and pregnant. Single postcoital dose of 1. cephalexin (250 mg) 2. nitrofurantoin (50 mg)

5. **Education:**
   a. Drink plenty of water (six to eight glasses) every day.
   b. Do not resist the urgent urination, Bacteria can grow when urine stays in the bladder too long.
   c. Women should wipe from front to back to prevent bacteria from entering the vagina or urethra.
   d. Cleanse the genital area before and after sexual intercourse.
   e. Urinate shortly after sex. This can flush away bacteria that might have entered the urethra during sex.
   f. Avoid using feminine hygiene sprays and scented douches, which may irritate the urethra.

g. For women, using a diaphragm or spermicide for birth control can lead to UTIs by increasing bacteria growth.

PRIMARY HEALTH CARE BOOKLET

DIABETES MELLITUS

كتابة: عقيل المهدلي ، أحمد الذكير / مراجعة وتعديل: عبدالعزيز الزهراني

Diabetes diagnostic criteria

Diabetes Mellitus

Epidemiology

3. **World wide:**
   a. 382 million people have diabetes in 2013; by 2035 this will rise to 592 million (5% of the world)
   b. Diabetes deaths occur in low and middle-income countries.
   c. WHO projects that diabetes will be the 7th leading cause of death in 2030.
4. **Saudi Arabia:**
   a. Around 20% of the population by now (2.8 M)
   b. The Kingdom has the 7th highest rate in the world in terms of diabetes incidence

Type 1	Type 2
Usually young	Usually older
Short history- acute onset	Insidious onset
Not overweight	Often overweight
Insulin deficiency	Insulin resistance
Rare	Common
Requires insulin from diagnosis	Diet and lifestyle change can reverse it Then add oral medications May require insulin
Often random	Strong family history

Definition

A metabolic disease associated with glucose metabolism abnormalities as a result of insulin deficiency or insulin resistance that is also related to chronic complications affecting nerves, vessels, kidneys, etc.

Diagnosis

Diabetes mellitus is characterized by recurrent or persistent hyperglycemia, and is diagnosed by demonstrating any one of the following:

Fasting plasma glucose level	Plasma glucose	Casual (Randome) plasma glucose	Hb A1C
≥ 7.0 mmol/l	≥ 11.1 mmol/l	≥ 11.1 mmol/l (200 mg/dl)	Glycated hemoglobin
(126 mg/dl)	two hours after a 75 g oral glucose load → glucose tolerance test	Symptoms of hyperglycemia	≥ 6.5%.

Secondary causes of Diabetes Meillitus:

- **Drugs:** Steroids, thiazides.
- **Pancreatic disease:** Pancreatitis, surgery, cancer, hemochromatosis, and cystic fibrosis.
- **Endocrine disease:** Cushing's disease, acromegaly, thyrotoxicosis, pheochromocytoma
- **Others:** Glycogen storage diseases, insulin receptor antibodies.

Condition	2 hour glucose	Fasting glucose	HbA_{1c}
	mmol/l(mg/dl)	mmol/l(mg/dl)	%
Normal	<7.8 (<140)	<5.6(<100)	<5.7
Impaired fasting glycaemia	<7.8 (<140)	≥ 5.6(≥100) & <7.0(<126)	5.7–6.4
Impaired glucose tolerance	≥7.8 (≥140)	<7.0 (<126)	5.7–6.4
Diabetes mellitus	≥11.1 (≥200)	≥7.0 (≥126)	≥6.5

Screening for diabetes:

1. Routine screening for type 1 diabetes cannot be recommended except for research purposes.
2. We screen people with risk factors for Type 2 Diabetes Mellitus, and those are:
   a. Age ≥45 years
   b. Overweight (body mass index ≥25 kg/m2)
   c. Family history of diabetes mellitus in a first-degree relative
   d. Habitual physical inactivity
   e. History of gestational diabetes mellitus
   f. Hypertension (blood pressure ≥140/90 mmHg)
   g. Dyslipidemia (defined as a serum HDL concentration ≤35 mg/dL (0.9 mmol/L) and/or a serum triglyceride concentration ≥250 mg/dL (2.8 mmol/L)
   h. Previously identified A1C ≥5.7 percent, impaired glucose tolerance or impaired fasting glucose
   i. Polycystic ovary syndrome
   j. History of vascular disease

Symptoms of DM:

- Wight loss
- Polydipsia
- Polyuria
- Lethargy
- Irritability
- Blurred vision

Screening tests:

1-) Fasting plasma glucose (FPG).

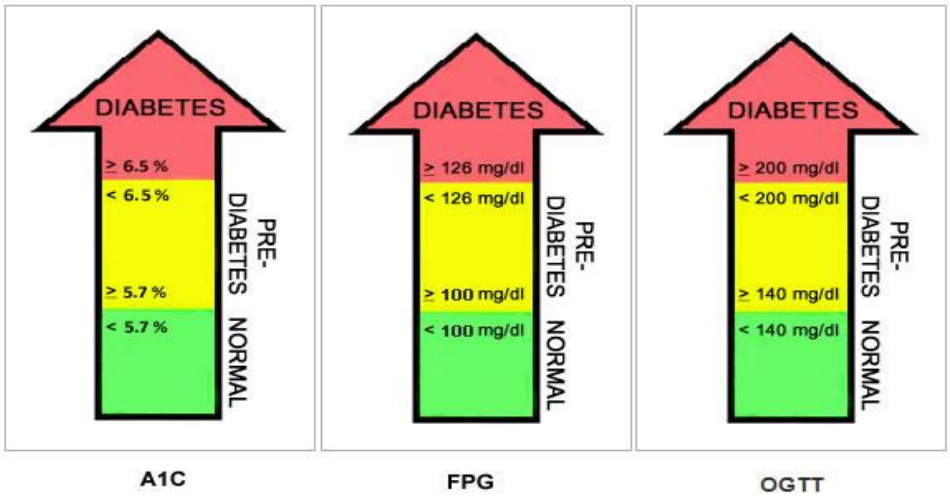
2-) 2-hour plasma glucose during an oral glucose tolerance test (2-h OGTT).

3-) Glycated hemoglobin (A1C).

Pre-diabetes and how to prevent development of diabetes

1. Pre-diabetes: when a person's blood glucose levels are higher than normal but not high enough for a diagnosis of diabetes.
2. Before people develop type 2 diabetes, they almost always have "pre-diabetes"

3. It is sometimes referred to as impaired glucose tolerance (IGT) or impaired fasting glucose (IFG), depending on what test was used when it was detected.
4. This condition puts the patient at a higher risk for developing type 2 diabetes and cardiovascular disease.
5. Patients with IGT, IFG, or an A1C of 5.7–6.4% (pre-diabetic) should be:
    a. Referred to an effective ongoing support program targeting weight loss and increasing physical activity.
    b. Metformin therapy for prevention of type 2 diabetes.
    c. At least annual monitoring for the development of diabetes.



When to add Metformin in pre-diabetes?

1. Hypertension.
2. Low HDL cholesterol.
3. Elevated triglycerides.
4. Family history of diabetes (first-degree relative).
5. Obese.
6. Under 60 years of age.

Goals to be achieved in terms of managing and controlling a diabetic patient:

1. HbA1C:

1. Lowering A1C to below or around **7%** has been shown to reduce microvascular complications of diabetes. Therefore, a reasonable A1C goal for many nonpregnant adults is <7%.

2. More stringent A1C goals (such as **<6.5%**) for selected individual patients, if this can be achieved without significant hypoglycemia or

other adverse effects of treatment. Appropriate patients might include those with short duration of diabetes, long life expectancy, and no significant CVD.

3. Less stringent A1C goals (such as **<8%**) may be appropriate for patients with a history of severe hypoglycemia, limited life expectancy, advanced microvascular or macrovascular complications, extensive comorbid conditions, and those with long-standing diabetes in whom the general goal is difficult to attain despite diabetes self-management education (DSME), appropriate glucose monitoring, and effective doses of multiple glucose-lowering agents including insulin.

2. Blood pressure:

1. People with diabetes and hypertension should be treated to a systolic blood pressure goal of **<140** mmHg.
2. Patients with diabetes should be treated to a diastolic blood pressure **<80** mmHg.
3. Lower systolic targets, such as <130 mmHg, may be appropriate for certain individuals, such as younger patients, if it can be achieved without undue treatment burden.

3. Triglyceride: <150 mg/dL (1.7 mmol/L)

4. LDL:

a. In individuals without overt CVD, the goal is LDL cholesterol <100 mg/dL (2.6 mmol/L).

b. In individuals with overt CVD, a lower LDL cholesterol goal of <70 mg/dL (1.8 mmol/L), using a high dose of a statin, is an option.

5. HDL:

a. >40 mg/dL (1.0 mmol/L) for men.

b. >50 mg/dL (1.3 mmol/L) for women

Statin therapy should be added to lifestyle therapy, regardless of baseline lipid levels, for diabetic patients:

1-) with overt CVD

2-) without CVD who are over the age of 40 years and have one or more other CVD risk factors (family history of CVD, hypertension, smoking, dyslipidemia, or albuminuria).

Management Modalities

1. Education
2. Diet
3. Exercise
4. Lifestyle modification

1. **Type 1 Diabetes Mellitus:** Insulin is the only treatment of choice in Type 1 Diabetes.
2. **Type 2 Diabetes Mellitus:** Three methods of treatment are available for diabetic patients:

1) Diet, Exercise and lifestyle advice alone
2) Oral hypoglycemic agents
3) Insulin

Oral Hypoglycemic Agents:

1. First line Agents:

a. Sulfonylurea:

1) MOA: They stimulate the release of insulin from the pancreatic B cells (insulin secretagogue)
2) Effective only if there is some residual endogenous insulin production
3) Weight gain
4) Be careful of hypoglycemia!! (Educate the patient*)

b. Biguanide (Metformin):

1) In the absence of contraindications, metformin is the first choice for oral treatment of type 2 diabetes
2) MOA: it decreases gluconeogenesis by the liver and increases peripheral utilization of glucose.
3) No weight gain, No hypoglycemia
4) GI side effects are common with metformin.
5) The most serious side effect is lactic acidosis (rare).
6) Contraindications: Impaired Renal Function.

2. Second Line Agents:

a. Glitazones: E.g. (rosiglitazone and pioglitazone)

1) Increase insulin secretion and increase insulin sensitivity
2) They cause fluid retention, so do not use if know/suspected heart failure

b. Incretins:

A group of gastrointestinal hormones that cause an increase in the amount of insulin released from the beta cells of the after eating, before blood glucose levels become elevated.

c. Meglitinides: E.g. (Repaglinide and nateglinide)

Stimulate insulin release

d. Insulin:

1) Insulin is the preferred second-line medication for patients with A1C >8.5 percent or with symptoms of hyperglycemia despite metformin titration
2) After maximum metformin and suphonylurea, you should consider adding Insulin and taper the Sulphonylurea

Insulin type	Onset of action	Peak effect	Duration of action
Lispro, aspart, glulisine	5 to 15 minutes	45 to 75 minutes	Two to four hours
Regular	About 30 minutes	Two to four hours	Five to eight hours
NPH	About two hours	4 to 12 hours	18 to 28 hours
Insulin glargine	About two hours	No peak	20 to >24 hours
Insulin detemir	About two hours	Three to nine hours	6 to 24 hours*
NPL	About two hours	Six hours	15 hours
Insulin degludec	About two hours	No peak	>40 hours

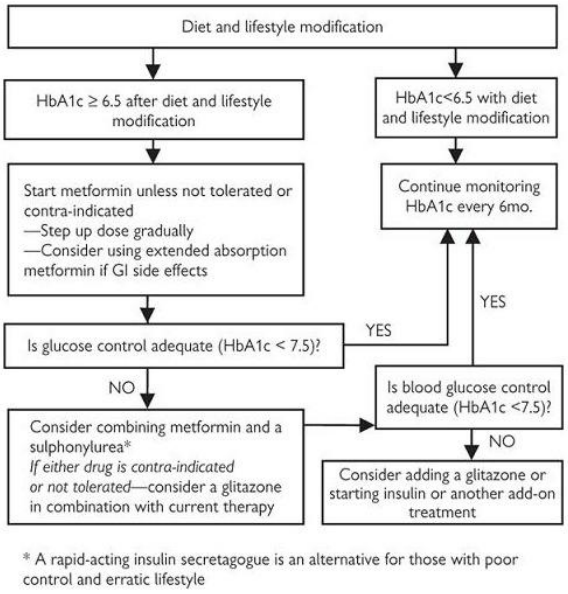


Fig. 12.1 Using oral hypoglycaemic agents in type 2 DM

Annual checkups - Investigations	
Eyes	At optometrist (Refer to Ophthalmology)
Feet, with instructions for self-care	Visual Inspection → each visit Circulation and feeling (Comprehensive) → Annually
Diet	Advice yearly; more often e.g. if overweight and trying to lose weight
Weight	2 weekly if trying to lose weight
Tests for neuropathy	Motor, sensory and autonomic
Blood pressure, thyroid, electrolytes, cholesterol, tests	Statin treatment for most non-pregnant patients, especially if cholesterol is more than 5.
Full blood count	type 1 diabetes
If fluctuating blood sugars (insulin users)	Test for thyroid disease, Addison's, Cushing's, coeliac disease. Consider depression and needle phobia.
Kidneys	protein in urine (microalbuminuria) Albumin to creatinine ratio / 24 hr. urine collection for protein / Creatinine Clearance

Important aspects of clinical examination:

- Amputation and foot ulceration, consequences of diabetic neuropathy and/or peripheral artery disease (PAD) are common.
    - Risk of ulcers or amputations is increased in people who have the following risk factors:
        - Previous amputation
        - Past foot ulcer history
        - Peripheral neuropathy
        - Foot deformity

- Peripheral vascular disease
- Visual impairment
- Diabetic nephropathy (especially patients on dialysis)
- Poor glycemic control
- Cigarette smoking

Vascular Examination:

- Palpation of pulses - Doppler (ABI) - Skin/Limb color changes – Edema - Temperature (Dermal Thermometry) - Atrophy .

Neurologic Examination

- Vibration (128Hz) - Pressure Semmes-Weinstein 10 gram monofilament - Light touch (cotton) - Two-point discrimination - Pain (pin-prick) – Temperature - Reflexes (patella & achilles) - Clonus testing - Romberg test - Babinski test - Gait

Key components of the diabetic foot exam

Inspection
Dermatologic
Skin status: color, thickness, dryness, cracking
Sweating
Infection: check between toes for fungal infection
Ulceration
Calluses/blistering: hemorrhage into callus?
Musculoskeletal
Deformity, eg, claw toes, prominent metatarsal heads, Charcot joint
Muscle wasting (guttering between metatarsals)
Neurological assessment
10-g monofilament + one of the following four
Vibration using 128-Hz tuning fork
Pinprick sensation
Ankle reflexes
VPT
Vascular assessment
Foot pulses
ABI, if indicated

- **Diabetic eye disease may include:**
- Diabetic retinopathy
- Cataract
- Glaucoma
- Refractive Error

They all increase the risk of visual loss among diabetic patients.

PRIMARY HEALTH CARE BOOKLET

Sexually transmitted infections

كتابة: عبدالعزيز المنيع ، فيصل الماضي ، أحمد المطلق / مراجعة وتعديل: سليمان الشمري

Sexually transmitted infections

Definition:

"Sexually transmitted infections (STIs) are infections that are spread primarily through person-to-person sexual contact. There are more than 30 different sexually transmissible bacteria, viruses and parasites." World Health Organization.

Table .1/ Common **bacterial** infections

Organism	Causes
Neisseria gonorrhoeae	Gonorrhoea or gonococcal infection
Chlamydia trachomatis	Chlamydial infections
Treponema pallidum	Syphilis
Haemophilus ducreyi	chancroid
Klebsiella granulomatis	Granuloma inguinale or donovanosis

Table .2/ Common **viral and Parasites** infections

Organism	Causes
Human immunodeficiency virus	AIDS
Herpes simplex virus type 1 & 2	Genital herpes
Human papilloma virus	Genital warts and certain subtypes lead to cervical cancer in women
Hepatitis B virus	Hepatitis and chronic cases may lead to cancer of the liver
Parasites: Trichomonas vaginalis	vaginal trichomoniasis

How to approach a patient with STI:

1. Patients presenting with possible STIs are frequently anxious, embarrassed and concerned about confidentiality. *The clinical setting* ***must ensure privacy*** *and reinforce confidentiality.*
2. **History:**
   - **A.** The three most common symptoms are:
     - Vaginal discharge
     - Urethral discharge
     - Genital ulceration
   - B. Also any associated fever, pain, itch, malodour, genital swelling, skin rash, joint pains and eye symptoms.
   - C. All patients should be asked about dysuria, haematuria and loin pain
   - D. In women, menstrual, contraception and obstetric history should be obtained

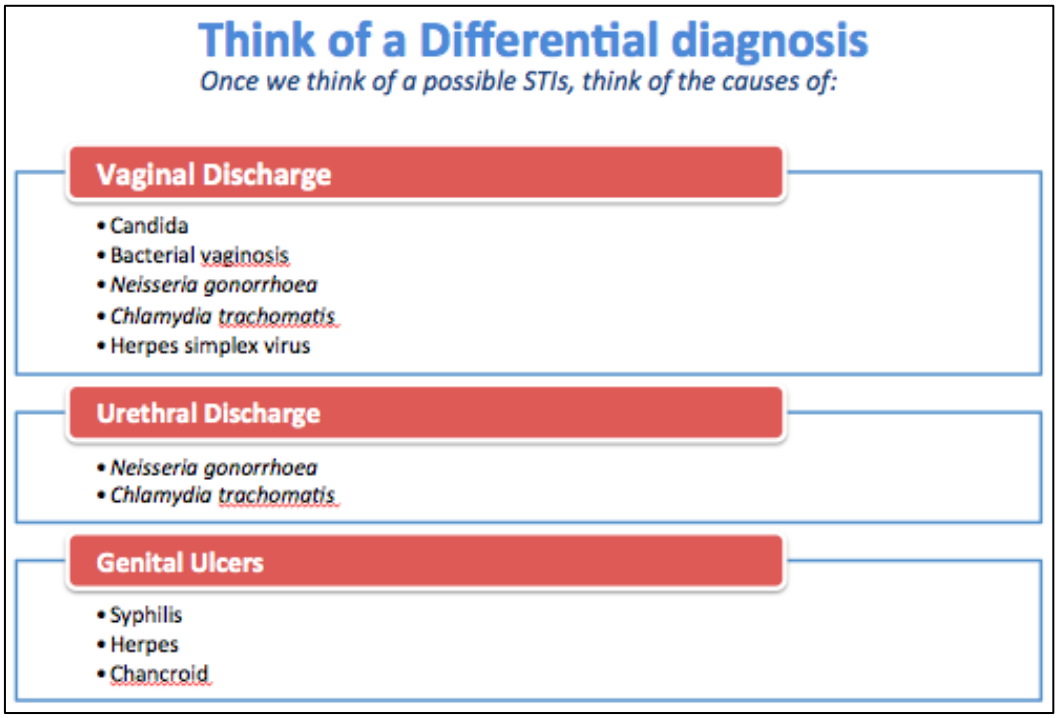
E. What essential questions must be asked?

a) You must take a sexual history, perhaps prefacing this by saying, **'I understand you are married. When did you last have sex with your wife?'.**

b) The next question should be, **'When did you last have sex with anyone else?'.** Let's say he answers somewhat gloomily, 'Last week'.

c) The questioning should continue along the following lines: **'Is this a regular partner or was it more of a "one-off"?'; 'Did you use a condom?'** and **'Was this partner female or male?'.**

Differential Diagnosis



3. *Examination:*

A. General examination must include the mouth, throat, skin and lymph nodes in all patients

B. The inguinal, genital and perianal areas should be examined with a good light source.

C. The groins should be palpated for lymphadenopathy and hernias.

D. The pubic hair must be examined for nits and lice.

E. The external genitalia must be examined for signs of erythema, fissures, ulcers, chancres, pigmented or hypopigmented areas and warts.

F. The urethral meatus is located and the presence of discharge noted
G. The cervix should be inspected for ulceration, discharge, bleeding and ectopy and the walls of the vagina for warts.

Bacterial infections

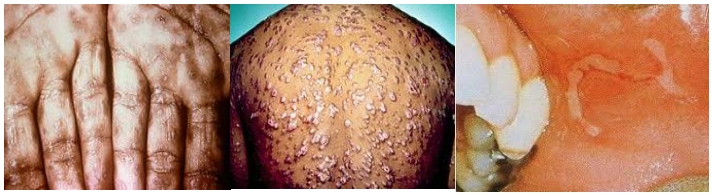
1. Syphilis:

Definition

Syphilis is infection with the bacteria Treponema pallidum which spread through broken skin or mucous membranes. It has several stages:

1) Primary syphilis: The first stage present with chancre (a firm, painless, non-itchy skin ulceration) that form at the site of infection about 2-3 weeks after pateints are first infected. He may not notice the sores or any symptoms. The sores disappear in about 4-6 weeks, even without treatment. The bacteria become dormant (inactive) in the system at this stage.

2) Secondary syphilis: Occurs about 5 weeks after the first sores heal. About 33% of those who do not have their primary syphilis treated will develop this second stage. These symptoms are ;diffuse rash which frequently involves the palms of the hands and soles of the feet +snail track ulcer. They will often also go away without treatment again, the bacteria become dormant (inactive) in the system.



3) Latent syphilis: No symptoms just sero-postivevity in the blood.

4) Tertiary syphilis: Final stage of syphilis 3-10 years after initial infection. The infection spreads to the brain, nervous system(causing meningitis siezures, dementia), heart, skin, and bones. The dormant bacteria may be detectable either by seeing the damage they cause to a part of the body, or through a blood test for syphilis.

Investigations

a. Dark field microscopy of smear: from primary or secondary lesions. May be negative.
b. Serologic tests: (commonly used)

i. **Nontreponemal tests:**
The tests are called rapid plasma reagin (RPR) and venereal disease research laboratory (VDRL). Become positive during the primary stage. Used for screening and titer used to follow up therapy.

ii. **Treponemal tests:** Detects specific antibody to T.pallidum
1. Fluorescent treponemal antibody (FTA-ABS).
2. Microhemagglutination test (MHA-TP) (antigen attached to erythrocytes)
- Positive results confirm RPR and VDRL.
- Treponemal tests are positive at all stages of syphilis

iii. **IgM:** used to diagnose congenital syphilis.

Treatment: Antibiotics are effective early
1. Benzathine penicillin (1 dose, IM), if allergic: doxycycline for 2 weeks orally
2. If latent/tertiary: Benzathine penicillin (3 doses, IM) for 3 weeks 1 dose each week.

2. Chlamydia:

Definition

1. Chlamydia infection is a common sexually transmitted infection (STI) in humans caused by the bacterium Chlamydia trachomatis.
2. Incubation Period of Chlamydia: 7-21 days

Symptoms:

Some people refer to Chlamydia as a silent disease because there are rarely any noticeable symptoms initially. Experts say that approximately 50% of infected men and 70% of infected women will have no symptoms at all. Others will have such minor symptoms that the infection goes unnoticed.

1. **Women** Genital Chlamydia does not usually present symptoms in women. However, there may be non-specific symptoms, including:
   a. Cystitis: inflammation of the bladder.
   b. A change in vaginal discharge.
   c. Slight lower abdominal pain.
   d. Pelvic pain.
   e. Pain during sexual intercourse - may be every time, or intermittently.
   f. Bleeding between menstrual periods.
2. **Men** symptoms are usually from complications

Causes

Chlamydia may be transmitted by: **having unprotected sex with an infected person.** As Chlamydia infection often presents no symptoms, an infected person may pass it on to his/her sexual partner without knowing.

Childbirth - an infected mother can pass the infection on to her baby during childbirth. Sometimes the infection may lead to complications for the infant, such as pneumonia.

Diagnoses

1. Laboratory tests: **Nucleic acid amplification tests *(NAAT)***
   These tests find the genetic material (DNA) of Chlamydia bacteria. These tests are the most sensitive tests available, meaning that they are very accurate and that they are very unlikely to have false-negative test results.

   a. **Nucleic acid hybridization tests** *(DNA probe test):* A probe test also finds chlamydia DNA. A probe test is very accurate but is not as sensitive as nucleic acid amplification tests.
   b. **Enzyme-linked immunosorbent assay** *(ELISA, EIA):* This quick test finds substances (Chlamydia antigens) that trigger the immune system to fight Chlamydia infection.
   c. **Direct fluorescent antibody test** *(DFA):* This quick test also finds Chlamydia antigens.
   d. **Chlamydia cell culture**: Cell culture is more expensive and takes longer (two days) than the other tests. The culture must be grown in a laboratory.

Treatment

1. **Antibiotics**: are at least 95% effective in treating Chlamydia
   a. **Azithromycin**
   b. **Doxycycline**

   Some patients may have the following **side effects** when they take the antibiotics:
   i. Diarrhea
   ii. Abdominal cramps
   iii. Dyspepsia (Upset stomach)
   iv. Nausea

Complications

Early diagnosis and treatment greatly reduces the risk of complications. Complications can be prevented with regular screening, or by seeking medical attention as soon as symptoms

appear.

Chlamydia complications:

1. **Women:**
    1. **Pelvic Inflammatory Disease (PID)**
    2. **Cervicitis**: inflammation of the cervix
    3. **Salpingitis** - inflammation of the fallopian tubes.
    4. **Bartholinitis**: inflammation of the Bartholin gland, which produces the lubricating mucus to make sexual intercourse easier
2. **Men:**
    1. **Fertility.**
    2. **Urethritis**
    3. **Epididymitis**
    4. **Reiter syndrome**

3. Gonorrhoea:

Definition

Gonorrhoea is caused by infection with Neisseria gonorrhea and may involve columnar epithelium in the lower genital tract, rectum, pharynx and eyes. Transmission is usually the result of vaginal, anal or oral sex. Gonococcal conjunctivitis may be the result of accidental infection from contaminated fingers. Untreated mothers may infect their babies during delivery, resulting in ophthalmia neonatorum. Infection of children beyond the neonatal period is usually indicative of sexual abuse.

Clinical features:

1. The incubation period is usually 2–10 days.
2. **In men:**
    a. The anterior urethra is commonly infected, causing urethral discharge and dysuria, but symptoms are absent in about 10% of cases.
    b. Examination will usually show a mucopurulent or purulent urethral discharge.
3. **In women:**
    a. The urethra, paraurethral glands/ducts, Bartholin's glands/ducts or endocervical canal may be infected.
    b. About 80% of women who have gonorrhoea are asymptomatic. There may be vaginal discharge or dysuria but these symptoms are often due to additional infections such as

chlamydia, trichomoniasis or candidiasis, making full investigation essential.

c. Lower abdominal pain, dyspareunia and intermenstrual bleeding may be indicative of PID.

d. Clinical examination may show no abnormality or pus may be expressed from urethra, paraurethral ducts or Bartholin's ducts. The cervix may be inflamed, with mucopurulent discharge and contact bleeding.

4. Gonococcal conjunctivitis is an uncommon complication, presenting with purulent discharge from the eye(s), severe inflammation of the conjunctivae and oedema of the eyelids, pain and photophobia.
5. Conjunctivitis must be treated urgently to prevent corneal damage.

Investigations:

Gram-negative intracellular diplococcimay be seen on microscopy of smears from infected sites (see Fig. 15.1, p. 413). The diagnosis must be confirmed by culture or nucleic acid amplification test (NAAT) such as polymerase chain reaction (PCR).

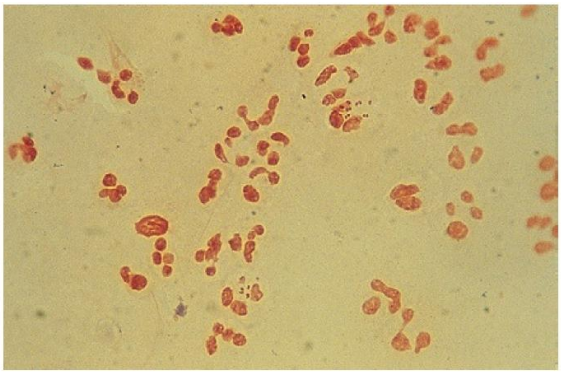


Fig. 15.1 A Gram-stained urethral smear from a man with gonococcal urethritis. Gram-negative diplococci are seen within polymorphonuclear leucocytes.

Management of adults:

Uncomplicated gonorrhoea responds to a single adequate dose of a suitable antimicrobial (many UK centres currently use oral cefixime 400mg, Box 15.8); cure rates should exceed 95%. Longer courses of antibiotics are required for complicated infection. Partner(s) of patients with gonorrhoea should be seen as soon as possible.

15.8 Treatment of uncomplicated anogenital gonorrhoea

Uncomplicated infection

- Cefixime 400 mg stat *or*
- Ciprofloxacin 500 mg orally stat[1,2] *or*
- Ofloxacin 400 mg orally stat[1,2] *or*
- Amoxicillin 3 g *plus* probenecid 1 g orally stat[3]

Quinolone resistance

- Ceftriaxone 250 mg i.m. stat *or*
- Spectinomycin 2 g i.m. stat[4]

Pregnancy and breastfeeding

- Cefixime 400 mg stat *or*
- Ceftriaxone 250 mg i.m. stat *or*
- Amoxicillin 3 g *plus* probenecid 1 g orally stat[3] *or*
- Spectinomycin 2 g i.m. stat[4]

Pharyngeal gonorrhoea

- Cefixime 400 mg stat *or*
- Ceftriaxone 250 mg i.m. stat *or*
- Ciprofloxacin 500 mg[1,2] orally stat *or*
- Ofloxacin 400 mg[1,2] orally stat

[1]Contraindicated in pregnancy and breastfeeding.
[2]If prevalence of quinolone resistance for *N. gonorrhoeae* < 5%.
[3]If prevalence of penicillin resistance for *N. gonorrhoeae* < 5%.
[4]May only be available in specialist clinics.

Delay in treatment may lead to complications (Box 15.9).

15.9 Complications of delayed therapy in gonorrhoea

- Acute prostatitis
- Epididymo-orchitis
- Bartholin's gland abscess
- PID (may lead to infertility or ectopic pregnancy)
- Disseminated gonococcal infection

Viral infections:

1. Genital Herpes:

Etiology

Herpes Simplex Virus 2

HSV replicates in the dermis & epidermis → travels via sensory nerves up to the dorsal root ganglia→ can be reactivated at any time.

Transmission

Sexual contact with ulcer

Asymptomatic individuals may still be shedding the virus

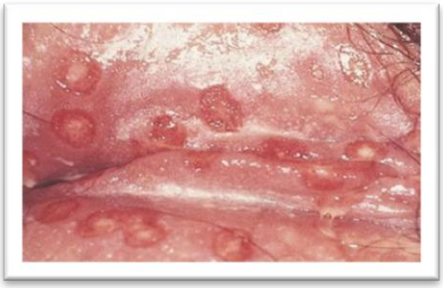
Presentation

Painful genital vesicles or pustules

Tender inguinal **lymphadenopathy** and **vaginal/urethral discharge**

Treatment

1. Antiviral Rx: **Acyclovir (p.o./topical for 7-10d) if primary.**
2. Oral if reoccurring
3. IV if immunocomprmised



2. Human papilloma virus

Etiology:

1) Human papilloma virus has over 100 subtypes, **(type 16,18)** cause cervical cancer. 6 and 11 cause genital warts.

2) **The most common sexually transmitted infection (STI).**

Presentation:

Pinkish/white small lumps or larger cauliflower-shaped lumps on the genital area.

Investigation

Screen for cervical cancer by **The Pap test** (or Pap smear) looks for precancers, cell changes on the cervix.

Treatment:

1. Podopfilox lotion or gell
2. Cryotherpay

3. HIV

Presentation:

1) Primary infection: Mono like symptoms (fever, sweats malaise) occur within weeks after exposure

2) Asymptomatic infection:
   - **a.** Normal CD4 cell count >500
   - **b.** Lasts for 5-8 years depending on treatment

3) Pre-Aids:
   - **a.** Evidence of immune system dysfunction

b. Generalized lymphadenopathy, fungal infection, skin manifestations

4) Aids:

a. <200 CD4 cells
b. Unusual cancers (kaposi sarcoma)
c. PCP infection : most common cause of death
d. TB

Diagnosis:

Table .3/ Diagnosis of HIV:

Types	Comments
1. ELISA	Screening test
	Sensitivity >99.5%
	Negative test excludes HIV
2. Western Blot	Confirmatory test
3. **PCR** (Polymerase Chain Reaction)	Confirmatory test
	To asses viral load

Treatment: Antiviral Therapy

Triple therapy HAART:

1) 2 nucleoside reverse transcriptase inhibitors
2) Protease inhibitor

PRIMARY HEALTH CARE BOOKLET

Common psychiatric problems

كتابة: عبدالرحمن الجاسر ، أحمد الحمود / مراجعة وتعديل: سعود الرميح

Common psychiatric problems

Epidemiology

A cross-sectional study was conducted in 10 secondary schools for girls using the Arabic version of the symptom-revised checklist 90 (SCL 90-R), a mental health questionnaire that was administered to the girls by fourth-year female medical students.

The most prevalent mental symptoms in the 545 female students were phobic anxiety (16.4%), psychoticism (14.8%), anxiety (14.3%), and somatization (14.2%). The prevalence of depression, paranoid ideation and interpersonal sensitivity amounted to 13.9%, 13.8% and 13.8%, respectively. The least prevalent mental symptoms were hostility (12.8%) and obsessive-compulsive behavior (12.3%). Overall, psychological symptoms (in terms of a positive global severity index) were found in 16.3% of the girls. In a multivariate logistic regression analysis, no significant relationship was found with sociodemographic factors.

Depression

Definition

Depressive disorders are characterized by persistent low mood, loss of interest and enjoyment, neurovegetative disturbance, and reduced energy, causing varying levels of social and occupational dysfunction. Depressive symptoms include **depressed mood**, **anhedonia**, **weight changes**, **libido changes**, **sleep disturbance**, **psychomotor problems**, **low energy**, **excessive guilt**, **poor concentration**, and **suicidal ideation**.

Etiology

The etiology of depression remains poorly understood. Susceptibility to a depressive disorder is 2 to 4 times greater among the first-degree relatives of patients with a mood disorder than among other people. It is unclear whether a gene-environment interaction can help explain susceptibility to depression or predict response to treatment. A meta-analysis proposed by the National Institute of Mental Health in 2009 supported the previous finding that stressful life events have a potent relationship with the risk of depression. However, other studies suggest a role for genetic polymorphisms in predicting medication adverse effects.

Classifications

According to the DSM Classification:

1. **Major Depressive Disorder** (Unipolar Depression):
   2 weeks of depression at least and 5 symptoms.
2. **Dysthymic Disorder** (Chronic Depression):
   Low-grade depression for at least 2 years.
3. **Postpartum Depressive Disorder.**
4. **Seasonal Depressive Disorder** (Usually in Winter).
5. **Depressive Disorder NOS** (Not Otherwise Specified).

Diagnosis

Major Depressive Disorder: (DSM-IV-TR Criteria)

1. Presence of a single or more major depressive episode (each separated by at least 2 months) **for at least 2 weeks**.
2. The major depressive episode is not better accounted for by schizoaffective disorder and is not superimposed on schizophrenia, schizophreniform disorder, delusional disorder, or psychotic disorder not otherwise specified.
3. There has never been a manic episode, a mixed episode, or a hypomanic episode.

Major Depressive Episode:

1. 5 of the mentioned clinical features & at least one of the symptoms is either 1) depressed mood or 2) loss of interest or pleasure.
2. The symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.
3. The symptoms are not due to the direct physiological effects of a substance (e.g. a drug of abuse, a medication) or a general medical condition (e.g., hypothyroidism).
4. The symptoms are not better accounted for by grief.

Dysthymia Depressive Disorder: (DSM-IV-TR Criteria)

1. 2 of the mentioned clinical features for at least 2 years.
2. During the 2 years there has to be no major depressive episode.
3. There has never been a manic episode, a mixed episode, or a hypomanic episode.
4. The symptoms are not due to the direct physiological effects of a substance (e.g. a drug of abuse, a medication) or a general medical condition (e.g., hypothyroidism).
5. The symptoms are not better accounted for by grief.

Management

1. **Pharmacological Therapy:**
   a. Usually 3-5 weeks for desired effect, but unfortunately side effects can start within few days.

b. These groups are more beneficial than tricyclic drugs (especially in dysthymic disorder).
c. Selective Serotonin Reuptake Inhibitors (SSRI).
d. Selective Serotonin–Norepinephrine Reuptake Inhibitors (e.g. Venlafaxine, Duloxetine).
e. Monoamine Oxidase Inhibitors (MAOI): Don't give with SSRI or Tricyclic antidepressants.

2. **Psychological Therapy:**
   a. Supportive Therapy.
   b. Cognitive & Behavior Therapy.
3. **Electroconvulsive Therapy (ECT):**
   a. As a last resort.
   b. Safer in pregnant women than antidepressant.

Prognosis

1. Approximately 20 weeks for recovery.
2. Relapse in 25% of patients.

When to Refer to Psychiatrist for Admission

1. Suicidal or Homicidal Patients.
2. Severe Psychomotor Retardation and Malnutrition (For ECT).
3. Diagnostic Purpose.
4. Severe Depression With Psychotic Features (Possible ECT).

Summary

Characterized by persistent low mood, loss of interest and enjoyment, and reduced energy. It's common in primary care, affecting 5% to 10% of patients in this setting. Often have a personal or family history of depression; have experienced a recent stress, trauma, or loss; or have comorbid medical illness. Recommendations suggest that centers screening adults should have systems in place that ensure positive screening results are followed by accurate diagnosis, effective treatment, and careful follow-up. Most patients respond well to psychotherapy, antidepressants, or a combination of both. Suicidal ideation can occur before, during, or after treatment, so clinicians should assess this at each visit.

Anxiety

Definition:

Anxiety is a normal feeling of apprehension in certain threatening situation.

Anxiety disorders: are abnormal states in which the most striking features are worry, dread and physical symptoms of anxiety that indicate a hyperactive autonomic nervous system and not caused by organic brain disease, medical illness nor Psychiatric disorder.

Etiology:

1. Presence of physical or emotional trauma.
2. Genetic factors (their first-degree relatives (odds ratio 6.1) developing the disorder).

Primary anxiety disorders:

1. **Generalized anxiety disorder.**
2. **Agoraphobia without history of panic disorder.**
3. **Panic disorder without or with agoraphobia.**
4. **Specific phobias and social phobia (social anxiety disorder).**
5. **Obsessive-compulsive disorder.**
6. **Acute stress disorder.**

Diagnosis of anxiety

PRIMARY ANXIETY-SPECTRUM DISORDERS: (DSM-IV Diagnostic Criteria)

The following primary anxiety disorders are described in the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision (DSM-IV-TR):

1. **Generalized anxiety disorder:** Patients who have generalized anxiety disorder experience **chronic excessive nervousness**, **exaggerated worry**, **tension**, **and irritability that appear to have no cause or are more intense than the situation warrants**. Their worries are often related to their perceived inability to perform with punctuality and competence in various settings and circumstances. Over the course of the disorder, physical signs—such as **restlessness**, **difficulty in falling or remaining asleep**, **headaches**, **trembling**, **twitching, muscle tension**, or **sweating**—often develop, which lead to further worries. Patients with generalized anxiety disorder may also have other anxiety and mood disorders.

2. **Agoraphobia:** fear of any open or public space. The condition can be quite disabling. Patients with agoraphobia fear being in a situation in which they experience anxiety or panic and from which escape might be difficult or embarrassing. As a result, they avoid those

situations that cause anxiety or panic. It is the fear of the anxiety that leads to agoraphobia. Agoraphobia can be accompanied by panic disorder and panic attacks, or it can occur alone without a history of panic attacks.

3. **Panic disorder:** Patients with panic disorder usually describe periods of intense fear or discomfort that they call panic attacks. Very often, they seek medical treatment because they fear that their physical symptoms—which may include chest pain, dizziness, nausea, chills, trembling, and palpitations—are caused by a heart attack.
   Patients may worry about recurrent and often unexpected panic attacks. The anticipatory anxiety and intense fear of future attacks may lead to the development of phobic avoidance. The combination of panic symptoms and the phobic avoidance can impair the patient's professional, social, and familial functioning.

4. **Specific phobias:** Phobias are manifested by irrational fears when a person is exposed to or is in close physical contact with specific objects or situations that trigger intense anxiety. The intense anxiety can also be triggered when the person sees or hears the name of the object, or sees pictures of the object. Phobic avoidance develops, and the patient will altogether avoid all the specific things or situations that trigger the intense anxiety. The avoidance leads to an ongoing impairment in the patient's ability to function in settings where exposure to the specific object occurs.

5. **Social phobia (social anxiety disorder):** Social phobia is manifested by excessive, persistent fear of social and performance situations that is so severe that it disrupts daily life and relationships. Persons with social anxiety have a persistent, intense, and ongoing fear of being extremely embarrassed or being watched, judged by others, or humiliated by their own actions. The most common social phobia is fear of public speaking.

6. **Obsessive-compulsive disorder:** Patients with obsessive-compulsive disorder experience repetitive ideas (obsessions) that are distressing and provoke intense symptoms of anxiety. To counteract the anxiety, patients use certain sets of actions, or rituals, and repetitive behaviors (compulsions). The repetitive behaviors diminish the anxiety temporarily, only to have it return within a relatively short period of time. As a result, patients often

continue the compulsive behaviors, which consume most of their time, or they avoid situations with which the obsessions are associated, thus constricting their activities and range of behaviors. Patients with obsessive-compulsive disorder may have only obsessions or only compulsions or both obsessions and compulsions. They most often experience obsessions that they must avoid contamination, that actions or items need to be checked for completion, or that they must engage in certain detailed and elaborate activities to prevent future harm to oneself or others. Repetitive, intrusive thoughts or images about violence or sexual actions, or urges to engage in violence or sexual actions are also common. Despite patients' awareness of the irrational nature of their condition, they feel unable to control their obsessions or to prevent their compulsions. The disorder hinders mental, social, and academic performance; if untreated, it may lead to permanent disability because of the loss of meaningful interpersonal relations and employment.

7. **Acute stress disorder:** Patients with acute stress disorder experienced a traumatic event in which they were threatened or seriously injured, or they witnessed a traumatic event in which other persons were seriously injured or died. During the traumatic event, they responded with intense fear and helplessness. The condition is usually associated with **dissociative symptoms**, such as **numbing, detachment, a reduction in awareness of the surroundings, derealization, or depersonalization; re-experiencing of the trauma; avoidance of associated stimuli; and significant anxiety, including irritability, poor concentration, difficulty in sleeping, and restlessness**. The diagnosis of acute stress disorder is made **when the symptoms occur within 4 weeks of the traumatic event and are present for a minimum of 2 days and a maximum of 4 weeks**. The disorder may resolve with prompt intervention or with the passage of time; however, in some patients, acute stress disorder may progress into a more severe psychiatric condition, such as posttraumatic stress disorder.

8. **Posttraumatic stress disorder:** This disorder develops after a person experiences, witnesses, or confronts a physically and/or psychologically distressing event. The event may involve actual or threatened death or serious injury or a threat to the physical integrity of oneself or others. **Symptoms of posttraumatic stress disorder include re-experiencing the traumatic event, a**

consistent pattern of avoidance of themes associated with the traumatic event, and hyperarousal and autonomic hyperactivities that may be manifested by difficulties with sleep or concentration, exaggerated startle reactions and, at times, anger outbursts. The diagnosis is made if the **symptoms have been present for at least 1 month and cause clinically significant distress or impairment in functioning.**

Treatment

Pharmacotherapy and cognitive-behavioral (psychosocial) therapy are the most commonly used options available to primary care providers to treat patients with anxiety disorders Collaborative Care.

Pharmacotherapy:

a. First-line medications
- Selective-serotonin reuptake inhibitors (SSRIs) e.g. paroxetine
- Serotonin-norepinephrine reuptake inhibitors (SNRIs) e.g. Venlafaxine

b. Second-line medications
- Tricyclic antidepressant e.g. Imipramine
- Benzodiazepines e.g. diazepam

Treatment of anxious patients can be professionally rewarding for the primary care physician: many patients with anxiety disorders show remarkable improvement with treatment. For those patients who prove to be more difficult to treat, referral to a mental health professional should be initiated.

Somatization :

Introduction

Somatization is a syndrome of nonspecific physical symptoms that cannot be fully explained by a known medical condition after appropriate investigation. In addition, the symptoms may be caused or exacerbated by anxiety, depression, and interpersonal conflicts, and it is common for somatization, depression, and anxiety to all occur together. Somatization can be conscious or unconscious and may be influenced by a desire for the sick role or for personal gain.

Epidemiology

Somatization is common in the general population. More than 50 percent of patients presenting to outpatient medical clinics with a physical complaint do not have a medical condition.

Risk factors for somatization include:

1. Female sex.
2. Fewer years of education.
3. Minority ethnic status.
4. Low socioeconomic status.

Etiology

Controversy exists whether somatization is to be considered a purely psychiatric disorder, or if it should be viewed as a syndrome of multiple unexplained symptoms that complicate the presentation of a general medical condition.

1. The genetic basis for somatization is not clear. Some, but not all studies, indicate a familial pattern for somatization. The familial aggregation may be due to genetic or environmental factors, or both.
2. Childhood sexual abuse and recent exposure to physical or sexual violence are consistently associated with somatization in adult women.
3. Physical symptoms may offer a means to express distress when patients do not easily express emotions in words (alexithymia).

CLINICAL PRESENTATION

The essential feature of somatization is a **chronic history of unexplained physical symptoms, which the patient attributes to a nonpsychiatric disease.**

Somatizing patients present with a wide array of symptoms:

1. **Pain symptoms**, including headache, back pain, dysuria, joint pain, diffuse pain, and extremity pain.
2. **Gastrointestinal symptoms**, including nausea, vomiting, abdominal pain, bloating, gas, and diarrhea.
3. **Cardiopulmonary symptoms**, including chest pain, dizziness, shortness of breath, and palpitations.
4. **Pseudoneurologic symptoms**, including fainting, pseudoseizures, amnesia, muscle weakness, dysphagia, double or blurred vision, difficulty walking, difficulty urinating, deafness, and hoarseness or aphonia.

5. **Reproductive organ symptoms**, including dyspareunia, dysmenorrhea, and burning in sex organs.

The somatizing patient can also be recognized by the multiple unexplained symptoms, vague and inconsistent history, underlying sense of anguish, persistent unspoken demands, lack of factors that exacerbate or alleviate symptoms, and lack of positive findings on physical examination.

Coexisting psychiatric disorders

1. Somatization is strongly associated with anxiety.
2. In addition to depression and anxiety, somatization is often associated with personality disorders. The most common were avoidance, paranoia, self-defeating, and obsessive-compulsive

Medical evaluation

The evaluation of a patient presenting with possible somatization includes taking a history, performing a physical examination, reviewing laboratory data, and communicating with other clinicians.

1. **History of present illness**

   In taking a history, the clinician should pay attention to how the physical symptoms are related to the patient's emotions and social situation, and if any stressful personal events such as losses have occurred. The pattern of pain should be assessed. The clinician should determine what exacerbates or alleviates the symptoms, and why the patient believes he/she is suffering. The patient may be convinced he/she has a specific disease, is seriously ill, or is dying. Assess whether the patient can be reassured.

   The patient should be asked about medications, including over-the-counter, prescription, and complimentary medications. Additionally, patients should be questioned about substance use, including alcohol.

   It is important to ask whether the patient has experienced physical or sexual abuse, whether the patient feels safe in his/her current relationships, and whether he/she feels threatened or afraid in any way, either at home or in other settings.

2. **Past medical illness**

   Some patterns of somatization are characterized by a longitudinal course beginning in childhood. Patients may report that their parents were attentive only when they were sick.

Ask about a lifetime history of anxiety disorder, depressive disorder, or multiple unexplained symptoms. In addition, prior treatment with psychotropic medications should be assessed.

3. **Family history**

The family history should be examined for a model of disability or somatization, and the presence of depression or anxiety disorder.

4. **Social history**

Consider the social context in which the physical symptoms appear, including likely stresses at the patient's stage of development. Financial pressures, work, unemployment, disability history, history of arrests, time in prison may provide context for the symptoms.

5. **Physical examination**

The physical examination should satisfy the clinician that the patient does not have a medical disease. The examination provides a baseline for detecting change over time. Additionally, patients believe their complaints are taken seriously when a physical examination is performed.

6. **Laboratory evaluation**

Laboratory testing should be done judiciously to evaluate current and new physical symptoms. Often, extensive testing has previously been done to look for a diagnosis.

Diagnosis

1. Somatization is too often a **diagnosis of exclusion**.
2. The DSM-IV establishes the following five criteria for the diagnosis of this disorder:
   a. History of somatic symptoms prior to the age of 30
   b. Pain in at least four different sites on the body
   c. Two gastrointestinal problems
   d. One sexual symptom.
   e. One pseudoneurological symptom.

Multisomatoform disorder:

1. **Factitious disorder:** The essential feature of factitious disorder is intentionally faking symptoms in order to assume the sick role, i.e.

to be a patient. **No external incentives such as financial gain are present**. Patients with factitious disorder tend to have some medical knowledge.

2. **Malingering:** The essential feature of malingering is intentionally faking or grossly exaggerating symptoms **for an obvious, external incentive** such as avoiding work, avoiding criminal prosecution, obtaining financial compensation, or obtaining medications. The motivation for symptom production in malingering is an external incentive, whereas in factitious disorder the motivation is to assume the role of patient.

Screening

Primary care and other clinicians can use a brief screening instrument to assess for somatoform disorders.

Patient Health Questionnaire: Screening for Somatoform Disorders

During the past four weeks, how much have you been bothered by any of the following problems?

	Not at all	*A little*	*A lot*
Stomach pain			
Back pain			
Pain in your arms, legs, or joints (knees, hips, etc.)			
Menstrual cramps or other problems with your periods			
Pain or problems during sexual intercourse			
Headaches			
Chest pain			
Dizziness			
Fainting spells			
Feeling your heart pound or race			
Shortness of breath			
Constipation, loose bowels, or diarrhea			
Nausea, gas, or indigestion			

NOTE: *If a patient reports being bothered "a lot" by at least three of the symptoms without an adequate medical explanation, a somatoform disorder should be considered.*

Differential diagnosis

The symptoms that occur in somatization occur in many other medical and psychiatric conditions, like:

1. **Depression.**
2. **Panic disorder.**

3. **Substance use disorder**: Many symptoms of substance intoxication and withdrawal also occur in somatization.

Management

1. After appropriate investigation, inform the patient that no further investigations are indicated.
2. Limit the number of doctors consulted.
3. Limit the number of invasive investigation.
4. Limit the amount of medication. Benzodiazepines, stimulants and analgesics should be strenuously limited.
5. Diagnose and adequately treat comorbid psychiatric disorders. Be alert for depression and anxiety. Personality disorder will make management more difficult.
6. Encourage return to normal activities. Encourage hobbies, exercise, education and cultural pursuits – these will distract the patient from his/her body, stretch and strengthen the body and assist the return to normal function. Reward attempts at activities with praise.
7. Educate and involve the family in management.
8. Understand the need to repeat the reassurance, encouragement of activities and conditions of care.

Psychotherapy

Introduction

Psychotherapy is an interpersonal treatment based on psychological principles. It is individualized to the patient, seeking to help him or her with a psychiatric disorder, problem, or adverse circumstance.

There are many types of psychotherapy with varying methods and levels of empirical support. The choice of the most appropriate type of psychotherapy is in part based upon the patient's specific problem or diagnosis.

1. **Evidence-based psychotherapies**
   In some cases, psychotherapy may be more effective when administered in conjunction with medication. Efficacy data are described in detail separately under individual disorders.
2. **Cognitive and behavioral therapies**
   CBT often includes education, relaxation exercises, coping skills training, stress management, or assertiveness training. In cognitive therapy, the therapist helps the patient identify and correct distorted, maladaptive beliefs. Behavioral therapy uses thought

exercises or real experiences to facilitate symptom reduction and improved functioning. Cognitive behavioral therapy is an evidence-based treatment for psychiatric disorders including depression, generalized anxiety disorder, post-traumatic stress disorder, panic disorder, eating disorders, and obsessive-compulsive disorder.

3. **Psychodynamic psychotherapy**

   Psychodynamic therapy primarily relies on developing patient insight. Psychodynamic psychotherapy is based upon the idea that childhood experiences, past unresolved conflicts, and previous relationships significantly influence an individual's current situation in life.

4. **Interpersonal psychotherapy**

   Interpersonal therapy (IPT) addresses interpersonal difficulties that lead to psychological problems. Interpersonal psychotherapy focuses on the individual's interpersonal life in four problem areas: grief over loss, interpersonal disputes, role transitions, and interpersonal skill deficits.

5. **Motivational interviewing**

   Motivational interviewing is a type of psychotherapy that is used in primary care and mental health care to encourage patients to change maladaptive behaviors.

6. **Supportive psychotherapy**

   Supportive psychotherapy or counseling is widely used in medical practice, e.g. to help individuals cope with illness, deal with a crisis or transient problem, and maintain optimism or hope. Techniques vary but most models emphasize communication of interest and empathy; supportive therapy may also include guidance on available services, advice, respect, praise, and/or encouragement.

Format of psychotherapy

In addition to the orientation of therapy, treatment is offered in different formats:

1. Individual therapy: Individual therapy is the most commonly practiced format of psychotherapy.
2. Couple therapy.

3. Family therapy.
4. Group therapy.

Indications for psychotherapy

The clinician should consider initiating or referring a patient for psychotherapy for the following purposes:

1. Treatment of a psychiatric disorder, with the goal of reducing or ameliorating symptoms and improving functioning.
2. Changing maladaptive thoughts, behaviors, or relationships.
3. Providing support when a crisis, a difficult period, or a chronic problem impairs functioning.
4. Enhancing a patient's capacity to make behavioral changes, e.g. losing weight, quitting smoking, or increasing adherence to medical treatment.

COUNSELING

Definition

An interactive learning process contracted between counselor(s) and client(s), which approaches in a holistic way, social, cultural, economic and emotional issues.

Goal

1. Information.
2. Education.
3. Understanding.

Indication

It's indicated for any presenting difficulty.

Techniques

1. Listening.
2. Discussion.
3. Problem solving.
4. Enable decision-making.
5. Enable Learning.

PRIMARY HEALTH CARE BOOKLET

Screening for common cancers

كتابة: عبدالله المزيرعي، يوسف العمار، خالد العنزي / مراجعة وتعديل: عبدالسلام باقيس ، عبدالله الوهيبي

Screening for common cancers

Screening Definition:

Is a strategy used in a population to identify an unrecognized disease in individuals without signs or symptoms.

Criteria for screening: (Wilson criteria)

- **Knowledge of disease:**
  - The condition should be important.
  - There must be a recognizable latent or early symptomatic stage.
  - The natural course of the condition, including development from latent to declared disease, should be adequately understood.
- **Knowledge of test:**
  - Suitable test or examination.
  - Test acceptable to population.
  - Case finding should be continuous.
- **Treatment for disease:**
  - Accepted treatment for patients with recognized disease.
  - Facilities for diagnosis and treatment available.
  - Agreed policy concerning whom to treat as patients.
- **Cost considerations:**
  Costs of case finding (including diagnosis and treatment of patients diagnosed) economically balanced in relation to possible expenditures on medical care as a whole.

The Common Cancers should be screened in family practice Clinic:

1. Cervical cancer.
2. Breast Cancer.
3. Colon Cancer.
4. Prostate Cancer.

Box .1

Wilson criteria (using the mnemonic IATROGENIC)

- **I**mportant – the condition should be an important one
- **A**cceptable treatment for the disease
- **T**reatment and diagnostic facilities should be available
- **R**ecognizable at an early stage of symptoms
- **O**pinions on who to treat as patients must be agreed
- **G**uaranteed safety e.g. low radiation exposure
- **E**xamination must be acceptable by the patient
- **N**atural history of the disease must be known
- **I**nexpensive test
- **C**ontinuous screening i.e. not a one-off

Cervical cancer

Incidence of cervical cancer:

7. Cervical cancer is the 12th most common cancer among women females in the UK (2010), according for 2% of all new cases of cancer in females.
8. In 2010 there were 2,851 new cases of cervical cancer in the UK.
9. The crude incidence rate shows that there are around 9 new cervical cases for every 100,000 females in the UK
10. Cervical screening is not a test for cancer.
11. It is a method of preventing cancer by detecting and treating early abnormalities which, if left untreated, could lead to cancer in a woman's cervix.
12. Early detection and treatment can prevent 75 per cent of cancers developing but like other screening tests, it is not perfect. It may not always detect early cell changes that could lead to cancer.

So, how to screen?

Liquid-based cytology (LCB) is the method used to obtain cervical sample for examination in the laboratory.

Who is eligible for cervical screening? (Taple.1)

Table .1/ Recommended frequency of screening in each age group :

Age group	Frequency of screening
25	First invitation
25-49	Every 3 years
50-64	Every 5 years
65+	Only screen those who have not been screened since age 50 or have had recent abnormal tests

HPV and Cervical Cancer:

Certain HPV types are classified as "high-risk", The most common of the high-risk strains of HPV are types 16 and 18, which cause about 70% of all cervical cancers

Symptoms of High-Risk HPV Infection and Tests:

1. When infection with high-risk HPV types occurs, there are usually no symptoms.
2. The first clue is abnormal Pap test (Pap smear).
3. If the Pap test results are unclear, order a HPV test to check the DNA type of the virus.
4. It does not identify cancer. But it tells she has a type of HPV capable of causing cancer.
5. The Gardasil HPV vaccine protects against several high-risk strains of HPV, including HPV types 16 and 18.
6. Cervical screening aims to detect early abnormalities of the cervix (intraepithelial neoplasia), which, if untreated, could lead to cancer of the cervix.
7. Cervical screening is not a test for cancer.
8. Regular cervical screening prevents about 75% of cancers from developing.

Colon cancers

Incidence & prevalence:

1. About one in 20 people in the UK will develop Colon cancer during their lifetime.
2. It is the third most common cancer in the UK
3. It is the second leading cause of cancer deaths, with over 16,000 people dying from it each year.

Risk factors **(Box.2)**

Screening: **(**by fecal occult blood FOB**)**

1. Every two years if still within the eligible age range for routine screening.

2. If the result is abnormal, they will be referred for a colonoscopy.
3. If the result is unclear, FOB test will need to be repeated.

Fecal occult blood test:

1. Fecal occult blood (FOB) test works by detecting tiny amounts of blood which cannot normally be seen in colon motions.
2. The FOB test does not diagnose Colon cancer, but the results will indicate whether further investigation (usually a colonoscopy) is needed.
3. **Fecal occult blood = screening test for colon cancer**
4. **Colonoscopy = diagnostic test for colon cancer**

What is a FOB test kit?

The test kit is used to collect samples of colon motions (feces) which are then analyzed to detect tiny traces of blood, invisible to the naked eye.

Advantages and disadvantages of FOB (taple.2)

Box .2

Risk factors for colon cancer	
• over age of 60 year • A previous Colon polyp • Personal history of IBS • Diet: high fat & red meat, low vegetables, folate and fibre • Lack of exercise	• Obesity • Family history • Smoking and alcohol • Personal history of colon cancer

Table .2/ Advantages and disadvantages of FOB:

	Advantages	**Disadvantages**
	Screening can detect Colon cancer at its earliest stage.	Screening may not prevent cancer.
	With regular screening, fewer people die from colon cancer.	FOB test may not always be reliable
	Removing polyps that are discovered through screening can reduce the chances of developing colon cancer.	There are risks associated with having a colonoscopy.
		Colon cancer can start to develop in the two years between screening tests.

Prostate cancers

Incidence & prevalence:

1. Prostate cancer is the most common cancer in men in the UK and accounts for around a quarter of all new male cancer diagnoses.
2. In 2009, there were 34,593 new cases of prostate cancer diagnosed in England, an age-standardized rate of 107.6.

Screening tests for prostate cancer:

1. **Prostate specific antigen (PSA) test**
   a. it is a blood test to check the level of PSA in blood. Most healthy men have levels under 4 nanogram per milliliter of blood (taple.3).
   b. The PSA test is currently the best method of identifying localized prostate cancer
   c. BPH & prostatitis are causes of high PSA
2. **Digital rectal examination:** Doctor inserts a gloved, lubricated finger into the rectum to feel for any bumps or hard areas on the prostate that may need to be tested for cancer. **This test may be done with the PSA or the PSA may be done alone.**
3. if the results of the PSA ± DRE suggest that having a prostate cancer, do a prostate biopsy to find out.

Table .3/ PSA cut-offs in different age groups:

Age group	PSA cut-off
50-59	≥3
60-69	≥4
70 or over	>5

To screen or not to screen?

1. There is controversy about which screen-detected lesions will become clinically significant. Current methods of screening involve measurement of PSA, followed by Transrectal ultrasound scanning and biopsy, but these lack adequate specificity and sensitivity. There are three major treatment options for localized disease: radical prostatectomy, radical radiotherapy, and monitoring with treatment if required.
2. There is no randomized controlled trial evidence to suggest a survival advantage of any of these treatments, and each has risks.

Breast cancer

Incidence

1. Worldwide in 2008, there were 1,383,500 estimated new cases of breast cancer .
2. Breast cancer accounts for 23 percent of all cancer cases, 14 percent of cancer deaths, and is the second leading cause of cancer death in women.
3. In the United States, it is estimated that approximately 234,580 women will be diagnosed with invasive breast cancer, and 40,030 women will die from the disease in 2013

Risk Group:

1. Healthy women aged 50–70 years are eligible for routine breast screening.
2. Women at increased risk of breast cancer (such as with a strong family history of breast cancer) may be eligible for breast screening before 47 years of age.
3. Hormone Replacement Therapy (HRT):
   Long-term use of combined estrogen and progesterone increases the risk of breast cancer. This risk seems to return to that of the general population after discontinuing them for five years or longer.
4. Increase Menstrual periods: either by early menrach or late menopuse:
   Women who have had more menstrual cycles because they started menstruating early (before age 12) and/or went through menopause later (after age 55) have a slightly higher risk of breast cancer. The increase in risk may be due to a longer lifetime exposure to the hormones estrogen and progesterone.

Mammography: screening test for breast cancer

1. Breast screening uses mammography (radiography) to find small changes in the breast before there are any other signs or symptoms of breast cancer.
2. **What happens at the screening unit?**
   a. The breasts are X-rayed one at a time.
   b. The breast is placed on the X-ray machine and gently but firmly compressed with a clear plate. Two X-rays are taken of each breast at different angles.
   c. Most women find the compression uncomfortable and occasionally it may be painful. However, the compression is necessary to ensure the mammogram is clear. Any discomfort will be over quickly.
3. **What are the benefits and harms of mammography?**

The benefits include:

a. Reduction in mortality. Breast screening detects breast cancers and saves lives, with the greatest reduction in mortality seen in women 50–70 years of age.
b. More breast-conserving treatment due to an increase in the early detection of breast cancer.

The harms include:

a. Over-diagnosis leading to unnecessary treatment. (Over-diagnosis refers to the detection of breast cancers through screening that would not have been diagnosed without screening and would not have threatened the lives of the women concerned.)
b. False-positive mammograms leading to unnecessary further investigations.
c. False reassurance due to missed cancer and incorrect diagnosis.
d. Pain and discomfort due to mammography.
e. Psychological distress.
f. Radiation exposure, which may increase the risk of breast cancer.

PRIMARY HEALTH CARE BOOKLET

Bronchial Asthma

كتابة: هاشم المحمود ، حسام الرزقان / مراجعة وتعديل : عبدالله الوهيبي

Bronchial Asthma

Definition

It's a chronic disorder of the airways, characterized by variable reversible and recurring symptoms related to airflow obstruction, bronchial Hyper-responsiveness, and an underlying inflammation.

Pathophysiology

Asthma is a chronic inflammatory disease with a variable course characterized by episodic attacks of acute inflammation. Acute inflammation in asthma is associated with bronchoconstriction, plasma exudation/edema, vasodilatation, and mucus hypersecretion. Chronic inflammation in asthma is associated with subepithelial fibrosis, smooth-muscle hyperplasia/hypertrophy, mucus gland hyperplasia, and new-vessel formation. If asthma remains uncontrolled or poorly controlled, the underlying chronic inflammation may lead to structural changes (remodelling) that reduce the extent of airway response to therapy.

Asthma triggers

1. Allergens: food, pollens, molds, dust mites, and pet dander.
2. Irritants: tobacco smoke, smoke from wood-burning, perfumes, cleaning agents.
3. Physiological factors: stress, gastroesophageal reflux (GERD), respiratory infection (viral, bacterial) and rhinitis.
4. Pharmacological factors: aspirin or other NSAIDs, beta blockers and sulfites.
5. Physical factors: exercise, hyperventilation, cold air.

Exercise induced asthma (EIA)

1. Asthma symptom that induced by exercise.
2. People with exercise-induced asthma are believed to be more sensitive to changes in the temperature and humidity of the air.
3. Symptoms usually begin about 5- 10min after finishing exercise.
4. Rarely starts during the exercise.
5. Typically disappear within an hour, but they may last longer.
6. It can happen under any weather but cold and dry weather is the most common.

How to Approach a Patient with Bronchial Asthma

5. **Obtain a detailed History**
   - Analyze the chief complaint.
   - Associated symptoms :

1- Coughing (hemoptysis , sputum)
2- Wheezing
3- Chest tightness
4- Hoarseness and hyperventilation
5- shortness of breath

- Risk Factors: (TB , Traveling , Animal contact , smoking)
- Past medical and surgical and allergic history (history of atopy: allergic rhinitis or eczema) and medication history
- Family and social history
- Ask about:
  1- Frequency of attacks and what he did to control it
  2- recurrent attacks of wheezing
  3- cough patterns (early morning , late at night)
  4- wheezing or coughing after: (exercise or on exposure to dusts or smoke)
  5- worsening of symptoms when taking aspirin or NSIDs or B-blockers
  6- Family history of asthma or other or other atopic conditions, such as eczema or allergic rhinitis?
  7- if he experience wheezing, chest tightness, or cough after exposure to pollens, dust, feathered or furry animals, exercise, viral infection, or environmental smoke (cigarettes, burning incense "Bukhoor", or wood?
  8- Patient's cold "goes to the chest" or take more than 10 days to clear up?
  9- Hospitalization due to asthma or any other lung disease
  10- Improvement of symptoms after treatment
  11- Admission to the ICU due to asthma? Ever been intubated?

6. **Physical Examination**
   - **Inspection:** Shape, Breathing, Deformities, Scars, Apex beat, Skin changes & hair distribution.
   - **Palpation:** Apex beat, Tracheal deviation, Tracheal tug, Tactile vocal fermitus, Rib fracture, Chest expansion.
   - **Percussion:** Over the supraclavicular area, clavicle and intercostals space.
   - **Auscultation:** Breathing sound, Added sounds, vocal resonance.
   - Possible Findings :
     1- Wheeze /Rhonchi.
     2- Tachypnea.
     3- Use of accessory muscles of respiration.
     4- Paradoxical pulse (an exaggerated fall in systolic blood pressure during inspiration).

5- Prolonged expiratory phase
6- Cyanosis of nails.
7- Signs of allergy in skin, nose, eyes.

Investigation

1. **Pulmonary Function Tests**
   a. **Spirometry Test:** The most reliable way to determine reversible airway obstruction is with spirometry, a test that measures the amount of air entering and leaving the lungs. It is now preferred over peak flow measurement for initial confirmation of obstruction of airways in the diagnosis of asthma
   b. **Peak Expiratory Flow:** Peak flow diaries may also be helpful for patients with moderate or severe asthma. They can provide an objective warning of clinical deterioration. it is useful for patients to perform themselves
   c. **Bronchial provocation Challenge Testing :** Not for everyone! only done when symptoms suggest asthma, but normal spirometry , it may cause a severe asthma attack. Also used for diagnosis of occupational asthma
2. **Other supportive tests**
   a. Arterial Blood Gases
   b. Allergy Skin test
   c. Level of specific IgE in the serum
   d. Chest X-ray

Management

1. The goal of management should be to obtain and sustain complete control.
2. Global Initiative for Asthma (GINA) 6-point plan:
   a. Educate patients to develop a partnership in asthma management (Box.2)
   b. Provide regular follow-up care
   c. Avoid exposure to risk factors (allergens…)
   d. Assess and monitor asthma severity with symptom reports and measures of lung function as much as possible
   e. Establish medication plans for chronic management in children and adults
   f. Establish individual plans for managing exacerbations
3. Poor Asthma control could be due to:
   a. Inhaler technique.
   b. Adherence to prescribed regimen.

c. Environmental changes.

d. Also consider alternative diagnoses

Table.1/ Pharmacologic medications for asthma

controller	Reliever
Drugs taken daily on long term to keep asthma under control.	Used as needed! Act to quickly reverse broncho-constriction
1. Inhaled corticosteroid: most effective. (Fluticasone, budesonide) 2. Long acting beta 2 agonist: NOT used as monotherapy. Formetrol 3. Leukotriene receptor antagonist.(mast cell stabilizer) 4. Theophylline.	1. Short acting beta 2 agonist.(salbutamol) 2. Anticholinergic (**Ipratropium)**

Symbicort is budesonide + formetrol.

Table .2/ Classification, Diagnosis and Management of Asthma

Classification	Daytime symptoms	Nighttime symptoms	Pulmonary function tests	Management strategy
Mild intermittent (Step 1)	≤2 times/week	≤2 times/month	FEV1 or PEF ≥80% of predicted; PEF variability ≤ 20%.	Short acting bronchodilators only when needed
Mild persistent (Step 2)	2-6 times/week	> 2 times/month	FEV1 or PEF ≥80% of predicted; PEF variability 20-30%.	Step 1 medications plus daily low dose inhaled steroid (or mast cell stabilizer)
Moderate persistent (Step 3)	Daily	1 time/week	FEV1 or PEF 60-80% of predicted; PEF variability ≤ 30%.	Step 2 medications plus long-acting bronchodilators and antileukotriene trial.
Severe (Step 4)	Continuous	Frequent	FEV1 or PEF ≤60% of predicted; PEF variability ≤ 20%.	Step 3 medication plus high dose inhaled steroids, systemic steroids, and other therapies.

Box .1

Special Situations in Management
1. Asthma and pregnancy: salbutamol and low doses of ICS. 2. Exercise induced asthma: SABA before exercise + warm up.

Box .2

Patients education (What we should tell asthma patients)
1. Avoid risk factors. 2. Take medications correctly. 3. Understand the difference between "controller" and "reliever" medications. 4. Monitor their status using symptoms and, if available, PEF, or ACT. 5. Recognize signs that asthma is worsening and take action. 6. Seek medical help as appropriate

Box .3

Management of Acute Asthmatic Attack:
▪ Oxygen to achieve O2 saturation >= 90%. ▪ Inhaled SABA continuously for 1 hour. ▪ Systemic corticosteroids if no immediate response.

Table.3/ Comparison between Asthma and COPD

	Asthma	**COPD**
age	Childhood or adolescence	Elderly or more advanced age
Smoking history	No common	Almost always present, significant
Symptoms	Intermittent	Progressive
Co-existing conditions	Allergic rhinitis, eczema (immunity related diseases)	CAD or osteoporosis (smoking related disease)
Inhaled steroids	Standard treatment	Not very beneficial
Forced expiratory volume in first sec (FEV1) changes	return to normal between asthma attacks or after reliever therapy	generally not reversible

PRIMARY HEALTH CARE BOOKLET

ARTHRITIS

كتابة: سعود الرميح ، باسل الصقير ، نايف السليمان / مراجعة وتعديل: باسل الصقير

Approach to patient with arthritis

Definitions:

1. **Arthropathy:** A disease or abnormality of a joint.
2. **Arthritis:** Inflammation of a joint, usually accompanied by pain, swelling, and stiffness, and resulting from infection, trauma, degenerative changes, metabolic disturbances, or other causes.

Main complaint in arthritis:

1. Pain
2. Stiffness
3. Swelling
4. Limitation of movement
5. Weakness
6. Fatigue

Important aspect in history of arthritis:

1. Duration and onset
2. Number of joint involved
3. Distribution of joint involved
4. Temporal pattern of involvement in polyarthritis
5. The presence and duration of morning stiffness
6. The presence of joint swelling
7. Extra-articular complaint
8. Associated medical illness
9. Past medical, surgical and drug history
10. Family history

The onset could be:

1. Sudden (abrupt), e.g. in trauma, gout, pseudogout and infection
2. Gradual (insidious), e.g. in rheumatoid arthritis, osteoarthritis

The duration could be:

1. Acute i.e. <6weeks, e.g. in septic arthritis, gout and pseudogout
2. Chronic i.e. >6weeks e.g. in RA, ankylosing spondylitis

The arthritis can be:

1. Monoarthritis i.e. affecting only one joint
2. Oligoarthritis i.e. affecting 2-4 joints
3. Polyarthritis i.e. affecting 5 joints or more

Examples of some joints that are frequently affected by different types of arthritis:

1. DIP:
   Usually involved in psoriatic arthritis, gout and osteoarthritis
   Always spared in RA

2. lumbar spine:
   Usually involved in ankylosing spondylitis. And Spared in RA

In polyarthritis the joints involved can be:

1. **Symmetric arthritis**
   Involvement of the same joints on each side of the body like in: RA and SLE.
2. **Asymmetric arthritis**
   Like in psoriatic arthritis, reactive arthritis and Lyme arthritis.

The temporal patterns of involvement of polyarthritis are:

1. **Migratory pattern**
   Inflammation for only a few days in each joint (eg, acute rheumatic fever, disseminated gonococcal infection).
2. **Additive pattern**
   Inflammation persists in involved joints as new ones become affected (eg. RA, SLE)
3. **Intermittent pattern**
   Episodic involvement occurs, with intervening periods free of joint symptoms (eg, gout, pseudogout, Lyme arthritis).

Sometimes arthritis is accompanied by some extra-articular manifestation like:

1. Constitutional symptoms e.g. fever in septic arthritis
2. Skin lesions e.g. in SLE, Lyme disease and psoriatic arthritis
3. Ocular symptoms like:
   a. Episcleritis and scleritis in RA
   b. Anterior uveitis in ankylosing spondylitis,
   c. Iridocyclitis in juvenile RA
   d. Conjunctivitis in reactive arthritis

Diagnosis:

You need first to decide whether the complaint is acute or chronic, then whether it is polyarthritis or monoarthritis. Then you need to differentiate between inflammatory arthritis and non-inflammatory, this table summarizes the important differences between them:

Table .2/Inflammatory VS. Non-inflammatory arthritis:

Feature	Inflammatory	Non-inflammatory
Pain (when?)	Yes (AM)	Yes (PM)
Swelling	Soft tissue	Bony
Erythema	Sometimes	Absent
Warmth	Sometimes	Absent
Aggravating factor	Rest	Movement
Relieving factor	Movement	Rest
Morning stiffness	> 30 minutes	< 30 minutes
Systemic features	Sometimes	Absent
ESR, CRP	High	Normal
Synovial fluid WBC	WBC >2000	WBC < 2000
Examples	Septic, RA, SLE, Gout	Trauma, Hemarthrosis, Osteoarthritis

Acute monoarthritis: Can be caused by:

1. Bacterial infection of the joint space (septic arthritis)
2. Crystal-induced arthritis: Gout and pseudogout.
3. Trauma.

Acute polyarthritis: are caused by:

1. Infection like: gonococcal SA , Lyme disease and some viruses
2. Non-infective like: polyarticular gout, rarly RA, SLE, psoriatic and reactive arthritis.

Chronic monoarthritis: are caused by:
-Inflammatory like: infective (lyme disease, fungal, TB), non-infective (gout, RA, SLE).
-Non-inflammatory like: osteoarthritis.

Chronic oligoarthritis too, can be caused by:
-Inflammatory process like in: ankylosing spondylitis, enteropathic arthritis and gout
-Non-inflammatory like in: osteoarthritis.

Chronic polyarthritis is also can be:
-Inflammatory like: RA, SLE and gout
-Non-inflammatory like: primary generalised osteoarthritis and pseudogout

1) Septic arthritis

1. An acute form of arthritis characterized by bacterial inflammation of a joint caused by the spread of bacteria through the bloodstream from an infection elsewhere in the body or by contamination of a joint during trauma or surgery. The joint is stiff, painful, tender, warm, and swollen. 8-27% of adults presenting with one or a few acutely painful joints.

2. ***Risk factors:***

    1. Prosthetic joint, Joint surgery
    2. Skin infection
    3. RA
    4. DM
    5. IV drug users, alcoholism
    6. Intra-articular steroid injection

3. ***Sources of infection:***

    1. Hematogenous (72%)
    2. Direct inoculation of bacteria (trauma, surgery..)
    3. Extension from existing osteomyelitis in adjacent bone.

4. ***Microbiology:***

    1. The most common worldwide is N.Gonorhea ,
       Staph aurous is the most common in our community
    2. Other G +ve
    3. G –ve (usually in trauma, IV drug users, Immunocompromised, neonates and elderly)

ž ***Clinical presentation: (acute)***

1. Hot, painful, swollen and red joint
2. Restricted movement of the joint
3. Patient usually febrile,(afebrile in elderly and Imunocompromized)
4. Usually monoarthritis. Knee (50%) , hip , wrist, ankle and other could be affected.
5. 20% oligoarthritis, polyarthritis usually in RA

ž ***Important points in history:***

1. acuteness
2. if pain is superimposed on chronic pain
3. History of joint disease or trauma

4. No. of joint affected
5. Extra-articular symptoms
6. History of vascular invasion (catheterization, iv drug..etc)
7. Sexual activity
8. Condition that could affect immune system
9. Previous surgery in this joint

ž ***Physical examination:***

1. Sign of erythema ,swelling, warmth, tenderness, effusion and limitation of active and passive ranges of motion in the affected joint
2. Note: previous finding might be muted in elderly, immunocompromised and IV drug abusers!
3. Red flag, highly associated with morbidity, or even mortality.
4. **Urgent referral to orthopedic!**

ž ***Investigation:***

1. **Joint aspiration:**
    1. Gross examination
    2. G-stain
    3. cell count
    4. Culture &Sensitivity (the gold standard!)
    5. PCR: in: N .Gonorhea
    6. Glucose & protein
2. **Blood test:**
    1. CBC
    2. ESR
    3. CRP
    4. C&S
3. **Imaging studies**:
    1. X-ray, US, CT, MRI
    2. Limited value, used to role out osteomyelitis
4. **Gonococal arthritis:** (N. gonorrheae):
   Most common cause of acute infection arthritis in young sexually active adults, if suspected, obtain cultures from appropriate mucosal surfaces in addition to the previous required investigation.

ž ***Treatment:***

1. Immediate empirical antibiotics:
   PRSP + 3rd generation cephalosporin

2. Drainage:
   Daily aspiration
   Surgical
3. Specific antibiotics after C&S

2) Osteoarthritis

5. Degenerative (non inflammatory) disorder of the articular cartilage associated with hypertrophic bone changes. The most common kind of Arthritis. Could be primary (genetics) or secondary (multifactorial).

ž ***Risk factors:***

1. Age
2. obesity
3. Gender (females are affected more than males)
4. Genetics or heredity.
5. trauma
6. metabolic (DM)
7. infectious
8. inflammatory (RA)
9. hematological (Sickle cell anemia)

ž ***History:***

1. Chief Complaint: **pain**
2. Associated with: stiffness, limitation of movement, deformity.
3. Usually chronic
4. **Mono**arthritis
5. Non symmetrical joint distribution
6. Morning stiffness less than 30 minutes
7. Pain worsened with movement and relieved by rest

ž ***Physical Examination:***

1. Bony swellings (bochards / heberden noduls)
2. deformity around joint margins (valgus or varus)
3. Joint-line tenderness.
4. Restricted range of movement.
5. Palpable coarse crepitus.
6. No sign of inflammation.

ž ***Investigations:***

1. **X – ray (most important)**
2. Decrease joint space
3. Subchondral cyst
4. Osteophytes
5. Deformity varus-vagus

6. All these findings could be seen in a patient with osteoarthritis

ž ***Management:***

1. Non pharmacological: weight reduction, physiotherapy.
2. Pharmacological: paracetamole if symptoms not relived: NSAID (oral or topical) like iboprfine.
3. In case of failure of the conservative treatment of osteoarthritis: Refer the patient to an orthopedic surgeon

3) Rheumatoid arthritis (RA):

1. It is a chronic systemic disease primarily of the joints, usually polyarticular, marked by inflammatory changes in the synovial membranes and articular structures and by atrophy and rarefaction of the bones. In late stages, deformity and ankylosis develop.
2. The annual incidence of rheumatoid arthritis (RA) has been reported to be around 40 per 100,000.

ž ***Etiology:***

ž RA is a form of autoimmunity, the causes of which are still **not completely known**, but many possible etiologies have been identified:

1. Genetic (some studies revealed a relationship between RA and HLA-DR4)
2. Hormonal
3. Infectious (viral "herpesvirus" or bacterial)

ž ***Risk factors***

ž Many factors may increase the risk of developing Rheumatoid Arthritis like:

1. **Age** (Although rheumatoid arthritis may develop at any age, it is more like to begin in people aged between 40 and 60 years).
2. **Gender** (The disease is two to three times more common in premenopausal women than in men but postmenopausal women have equal chances with men).
3. **Smoking**

ž ***Extra-articular manifestations:***

ž Rheumatoid arthritis is a systemic disease that can affect many organs and systems in the human body like:

1. Ocular: scleritis, conjunctivitis, and episcleritis.
2. Pulmonary: pleural effusion, pulmonary nodules, and interstitial lung disease.
3. Cardiac: myocarditis, ischemic heart disease, pericarditis, and arrhythmia.
4. Skin: rheumatoid nodules, and purpura.
5. Hematological: anemia of chronic disease, neutropenia, and splenomegaly.
6. And many others (physiological, endocrine, renal, skeletal ….).

ž ***Approach to patient with rheumatoid arthritis:***

ž Like any other disease the approach consist of four parts (history, physical examinations, investigations, and management).

ž ***History:***

ž There are some important questions that should be asked to any patient suspected to have any form of arthritis like:

1. **Duration** of the complaint:

Acute (less than 6 weeks) OR chronic (6 weeks or more)

2. **Number** of joints involved:

Usually rheumatoid arthritis patients have a polyarticular joint involvement.

3. **Distribution** of Joints Involved:

RA has a symmetrical joint involvement.

4. Pattern of involvement:

Inflammation persists in involved joints as new ones become affected (Additive).

5. Duration of **morning stiffness**:

Usually morning stiffness last for more than 30 minutes (it can reach one hour).

6. **Aggravating and relieving factors**:

Pain worse after a period of inactivity and relieved by movement.

7. History of joint **swelling**.
8. **Extra-articular** complaints.
9. **Family history.**

ž ***Physical examinations:***

ž One or more of these features may be seen in a patient with RA during physical examination:

1. Stiffness of the affected joint
2. Tenderness
3. Pain on motion

4. Swelling
5. Deformities
6. Limitation of motion
7. Extra-articular manifestations
8. Rheumatoid nodules (occur in approximately 25% of patients with RA)

ž ***Investigations:***

ž RA is a **clinical diagnosis**; no laboratory test is diagnostic, just supportive!

1. **Rheumatoid factor:**
   a. Auto antibodies to the Fc portion of IgG.
   b. Support a diagnosis of Rheumatoid Arthritis but **are not** by themselves diagnostic.
   c. Are seen in about **75% to 80%** of patients with RA.
   d. Are associated with a **poor prognosis** in patients with RA.
   e. Are seen in conditions other than RA like hepatitis C, sarcoidosis, pulmonary fibrosis, and many others.
2. **Anti-citrullinated protein antibodies (ACPA):**
   a. These are auto antibodies directed against an individual's own proteins (CCP) which can be detected by ELISA. These anti bodies are present in the majority of patients with RA.
   b. Accuracy (Anti-CCP Assay)
      - **Specificity 79%**
      - **Sensitivity 96-98%**
   c. Diagnosis more accurate when combined with RF+.
   d. Present in 50-60% early RA patients.

ž ***Diagnostic criteria:***

ž American College of Rheumatology (ACR)/European League Against Rheumatism (EULAR) 2010

ž ***Management:***

ž As a primary care physician you should

1. Confirm your diagnosis of RA form (history, Physical exams, and investigations).
2. As soon as the diagnosis of RA is confirmed combined care should be started:
   a. Patient Education 2.Start DMARD(s) within 3 months 3.Consider NSAIDs 4.Consider Local / Low-dose Steroid 5.Physical / Occupational Therapy.
   b. Referral to rheumatology clinic.

c. Follow up.

	Score
Target population (Who should be tested?): Patients who	
1) have at least 1 joint with definite clinical synovitis (swelling)*	
2) with the synovitis not better explained by another disease†	
Classification criteria for RA (score-based algorithm: add score of categories A–D; a score of ≥6/10 is needed for classification of a patient as having definite RA)‡	
A. Joint involvement§	
1 large joint¶	0
2–10 large joints	1
1–3 small joints (with or without involvement of large joints)#	2
4–10 small joints (with or without involvement of large joints)	3
>10 joints (at least 1 small joint)**	5
B. Serology (at least 1 test result is needed for classification)††	
Negative RF *and* negative ACPA	0
Low-positive RF *or* low-positive ACPA	2
High-positive RF *or* high-positive ACPA	3
C. Acute-phase reactants (at least 1 test result is needed for classification)‡‡	
Normal CRP *and* normal ESR	0
Abnormal CRP *or* abnormal ESR	1
D. Duration of symptoms§§	
<6 weeks	0
≥6 weeks	1

4) Gout:

1. It is an inflammatory arthritis associated with hyperuricemia and intra-articular **sodium urate crystals.**
2. Usually affect men (10:1), rare in premenopausal female.
3. The Prevalence in developed countries about 0.5 %.
4. Most common joint affected is 1st MTP joint.
5. Recurrence:
   a. 75% likelihood of a second attack within 2 years.
   b. Attacks tend to become Polyarticular.

ž ***Causes:***

ž **Hyperuricemia** is the most common cause and it could be because of:

1. Impaired excretion (90%): like renal disease, diuretics, NSAID use, and acidosis.
2. Increase production: like chemotherapy, chronic hemolysis, and blood cancers.

ž ***Risk factors:***

1. Alcohol

2. Dehydration
3. Urate stones
4. Diuretics use
5.

ž ***Clinical features (History and Physical examinations):***

1. Usually present as sudden onset severe pain.
2. Usually affects small joints.
3. swelling.
4. Erythema.
5. tenderness and warmth (signs of inflammation).
6. Middle age male, MTP of big toe

ž ***Investigations:***

1. **Joint aspiration and synovial fluid analysis** (under polarizing microscope).
   a. Needle-shape crystals negatively birefringent.
   b. Most specific diagnostic test.
2. **Serum uric acid**: Usually high, but can be normal.
3. **Plain Radiographs**: May show Bone erosions.

ž ***Management:***

1. **Asymptomatic hyperuricemia:**
   a. These patients should not be treated medically because 95% Of them remain asymptomatic.
   b. Recommend them to avoid risk factors like alcohol …etc.
2. **Acute gouty arthritis:**
   a. Bed rest. Early ambulation may precipitate a recurrence.
   b. Medications:
      1- NSAID. **Treatment of choice**, very effective. Eg: diclofenac 100 mg immediately, then 50 mg every 6-8 hours.
      2- Colchicine. Only If NSAIDs are not tolerated.
      3- Corticosteroid**.**
   c. Avoid secondary cause of hyperuricemia: Medication (thiazide), obesity, alcohol and dietary intake of purine.
   d. Consider giving prophylactic medication. If the patient had two or more attacks of gout in a year:
      - Increase the dose of NSAID
      - Or add another drug (NSAID + Colchicine)

PRIMARY HEALTH CARE BOOKLET

cardiovascular diseases and Dyslipidaemia

كتابة: سليمان الشمري ، عبدالسلام باقيس ، محمد الكربي / مراجعة وتعديل: عبدالله الوهيبي

Risk Assessment of cardiovascular diseases and Dyslipidaemia

Risk factors of CVD

1. **C-reactive protein**
    - **a.** Blood protein that signifies inflammation.
    - **b.** High levels maybe associated with an increased risk of developing (CAD).
    - **c.** 2010 ACCF/AHA guidelines state that measuring CRP can be useful for selecting patients for statin therapy & maybe reasonable for C.V risk assessment.
2. **Coronary artery calcification**
    - **a.** Coronary calcium scans use CT scan to check for calcium deposition in the coronary arteries.
    - **b.** A calcium score of zero in a person over 40 years of age indicates a 90-95% absence of significant coronary artery disease. A score of 1 to 10 is in keeping with a minimum plaque burden. A score of 11 to 100 is in keeping with a mild plaque burden; 100- 400 moderate plaque burden and over 400 extensive plaque burden with a very high likelihood of at least one significant coronary stenosis. ATP IV the cut of point of calcium score Is 300.
3. **Homocysteine**
    - **a.** Results from methionine breakdown.
    - **b.** Elevated levels have been shown to cause:
    - **c.** Atherosclerosis.
    - **d.** Venous thrombosis.
    - **e.** **Test:** Fasting homocysteine level.
        1. Less than 13 µmol/L is considered normal.
        2. Between 13 and 60 µmol/L is considered moderately elevated.
        3. Greater than 60 to 100 µmol/L is severely elevated.
    - **f.** **Management:** B6, B12 & folate supplementation decrease homocystein levels.

Cardiovascular Diseases includes Stroke, coronary heart disease, Aortic aneurysm and Peripheral artery disease

Table .1/ Risk factors of CVD:

	Non-modifiable	Modifiable	Emerging risk factors
	Age: **Males > 45** **Females > 55**	**Hyperlipidemia**	**Elevated high-sensitivity C-reactive protein**
	Male, Postmenopausal female	**Hypertension**	**Coronary artery calcification**
	Family history	**Diabetes**	**Elevated lipoprotein(a)**
	1st Degree male < 55	**Smoking**	**homocysteine**
	1st Degree female < 65	**Obesity, Metabolic syndrome**	**Fibrongin**
		Sedentary lifestyle	
		Heavy alcohol intake	

Primary prevention

1. **Diet**
    a. Advocate consumption of fruits, vegetables, low-fat dairy products, fiber, whole grains, and protein sources that are low in trans-fat, saturated fat and cholesterol.
    b. Reduced dietary sodium intake, increased consumption of fish that are high in omega-3 fatty acids decreases cardiovascular risk
2. **Weight Reduction**
    Goal: Achieve and maintain desirable weight (body mass index 18.5–24.9 kg/m2).
3. **DM**
    Goals: Normal fasting plasma glucose (<110 mg/dL) and near normal HbA1c (<7%).
4. **Physical activity**
    a. Goal: At least 30 min of moderate-intensity physical activity on most (and preferably all) days of the week.
    b. Increased physical activity begins with increasing lifestyle activities, such as walking
    c. A complete exercise program.
    d. More frequent exercise, provide more benefits.
5. **Blood Pressure**
    a. Goal:
        i. BP <140/90 mm Hg; or
        ii. BP < 140/80 mm Hg if the patient has DM or CKD.
    b. Initiate or maintain lifestyle modification, weight control, increased physical activity, sodium reduction.

c. For patients with BP ≥140/90 mm Hg (or 140/80 mm Hg for individuals with chronic kidney disease or diabetes):
d. Add BP medication, initially with beta-blockers and/or ACE inhibitors, with addition of other drugs (ex, Diuretics).

6. **Smoking cessation.**

Chest pain

Box .1

Chest pain (systems that can cause chest pain)
1- Musculoskeletal 2- Gastrointestinal 3- Cardiovascular 4- pulmonary

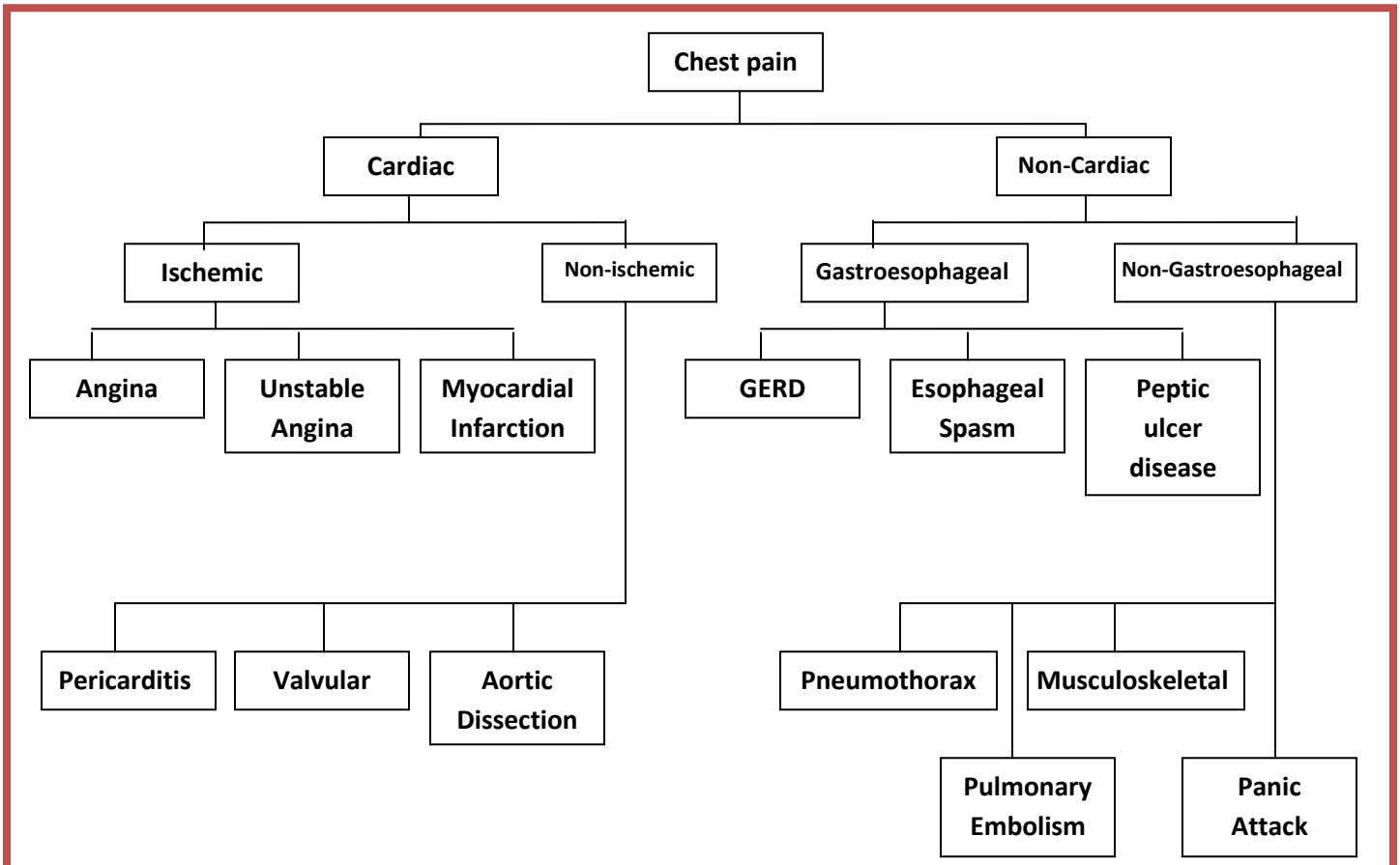


Figure.1 Differential Diagnosis of chest pain

Ischemic heart disease

Definition

1. An imbalance between the supply of oxygen and the myocardial demand resulting in myocardial ischemia.
2. **Angina pectoris**: Symptom not a disease Chest discomfort associated with abnormal myocardial function in the absence of myocardial necrosis.
3. **Myocardial infarction:** A clinical (or pathologic) event caused by myocardial ischemia in which there is evidence of myocardial injury or necrosis.

Presentation

1. Band like chest pain around the chest or central chest.
2. Pressure/dull ache with/without radiation to shoulders, arms (L>R), neck, and/or jaw.
3. Often associated with nausea, sweating, and/or shortness of breath.
4. REMEMBER (Patients may have no pain and may only complain of episodic shortness of breath, weakness, dizziness, collapse, sweating or nausea and vomiting).
5. Atypical symptoms do not necessarily rule out Acute Coronary Syndrome.

Post Myocardial Infarction management

1. **Life style and risk factors modification:**
   a. In DM and chronic kidney disease intensive interventions are recommended for patients with established CHD
   b. Smoking cessation
   c. Physical activity
   d. Weight management
   e. Blood pressure
2. **Medications:**
   a. Lipid management
   b. Antiplatelet and anticoagulation therapy
   c. Aspirin
   d. Clopidogrel
   e. Warfarin therapy
   f. Renin-angiotensin-aldosterone system inhibitors
   g. Angiotensin converting enzyme (ACE) inhibitors
   h. Angiotensin receptor blockers (ARBs)
   i. Aldosterone blockade

j. Beta blockers

3. **Family education**
   a. Review with patients and families how to recognize symptoms and what should they do?
   b. cardiopulmonary resuscitation (CPR) training
4. **Others :**
   a. Recommended:
      - give annual influenza vaccination
      - screen for depression
   b. Not recommended:
      - Hormonal therapy
      - vitamin E and/or vitamin C supplements
      - folic acid (with or without vitamins B6 and B12)

Dyslipidemia

1. Types of cholesterol in the body
   a. High-density lipoprotein (HDL)
   b. Low-density lipoprotein (LDL)
2. High LDL-C levels are known to increase the risk of heart disease and stroke.
3. Other important lipid abnormalities include:
   a. Low HDL-C (at any given LDL or TC level, reduced HDL-C is associated with an increased CHD risk)
   b. Elevated Triglycerides (independent predictors of CV disease)
4. Fasting lipid profile:
   a. Total Cholesterol
   b. LDL
   c. HDL
   d. TGs
5. While in non-fasting state you can measure only :
   a. Total cholesterol
   b. HDL
6. The ratio of total cholesterol/HDL-C has been shown to be the optimal predictor of CVD risk.
7. According to the American Heart Association (AHA), you should keep your cholesterol ratio at or below 5:1. The ideal cholesterol ratio is about 3.5:1.

Causes of Hypercholesterolaemia:

1. **Primary Causes:**
   a. Diet, Obesity, Sedentary life

b. Genetic, heterozygous and homozygous familial hypercholesterolaemia

2. Secondary Causes:

c. Hypothyroidism
d. Nephrotic Syndrome
e. Obstructive jaundice
f. Diabetes
g. Drugs: Steroids, Oestrogens, Progestins, Retinoic A.

Management:

1. Primary goal is to achieve target LDL level (Table.3)
2. Non HDL level is secondary goal
3. Very high TGs > 500 → aim is to prevent acute pancreatitis; Rx: low fat diet, weight reduction, Physical activity, Fibrate or Nicotinic acid
4. HDL is the tertiary target
5. **Pharmacological management:**
   a. LDL lowering drugs:
      - Statins (Simvastatin, Atorvastatin)
      - Ezetimibe (Zetia®) =decrease absorption of cholesterol.
   b. Non-HDL lowering drugs:
      - Nicotinic acid
      - Fibrates [Gemfibrozil]
      - Omega-3- Fatty acids (reduce mortality in patients with CAD by approximate 20% and 40%).

Table.2/ATP III Classification of LDL, HDL and total cholesterol levels (mg/dL)

LDL Cholesterol	
Optimal	**<100**
Near optimal	**100-129**
Borderline high	**130-159**
high	**160-189**
Very high	**≥ 190**
Total Cholesterol	
Desirable	**<200**
Borderline high	**200-239**
high	**≥240**
HDL Cholesterol	
Low	**<40**
High	**≥60**

Table.3/ Risk Categories that can modify LDL goals.

Risk Category	LDL goal
CHD and CHD risk equivalents (Box.2)	**<100**
>2 risk factors (Box.3)	**<130**
0-1 risk factor	**<160**

Box .2

CHD equivalents
• Clinical CHD • Symptomatic carotid artery disease • Peripheral arterial disease • Abdominal aortic aneurysm. • DM

Box .3

Major risk factors (risk factors that modify LDL goals)
• Cigarette smoking • Hypertension • HDL <40 mg/dL • Family history (1st degree) of premature CHD (<55 years in males, <65 years in females) • Age (males >45 years; Females >55 years)

Box .4

Framingham Risk score (To calculate risk for 10 years u have to have these five categories):
• AGE • TOTAL CHOLESTEROL LEVEL • HDL-C LEVEL • SMOKING • Systolic Bp

Drugs Affecting Lipoprotein Metabolism

Drug Class	Agents and Daily Doses	Lipid/Lipoprotein Effects		Side Effects	Contraindications
HMG CoA reductase inhibitors (statins)	Lovastatin (20-80 mg) Pravastatin (20-40 mg) Simvastatin (20-80 mg) Fluvastatin (20-80 mg) Atorvastatin (10-80 mg) Cerivastatin (0.4-0.8 mg)	LDL HDL TG	↓18-55% ↑5-15% ↓7-30%	Myopathy Increased liver enzymes	Absolute: • Active or chronic liver disease Relative: • Concomitant use of certain drugs*
Bile acid sequestrants	Cholestyramine (4-16 g) Colestipol (5-20 g) Colesevelam (2.6-3.8 g)	LDL HDL TG	↓15-30% ↑3-5% No change or increase	Gastrointestinal distress Constipation Decreased absorption of other drugs	Absolute: • dysbetalipoproteinemia • TG >400 mg/dL Relative: • TG >200 mg/dL
Nicotinic acid	Immediate release (crystalline) nicotinic acid (1.5-3 gm), extended release nicotinic acid (Niaspan®) (1-2 g), sustained release nicotinic acid (1-2 g)	LDL HDL TG	↓5-25% ↑15-35% ↓20-50%	Flushing Hyperglycemia Hyperuricemia (or gout) Upper GI distress Hepatotoxicity	Absolute: • Chronic liver disease • Severe gout Relative: • Diabetes • Hyperuricemia • Peptic ulcer disease
Fibric acids	Gemfibrozil (600 mg BID) Fenofibrate (200 mg) Clofibrate (1000 mg BID)	LDL *(may be increased in patients with high TG)* HDL TG	↓5-20% ↑10-20% ↓20-50%	Dyspepsia Gallstones Myopathy	Absolute: • Severe renal disease • Severe hepatic disease

* *Cyclosporine, macrolide antibiotics, various anti-fungal agents, and cytochrome P-450 inhibitors (fibrates and niacin should be used with appropriate caution).*

Table 7. Drugs Affecting Lipoprotein Metabolism

Drug Class, Agents and Daily Doses	Lipid/Lipoprotein Effects		Side Effects	Contraindications	Clinical Trial Results
HMG CoA reductase inhibitors (statins)*	LDL HDL TG	↓18-55% ↑5-15% ↓7-30%	Myopathy Increased liver enzymes	Absolute: • Active or chronic liver disease Relative: • Concomitant use of certain drugs†	Reduced major coronary events, CHD deaths, need for coronary procedures, stroke, and total mortality
Bile acid Sequestrants‡	LDL HDL TG	↓15-30% ↑3-5% No change or increase	Gastrointestinal distress Constipation Decreased absorption of other drugs	Absolute: • dysbetalipoproteinemia • TG >400 mg/dL Relative: • TG >200 mg/dL	Reduced major coronary events and CHD deaths
Nicotinic acid¥	LDL HDL TG	↓ 5-25% ↑15-35% ↓20-50%	Flushing Hyperglycemia Hyperuricemia (or gout) Upper GI distress Hepatotoxicity	Absolute: • Chronic liver disease • Severe gout Relative: • Diabetes • Hyperuricemia • Peptic ulcer disease	Reduced major coronary events, and possibly total mortality
Fibric acids§	LDL *(may be increased in patients with high TG)* HDL TG	↓5-20% ↑10-20% ↓20-50%	Dyspepsia Gallstones Myopathy Unexplained non-CHD deaths in WHO study	Absolute: • Severe renal disease • Severe hepatic disease	Reduced major coronary events

* *Lovastatin (20-80 mg), pravastatin (20-40 mg), simvastatin (20-80 mg), fluvastatin (20-80 mg), atorvastatin (10-80 mg), cerivastatin (0.4-0.8 mg).*
† *Cyclosporine, macrolide antibiotics, various antifungal agents and cytochrome P-450 inhibitors (fibrates and niacin should be used with appropriate caution).*
‡ *Cholestyramine (4-16 g), colestipol (5-20 g), colesevelam (2.6-3.8 g).*
¥ *Immediate release (crystalline) nicotinic acid (1.5-3 g), extended release nicotinic acid (Niaspan ®) (1-2 g), sustained release nicotinic acid (1-2 g).*
§ *Gemfibrozil (600 mg BID), fenofibrate (200 mg), clofibrate (1000 mg BID).*

تنسيق: يوسف المانع

متابعة وإشراف: يوسف المانع ، رائد الموسى