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Histopathology

CLINICOPATHOLOGICAL CASE REPORT: CHRONIC CALCULOUS CHOLECYSTITIS AND SPLENIC INFARCTS IN SICKLE CELL DISEASE.

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ABSTRACT A 24 year old male coming from tribal area with chief complaints of difficulty in breathing, fever with chills and left hypochondriac pain for 10 days. Patient was known case of sickle cell disease (SCD) admitted on 28/02/2025 in male surgery ward, where he was examined pallor was present with few prominent palpable cervical, axillary and right inguinal lymph nodes. On examination of the abdomen showed moderate splenomegaly with cholelithiasis. Contrast CT of the abdomen done, which showed splenomegaly with ill defined non enhancing hypodense lesions suggestive of splenic infarcts, gallbladder was distended and showed multiple tiny calculi of size 2mm-3mm and mild to moderate left sided pleural effusion with segmental collapse of underlying left lower lobe was noted. Patient was well investigated and treated for difficulty in breathing, fever with chills and acute abdominal pain. His laparoscopic splenectomy with laparoscopic cholecystectomy was done and gross specimen of spleen and gall bladder was send to pathology department for gross pathological and histopathological examination. Patient was discharge on 11/03/2025 in good condition.

KEYWORDS : Splenic Infarcts, Sickle Cell Disease, Chronic Calculous Cholecystitis

INTRODUCTION

Sickle cell anemia (SCA) is a rare hereditary condition that affects less than one million people annually. A structural variation of normal adult haemoglobin (HbA), sickle cell haemoglobin is brought on by a mutation in the Hbb gene that causes valine to be substituted for glutamic acid at position 6 of the haemoglobin molecule's beta globin subunit (βs)[1]. Any illness where HbS production has patho-physiological effects is referred to as sickle cell disease. Usually referred to be either SCD SS or Sickle Cell Anaemia[3,4] , the most prevalent form of SCD (>70% of SCD worldwide)[2] is caused by the homozygous inheritance of the βs mutation.

Case Report

A 24 year old male presented with complaints of difficulty in breathing, fever with chills and left hypochondriac pain for 10 days. On examination pallor was present with few prominent palpable cervical, axillary and right inguinal lymph nodes. Examination of the abdomen showed moderate splenomegaly with cholelithiasis. Investigations revealed anemia, mild leukocytosis and mild thrombocytosis when patient is admitted and peripheral smear showed microcytic hypochromic anemia and when patient came for follow up after 1.5 months peripheral smear showed normocytic normochromic anemia with sickle cells and target cells, tear drops and Howel Jolly bodies (Fig 5). Sickling solubility test using 2% sodium metabisulphite showed sickle shaped RBC. Liver function test, coagulation profile, renal function test and electrolytes were within normal limits. HPLC analysis showed S window 73% suggestive of SCD. Contrast CT of the abdomen showed splenomegaly with ill defined non enhancing hypodense lesions suggestive of splenic infarcts, gallbladder was distended and showed multiple tiny calculi of size 2mm-3mm and mild to moderate left sided pleural effusion with segmental collapse of underlying left lower lobe was noted. Under general anaesthesia laparotomy with splenectomy and prophylactic cholecystectomy were performed for hypersplenism.

We received specimens in two containers in the histopathology section of our department. In container (A) received a splenectomy specimen measured 15.5x9.5x3.5cm and weigh 450gm. External surface was grey smooth, glistening and at places the capsule was stripped off exposing fragile granular and showing congestion, laceration and fibrinous exudate (Fig 1). On cut section, yellowish brown irregular serpentine necrotic areas were seen (Fig 2). In container (B) received a cholecystectomy specimen measured 4.0x2.0x1.0cm. Outer surface smooth and glistening (Fig 8). On cut section, multiple blackish green stones came out. Inner surface was greenish and velvety. Representative sections were given. Hematoxylin and eosin (H&E) stained sections showed a marked congestion of red pulp due to pooling of red blood cells. Dilated sinusoids filled with abnormal sickled erythrocytes (Fig 3,4) . There are large areas showing necrosis and fibrosis of splenic architecture. Container (B) sections studied show ulcerated gallbladder mucosa. There is presence of lympho-plasmacytic infiltration reaching upto serosa. Congested and dilated serosal blood vessels are seen (Fig 9). Histopathological examination showed features of splenic infarct and Chronic Calculous Cholecystitis with changes of SCD. The patient received polyvalent vaccine at follow up.

Figures-

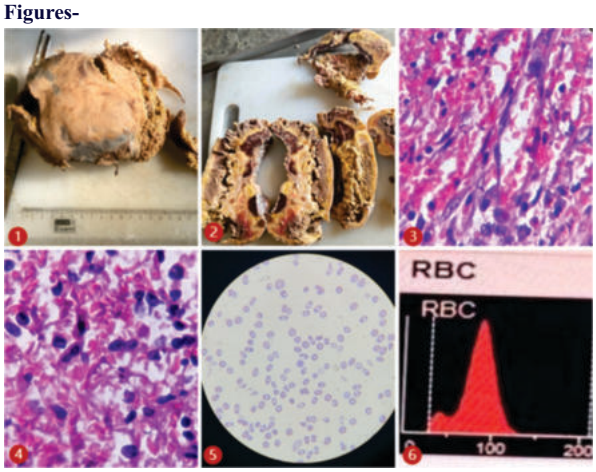

Figure 1- Gross Spleen Specimen: Diaphragmatic External Surface
Figure 2- Gross Spleen Specimen: On Cut Section, Inner Surface.
Figure 3 and 4- H&E Stain, x100: Spleen Shows Dilated Sinusoids Filled with Abnormal Sickled Erythrocytes.
Figure 5 Field Stain, x100: Peripheral Smear Showing Howel Jolly bodies.
Figure 6- Dimorphic RBCs Population.

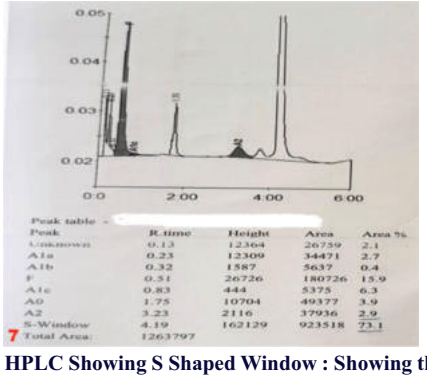

Figure 7- HPLC Showing S Shaped Window : Showing the Sickle Cell Disease.

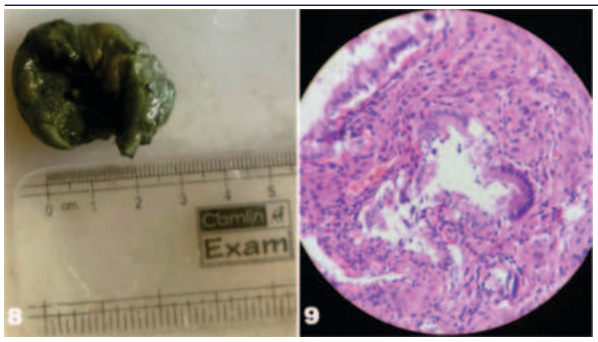

Figure 8- Gross Gall Bladder Specimen: Outer Surface and Inner Surface.
Figure 9- H&E stain, x40: Ulcerated Gallbladder Mucosa.

DISCUSSION

1-4% of the population has SCA. Related to hyperhemolytic crisis, acute sequestration crisis, and aplastic crisis. Splenic infarcts are nidus for the genesis of abscesses. Central India and Jharkand migrant labourers frequently suffer from SCA. About 22-44% is the prevalence in Central India.

When a splenic infarct is diagnosed, the most common symptom is still abdominal pain. Sickle hemoglobinopathies are among the most frequent causes of splenic infarction[1]. Hypoxic episodes in sickle cell disease patients are caused by acidity and cellular dehydration, which polymerises HbS within erythrocytes and deforms them into the distinctive sickle shape [2,3]. This sickling interacts dynamically with the vascular endothelium to cause ischaemia and reperfusion, vascular and inflammatory stress, and increased production of adhesion molecules, inflammatory cytokines, and vascular oxidases.

Anaemia, hypoxia, cholelithiasis, exhaustion, intolerance to exercise, hypercoagulability, and vasculopathy from chronic haemolysis result in endothelial nitric oxide depletion, which in turn produces ischaemic strokes and pulmonary hypertension[5,6]. The spleen is the most frequently afflicted and first organ to be impacted by SCA, while it can affect any region of the body. The spleen typically grows in the first decade of life, but subsequently gradually shrinks as a result of repeated incidents of vaso-occlusion and infarction, necessitating autosplenectomy [4].

CONCLUSION

Analgesics, hydration, and other supportive care techniques can be used to treat a splenic infarct in a non-infectious situation. Treatment for sickle cell hemoglobinopathies primarily consists of addressing hypoxia, though acidosis may also be necessary. Pseudocyst, abscess, haemorrhage, splenic rupture, and aneurysm are among the serious side effects of splenic infarct that call for immediate surgical intervention.

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