Case Series

Biliary collection masquerading as Grey Turner sign

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INTRODUCTION

Literature has documented a constellation of eponyms describing ecchymosis of abdominal wall as marker for a potentially serious cause of internal bleeding and mortality. Despite their rarity and limited diagnostic value, the presence of these dermatologic clues is a clinical dogma, whose mechanism and pathophysiology still needs to be elucidated thus, demanding clinical explanation and attention.

Conventionally Grey Turner sign (flank discoloration) has been associated with pancreatitis and the presence of retroperitoneal haemorrhagic fluid in contrast to our description of flank discoloration with biliary sanguineous collection in three different scenarios.1 There are other case reports as well attributing other causative pathologies.2-5

This case series emphasizes on the importance of physical examination, appropriate and timely intervention and to keep an open mindset while examining and re-evaluating such signs given its rarity and high mortality rate in order to develop a thorough, precise, evidence-based understanding of the anatomy, pathophysiology and management.
Hence the following series aims to supplement and improvise the existing knowledge pertaining to such rare conditions.

CASE 1

30-year-old gentleman presented to emergency department with complains of sudden onset diffuse abdominal pain, with history of jaundice 10 days back. Physical examination revealed tachycardia of 134 beats/min, blood pressure of 80/60 mmHg and diffuse abdominal tenderness. Patient was resuscitated and ultrasound abdomen pelvis revealed moderate fluid collection in abdomen, however the organ of concern couldn’t be discerned. Taking into consideration patient’s clinical condition he was taken up for exploratory laparotomy whereby there was intraoperative evidence of gall bladder perforation while liver and other organs appeared normal. Cholecystectomy was performed and drain inserted following which patient was shifted to Intensive care unit. Postoperatively patient’s vitals gradually improved, however a patch of flank discoloration appeared around drain site on POD-7. Clinically patient didn’t have any systemic signs suggestive of sepsis and laboratory values were normal except the haemoglobin had dropped to 10 g/dl. Patient was managed by application of glycerine to local site and by POD-15 had significantly reduced.

CASE 2

46-year-old gentleman was posted for elective interval cholecystectomy after a pre-anaesthetic check-up. Patient didn’t suffer from any comorbidity and neither had any positive medical or surgical history sans admission for acute cholecystitis 6 weeks back at a private setup whereby he was managed conservatively. Patient underwent laparoscopic cholecystectomy and entire procedure was uneventful. On postoperative day 5, patient developed right flank ecchymosis over an area measuring 5×4 cm². Clinically patient had fever of 100F and tachycardia of 105 beats/min, however was otherwise stable. Upon laboratory investigations, patient had leucocytosis of 10,000/mm³, while other parameters including haemoglobin, clotting profile, urine examination were normal. Patient was managed conservatively with MgSO₄ and glycerine dressing over the area of ecchymosis and with intravenous antibiotics to which he responded completely. By day 14, during regular OPD follow up ecchymosis had resolved completely.

Figure 1: (a) POD-7 ecchymosis in right flank, (b) POD-14 residual ecchymosis peri-drain region.

CASE 3

52-year-old gentleman with no known comorbidities underwent laparoscopic cholecystectomy for acute cholecystitis. On pod-2 patient complained of distension of abdomen and fever. On examination patient had a pulse rate of 106/min, blood pressure 130/80 mmHg, fever-101F and drain output: 50 cc bilious. Though there was distension of abdomen, clinically it was soft and ultrasound was suggestive of minimal fluid (non-tappable) in gall bladder fossa. Considering the above scenario patient was managed conservatively with antibiotics. However, on POD-4 patients tachycardia increased to 116 beats per min and drain output to 75 cc, hence patient was taken up for exploratory laparotomy whereby iatrogenic duodenal perforation of 3×3 cm was observed for which thorough wash was given and primary closure with gastrojejunostomy and feeding jejunostomy was done. Post operatively patient was kept on mechanical ventilator support for 4 days following which a large patch of ecchymosis extending from subcostal margin to right iliac region was noted. During this period of time patient was diagnosed to be in sepsis induced DIC (Hb: 10g/dl, Plt: 80,000, TLC count: 16,000, PT: 20 sec, APTT: 40 sec) which was managed by hiking up of antibiotics, fresh frozen plasma and
supportive management. On POD-14 patient had recovered from sepsis and was on jejunostomy feeds, however the ecchymotic patch was still present for which local ultrasound revealed mild subcutaneous oedema. Hence, patient was managed with glycerine dressing and by 1 month it was left to residual brownish pigmentation as shown below.

![Image: Residual brown pigmentation seen 1 month after operative procedure.]

**DISCUSSION**

In the year 1917, George Grey turner observed a palm size area of dirty-greenish discoloration and induration in loin region in a case of acute pancreatitis which according to him was never mentioned in any of the comprehensive and voluminous literature on pancreatitis before and henceforth the sign of flank discoloration came to be known as Grey Turner sign.¹

Though the sign has been conventionally associated with haemorrhagic and necrotizing pancreatitis, a large number of case reports have been reported worldwide associating flank discoloration to abdominal and retroperitoneal pathology causes namely ruptured hepatocellular carcinoma, idiopathic perineal haemorrhage, rectus sheath hematoma, spontaneous rupture and haemorrhage of adrenal cyst etc.²⁻³

This case series in turn highlights conditions whereby the mere presence of biliary sanguineous collection leads to flank discoloration. While reviewing the literature pertaining to similar such conditions there is dearth of data sans two case reports, one dating back to 1905, published in the journal of American medical association by Roshanoff in which he reported localized jaundice of umbilicus in case of rupture of common bile duct and peritonitis which later came to be known as Cullen sign.⁷

The second case report in 2017 by Mc Kelvie describing erythematous discoloration of flank following iatrogenic duodenal perforation post ERCP (endoscopic retrograde cholangiopancreatography).⁸

Based on microscopic, histologic and radiological analysis, Grey Turner's sign is attributed to haemorrhagic fluid tracking from anterior pararenal space (via the 2 leaves of posterior renal fascia) to lateral edge of quadratus lumborum muscle to the flank predisposed by anatomical defects of transversalis fascia.⁹⁻¹¹ Similarly Cullen sign is possibly related to the retrograde flow of bile through lymphatics via round ligament of liver from transverse fissure towards the navel.⁶ In both scenarios skin discoloration results from accumulation of blood in subcutaneous changes and varying pigmentation reflects stages of red blood cell breakdown.⁹⁻¹¹ Therefore pathophysiology underlying the above two signs is retroperitoneal haemorrhagic fluid tracking along the fascial planes to the abdominal wall musculature subsequently causing discoloration of periumbilical or flank tissue.⁹⁻¹¹ Based on above descriptions, it is possible that in our case series the biliary sanguineous collection could have possibly tracked to the flank region via the drain site due to defect in transversalis fascia. However more insight and studies are required as to ascertain which cases will develop above mentioned findings.

**CONCLUSION**

Despite its discovery more than a century ago, Grey Turner's sign and Cullen sign is scarcely observed and reported. The important point to be highlighted is the high mortality rate conventionally associated with Grey Turner's sign as shown by Jacobs et al, who reported a mortality rate ranging between 50 to 60% and Dickson and Imrie et al who cited an associated mortality rate of 37%,¹²⁻¹³ Both studies involved patients with acute pancreatitis and the percentage of patients presenting with either of the signs represented less than 2% of the total cases. In our case series there was no mortality however increased length of hospital stay and hence, morbidity. However, more cases need to be examined and reported to correlate with the morbidity and mortality and elucidate the prime causative factor in mortality i.e. whether it is the primary aetiology (pancreatitis, biliary peritonitis) or patient management or patient factor or a combination of above factors responsible for the same before any conclusion can be made.

Hence a greater number of cases is needed to justify the above observation with prime emphasis on the need for a keen-eyed observation and vigilant bedside physical examination.

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