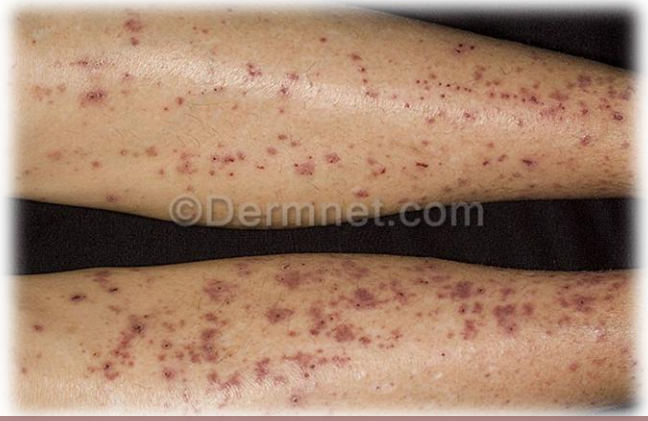
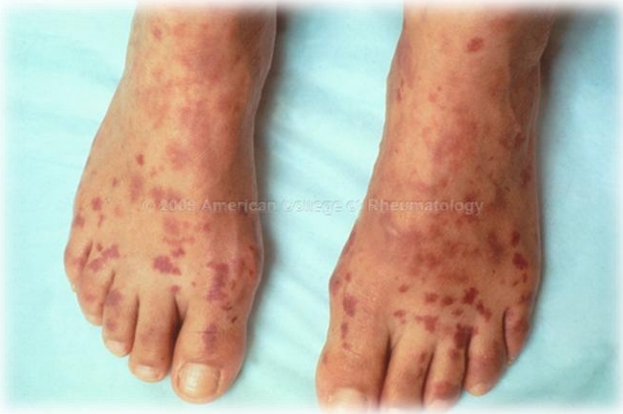




Fifth Lecture

Vasculitis

*432 **Pathology** Team*

Done By: Abrar AlFaifi _ Ethar Alqarni

Reviewed By: Abdulmajeed Aljasser

Extra info

Veins' vasculitis (phlebitis)

Arteries Vasculitis(arteritis)

Vasculitis: Inflammation of the blood vessels often associated with necrosis of the walls of blood vessels.

Classification of Vasculitis according to:

According to the cause:

1) Immune mediated

- Immune complex deposition
- ANCA: anti-nuetrophils cytoplasmic antibodies
- AECA: anti-endothelial cells antibodies

2)Infectious pathogens : invade the walls of the blood vessels

3)Physical and chemical Injury : Trauma,drugs,Toxins

#According to the size of the vessel affected:

Large vessel: (Granulomatous diseases) Takayasu's arteritis, Temporal arteritis (Giant cell arteritis)

Medium vessel:, Kawasaki disease, Polyarteritis nodosa (PAN)

Small vessel:, Microscopic polyangiitis(no asthma no granuloma), Wegener's granulomatosis(with Granuloma , No Asthma) , Churg–Strauss syndrome (with asthma,with Granuloma and Eosinophilia)

#According to the location of the affected vessels : limited to skin, Muscles, .. etc (additional wasn't mentioned in the slides)

How do we detect ANCA?

(for understanding) What are ANCAs ?

Anti-neutrophil cytoplasmic antibodies (ANCAs) are a group of autoantibodies against antigens in the cytoplasm of neutrophil granulocytes (the most common type of white blood cell) and monocytes. They are detected as a blood test in a number of autoimmune disorders, but are particularly associated with systemic vasculitis, so called ANCA-associated vasculitides.

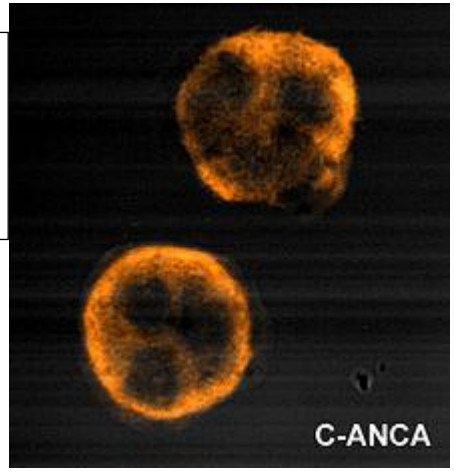
Immunofluorescence (IF) is used to detect ANCA, it may be used to help **differentiate ANCA patterns**.

ANCA can be divided into four patterns when visualised by IF (doc shaiesta only explained two which are mentioned in the slides):

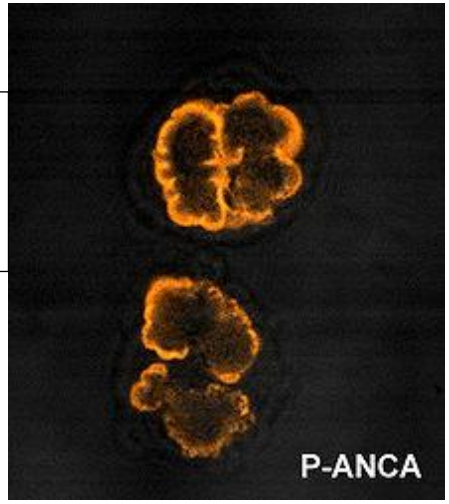
cytoplasmic ANCA (c-ANCA) : autoantibodies target proteinase-3 (PR-3)>> present with Wegener's syndrome

perinuclear ANCA (p-ANCA): autoantibodies target for myeloperoxidase (MPO)>>> present in Microscopic polyangitis and Churg-strauss sydrome

C-ANCA: the stain is distributed and you can find it surrounding the cell and also inside the cytoplasm



P-ANCA: the stain is more concentrated around the nuclear, as if it's surrounding it



Type of Vasculitis	Giant Cell(Temporal) Arteritis	Poyarteritis Nodosa
Nature	Chronic, typically granulomatous inflammation	Systemic involvement
Size/Type of vessels	Large-small arteries	Small or medium-sized muscular arteries (But not arterioles, capillaries, or venules)
Age	+50 old	young adults
Etiology	Immune origin,T cell mediated	Immune complex mediated
Symptoms	• Fever,Fatigue,weight loss • facial pain ,headache • Pain most intense along the superficial temporal artery, which is painful to palpation	episodic, with long symptom-free intervals • Fever and weight loss • Renal (arterial) involvement • Hypertension (developing rapidly) • Abdominal pain and melena (bloody stool) • Diffuse muscular aches and pains • Peripheral neuritis
clinical features	Nodularity, thickness and firm vessel	• the clinical findings may be varied and puzzling . • The course can vary from acute to chronic .
Microscopic changes	• Disruptions of the elastic lamina with inflammation and giant cells. • granuloma on the internal elastic lamina.	• Mixed infiltrate of ✓ Neutrophils ✓ eosinophils ✓ mononuclear cells • fibrinoid necrosis
Diagnosis depend on	• biopsy of an adequate segment • histological confirmation	Biopsy
Other information	- especially the Temporal arteries - Rarely affects the aorta (giant cell aortitis)	1. involve renal and visceral vessels but spare pulmonary circulation. 2. All stages from early to latemay coexist in **different** vessels or within the **same** vessel 3. No association with ANCA
associated with	__	30% have **hepatitis B antigenemia**
treatment	**corticosteroids**	

Type of Vasculitis	Wegener's granulomatosis	Churg-Strauss Syndrome	Microscopic polyangiitis
Nature	Necrotizing or granulomatous vasculitis **(necrotizing granulomas)**	• Eosinophil-rich and granulomatous inflammation • necrotizing vasculitis affecting small vessels	Pauci immune necrotizing vasculitis
Size/Type of vessels	small to medium-sized vessels	medium-sized vessels	small vessels
Age	40-50 years	—	—
Etiology	**(C-ANCA)**	**(P-ANCA)**	• **(P-ANCA)** • **an** antibody **response to antigens such as :** 1. drugs (e.g., penicillin) 2. microorganisms (e.g., streptococci) 3. heterologous proteins **4.** tumor proteins
clinical features	involving the respiratory tract	involving Lung (respiratory tract) and splenic vessels	• Hemoptysis • Hematuria and proteinuria • Bowel pain or bleeding • Muscle pain or weakness • Palpable cutaneous purpura
Microscopic changes	granulomatous inflammation including : • epithelioid cells • giant cells	—	• fibrinoid necrosis • extravasation of RBCs • neutrphilic infiltration with debris (leukocytoclasis) .
Other information	—	—	tend to be of the same age in any given patient
associated with	glomerulitis	asthma and eosinophilia	**Necrotizing glomerulonephritis** (90% of patients) and **pulmonary capillaritis**
treatment	**Corticosteroids , Immunosuppression**		

Questions:

1/ With what is PAN associated ?

Hepatitis B infection

2/ What is the most effective treatment ?

Steroids

3/ What are the characteristic serum markers for the following disorders :

Wenger granulomatous : c-ANCA

Churg-strauss : Eosinophil

4/ How is the temporal arteritis diagnosed ?

By history and physical exam. Definitive diagnosis is by temporal artery biopsy.

هذا العمل لا يغني عن المحاضرة الرئيسية , الأفضل الاطلاع عليه بعد المذاكرة عشان تكون الفكرة واضحة

بالتوفيق للجميع ^^