King Saud University
College of Medicine
Department of Medical Education

"... as dark as coffee ground"

Tutorial TWO

Year Two, GASTROINTESTINAL & HAEMATOLOGY Block

Curriculum Development Unit

Student's Case

Case 3; 2013

The Template of the PBL Cases is designed by Professor Samy A. Azer.

The Student Case and Tutor Guide are created by
Professor Samy A. Azer
Dr Othman Al Harbi
Dr Reem Sallam
Dr Rana Hassanato
Dr Ahmed Al-Humidi

Tutorial 2:
Discussion of Learning Issues (60 minutes)

Students: You should start by discussing your "learning issues" that you have identified at the end of tutorial one. You might spend about **60 minutes** on this task. A scribe on the whiteboard is needed to help in this process.

Once you have completed the discussion of your "learning issues", you might progress to these questions. Spend about **10 minutes** on discussing them in your group. A scribe on the whiteboard will help in this process.

Discussion Questions:

1. What is your final hypothesis? Give evidence.
2. What could possibly be the mechanisms underlying Saif's presenting problems?
3. What should the doctor do at this stage?

4. Do you know a Nobel prize laureate whose work has contributed to the advancement of our knowledge in physiology and/or pharmacology related to this case? What was exactly his/her work about? Give a summary.

   Student: You could also after the completion of this case submit your work about the Nobel Prize laureate for this case to Professor Samy Azer at (sazer@ksu.edu.sa) or hand it to him.

Progress 1 (30 minutes)

The doctor in the Accident & Emergency Department inserts two IV cannulas into Saif's median cubital veins, one cannula in each arm. The patient is given intravenous fluids to expand his plasma volume until he receives two blood units from the blood bank. He also sends blood to the laboratory for blood workouts (complete blood count, liver function tests, blood urea, creatinine and electrolytes, serology viral screening tests) and contact a gastroenterologist to review Mr Saif. The gastroenterologist reviews Mr Saif case, checks that Saif has received needed emergency management options, focuses on correcting the circulatory haemodynamic changes , and prepares Saif for an urgent gastroscopy to stop any further bleeding. The blood test results become available and are shown in the tables below:

Complete Blood Count:

Blood Test	Saif's results	Normal range
Haemoglobin	8.3	11.5-15.5 g/100ml
White blood cell count	9 x 10^9 /L	4-11 x 10^9/L
Packed cell volume (PCV)	49	37-47%
Mean corpuscular volume (MCV)	98	80-96 fl
Platelet count	89,000	160,000-500,000 mm^3

Liver Function Tests

Test	Saif's Results	Normal Range
Serum bilirubin	83	0-19 µmol/L
Aspartate Aminotransferase (AST)	72	0-40 IU/L
Alanine Aminotransferase (ALT)	59	0-50 IU/L
Alkaline Phosphatase (ALP)	175	0-120 IU/L
Gamma Glutamyltranspeptidase (γ- GT)	109	0-50 IU/L
Serum albumin	28	35-50 g/L
Prothrombin time	20 seconds	10-14 seconds

Serology Screening Tests:

- Hepatitis C virus: positive
- Hepatitis B virus: negative
- Hepatitis A virus: negative

Blood Biochemistry

Blood Test	Saif's Results	Normal Range
Serum Sodium	132	135-146 mmol/L
Serum Potassium	3.9	3.5-5.0 mmol/L
Blood urea	2.1	2.5-6.7 mmol/L
Serum Creatinine	120	79-118 µmol/L
Vitamin B_{12}	360	100-520 pmol/L
Folate- red blood cells	310	160-640 µg/L

Ultrasound:

- The liver surface is not smooth, it shows nodularity
- No thrombosis in the portal vein or intrahepatic veins.
- No masses suggestive of liver malignancy.
- There is a free fluid in the peritoneal cavity (ascites).

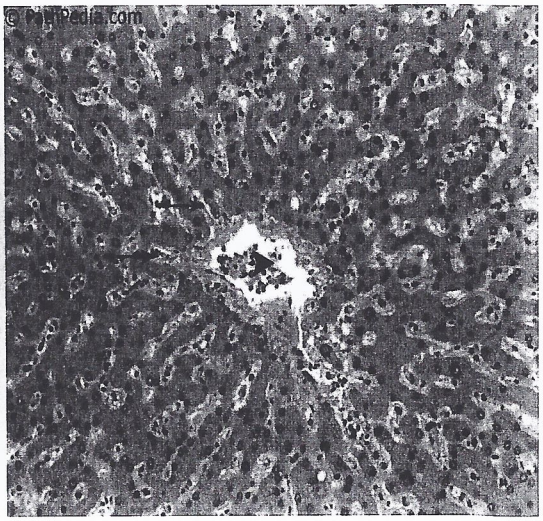
Liver Pathology:

Normally we do not recommend liver biopsy because of his prolonged prothrombin time and bleeding tendency. A liver biopsy is contraindicated in such situations. After correcting his coagulopathy, a liver biopsy was performed under ultrasound guidance. A normal liver histology is shown for comparison.

Normal liver histology (image)

Low power

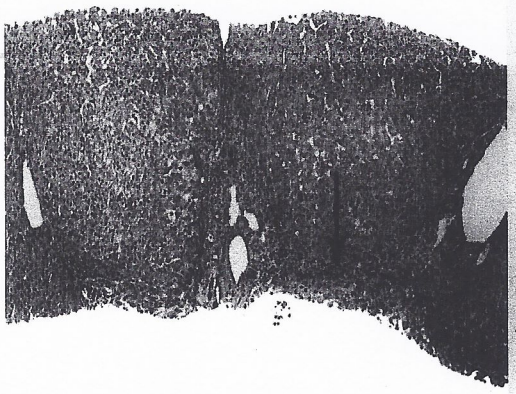
High power



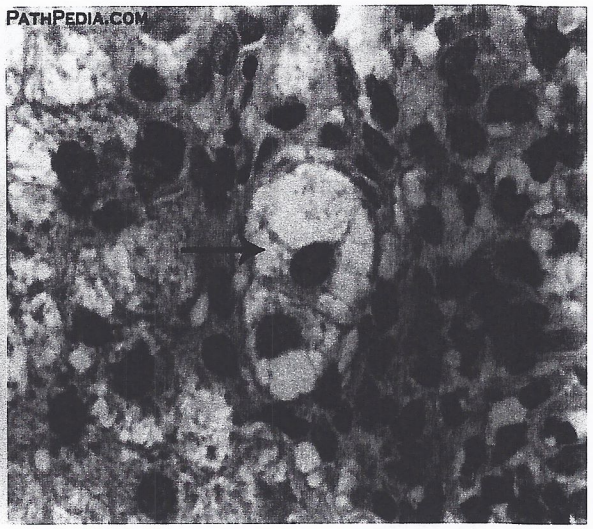
Source: pathpedia.com

Saif's biopsy images (image)

Low power



High power



Source: pathpedia.com

Report:

Low power: There is a general liver architectural distortion caused by hepatocellular necrosis with repair in the form of bridging fibrosis (arrow) which extend from one portal tract (number 1) to another portal tract (number 2).

High power: Evidence of inflammatory cellular injury confirmed by ballooning enlargement of hepatocytes (called ballooning degeneration), see black arrow. These changes although non-specific, they are commonly seen in viral hepatitis C infection.

Discussion Questions:

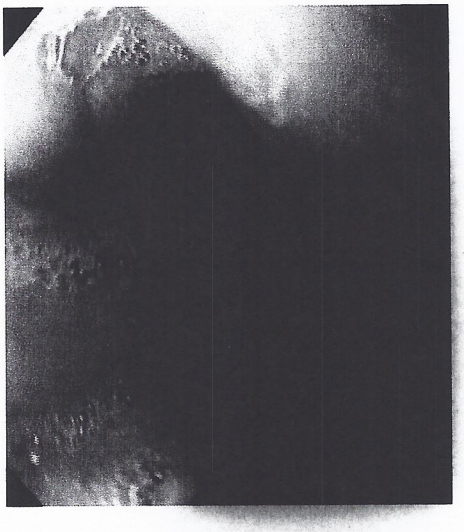
- Are there words that you do not understand?
- Summarize key information that you have obtained from this progress.
- On the basis of the new information, what is your final hypothesis?
- Summarise your management goals and your management options?.

Progress 2 (20 minutes)

The gastroenterologist examines Mr Saif , and reviews all blood test results. He confirms the diagnosis of liver cirrhosis, and portal hypertension, as evidenced by dilated abdominal veins, splenomegaly, and the presence of ascites. He mentions to Mr Saif that he needs an urgent gastroscopy to stop the blood which is most likely from bleeding dilated veins at the end of the oesophagus (the food pipe). At the mean time, he arranges to give him octrotide to decrease the pressure of the portal vein and the tendency for bleeding.

The garstorscopy images below show the lower end of the oesophagus before and after banding.

Before banding



After banding

Source: http://www.gastrohep.com/register.asp

During the gastroscopy procedure, the gastroenterologist identifies the source of bleeding and bands the bleeding oesophageal varices. He ensures that the bleeding has stopped.

Saif is treated in hospital for a few days. Before hospital discharge, the gastroenterologist explains to Mr Saif the nature of his illness and that his liver problem (medically known as liver cirrhosis) is most likely caused by hepatitis C virus infection. This might has happened many years ago and the virus has gradually caused a wide range of changes in the liver causing loss of its cells and hence deterioration of the liver functions. The changes in the liver also resulted in blocking the normal blood circulation in the liver and forced the blood to shift into the systemic circulation via other veins outside the liver, these veins are normally not functioning. This explains the dilated abdominal veins, and the dilated veins at the end of the oesophagus and the bleeding from the oesophageal veins and blood loss.

The gastroenterologist discusses with Saif that he might need a liver transplant ion and arranges for him to review the team at King Faisal Specialised Hospital, Liver Centre . In the mean time, the gastroenterologist commences Saif on diuretics (for ascites), beta blockers (to help in reducing the portal hypertension), and continues him on vitamin K injections (for his prolonged prothrombin time). He also advises him to stick to a low salt diet, decreases proteins in his diet and avoid constipation. Saif reviews the Liver Centre for further assessment.

Discussion Questions

- Are there words that you do not understand?
- Summarize key information that you have obtained from this progress.
- Construct a mechanism summarizing your final hypothesis with regard to the lesion, the mechanisms underlying Saif's problems. Provide supportive evidence from history, clinical examination and investigation results.

Case closure: (10 Minutes)

The team at King Faisal Specialized Hospital, Liver Centre evaluated Saif condition. However, two weeks later he developed another attack and vomited about two litres of blood. He was admitted again but he died the next day because of recurrent severe oesophageal varices bleeding and deterioration of his circulatory haemodynamics and liver functions.

Tutor's note:

In the last 10 minutes of the tutorial, you might encourage your group to discuss how they could work better as a group. What are the things they need to change and what things they need to improve? This discussion is very useful and will help the group to function better as they work on the next PBL case.

Challenging and Revision Questions

Tutors: Students could think about these questions on their own as they review the case. They might discuss their answers with their friends.

- Discuss the different cells in the liver and their functions.
- What are the mechanisms by which ascites and oedema of the lower limbs develop in Saif?.
- Discuss the pathogenesis of portal hypertension.
- Discuss the common causes of cirrhosis in Saudi Arabia.
- Discuss the pathology and pathogenesis of cirrhosis.
- Use basic sciences to interpret the symptoms, signs, and investigations results outlined in this case.

Learning Objectives:

On completion of this PBL package, students should be able to:

- Correlate the anatomical structures and cells of the liver with their functions.
- Discuss the pathology and pathogenesis of liver cirrhosis.
- Discuss the mechanisms underlying portal hypertension, portosystemic shunt, and associated complications including oesophageal varices, splenomegaly and development of ascites.
- Use basic sciences to interpret symptoms, signs and investigation results of a patient with liver cirhosis.
- Discuss the pathogenesis of haematemesis in a patient with liver cirhosis and portal hypertension
- Discuss a brief management plan showing management goals and management options in a patient with liver cirrhosis.
- Discuss the pharmacology of drugs used in the management plan.