## Case Series

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# Acute pancreatitis and relative polycythaemia: a case series

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#### **ABSTRACT**

Relative polycythaemia is an apparent rise in erythrocyte level in the blood. However, the underlying cause is reduced blood plasma. Relative polycythaemia is often caused by loss of body fluids seen in conditions such as burns, dehydration and stress manifesting itself as a raised Haemoglobin or haematocrit. This case series presents a clinical summary of three patients with acute pancreatitis (AP) and relative polycythaemia due to reduction in plasma volume as a result of intravascular volume depletion as demonstrated by increase in haemoglobin and haematocrit. Haem concentration may be considered as a marker for acute severe pancreatitis and correcting the volume depletion by adequate fluid improves the outcome of AP as seen in all our three patients.

**Keywords:** Acute necrotizing pancreatitis, Relative polycythaemia, Haem concentration

## INTRODUCTION

Acute pancreatitis (AP) is an inflammatory process of Pancreas, mostly develop as mild and self-limiting disease. But around 25% of the patients present in a severe form and the mortality among them is 30%. The ability to identify patients in severe pancreatitis is thus critical to the appropriate level of care and for intervention. Several prognostic factors and clinical and laboratory tests have been developed but so far no easy, accessible and economical marker is identified.

Polycythaemia is an unusual manifestation of AP and it is an apparent rise in erythrocyte level in blood. In severe pancreatitis, a lot of protein exudes from the pancreas in to the retroperitoneal space as well as peritoneal cavity resulting in intravascular hypovolemia. Intra vascular fluid depletion can manifest as haem concentration and it has been postulated that haematocrit could be a marker for diagnosis of severe pancreatitis.<sup>2</sup>

This case series presents three patients with AP and relative polycythaemia due to actual reduction in plasma volume as a result of intravascular volume depletion as demonstrated by increase in haemoglobin (Hb) and haematocrit (Hct) during acute pancreatitis and the significance of this association is discussed.

## **CASE SERIES**

#### Case 1

A 41 years old male presented to the OPD with complaints of pain in upper abdomen and vomiting since 1 week. He was a known alcoholic for past 20 years and stopped stopped since 3 years. His previous admission clinical reports showed the following parameters. Hb: 20g/dl (normal 13-17 g/dl) PCV: 60.7% (normal 44-54%) RBC 6.61 million/cumm (normal 4.5-5.5 million/cumm). CT abdomen revealed bulky head of pancreas with ill-defined hypo dense lesion in pancreaticoduodenal groove. Focal pancreatitis with mass head of pancreas was suspected.

On admission, physical examination revealed, pulse rate of 96 beats/min BP of 130/70 mmhg, abdomen was soft, tenderness in the epigastrium with a vague mass approx 3×3 cm was palpable in the epigastrium. Patient was investigated which showed Hb: 17.9 g/dl, PCV: 59.5%, RBC 5.64 million/cumm (Table 1). His serum amylase was 215U/l (normal 28-100U/l) and serum lipase was 380 U/l (normal 1-38U/l), CT abdomen revealed focal pancreatitis similar to previous episode (Figure 1 and 2).



Figure 1: Bulky body and tail with minimal fat stranding.



Figure 2: Ill-defined lesion is seen in the head of pancreas, loss of fat plane between the duodenum and head of pancreas.

Patient was treated conservatively with intravenous fluids, analgesics and proton pump inhibitors. A repeat Hb on day 7 was 15.5g%, PCV: 51.4, RBC 5.48 million/cumm (Table 1). Relative polycythaemia associated focal pancreatitis was confirmed and patient was managed conservatively.

## Case 2

A 28 years old male presented with acute onset of epigastric pain which radiated to the back and 5-6 episodes of vomiting of 1 day duration. He was a known alcoholic for 4 years and non-smoker. Examination revealed pulse of 89 beats/min BP 140/100 mmHg and tenderness in the epigastrium. Lab investigations showed

a Hb: 20.2 g/dl (normal 13-17g/dl) PCV 62.2% (normal 40-50%) RBC 6.53 million/cumm (normal 4.5-5.5 million/cumm) (Table 1). Serum amylase was 141 U/l, serum lipase 342 U/l. Patient was treated as acute pancreatitis and managed with intravenous fluids and analgesics. CT abdomen showed features of acute groove pancreatitis with minimal peri pancreatic fluid collection (Figure 3).



Figure 3: Pancreatic head with heterogenous enhancement with few non enhancing foci within it, focal necrotising pancreatitis (groove pancreatitis).

Repeat Hb on day 7 was 18.6g/dl, PCV 57.2%, RBC 6.04 million/cumm) (Table 1) which showed improvement in the haematocrit value again suggesting a relative polycythaemia associated with acute pancreatitis.

#### Case 3

27 year old male known alcoholic presented with severe epigastric pain and vomiting of 4 episodes for 1 day. He gave history of similar complaints in the past but no history of previous hospital admission. Examination revealed PR: 100 beats/min, RR: 26 cycles/min with SpO2 of 96% in room air and BP: 140/80 mmHg. Abdomen was soft and tender at the epigastric region. On further investigation his Hb was 18.1 g/dl (normal 13-17 g/dl) PCV 53.7% (normal 40-50%) RBC 5.76 million/cumm (normal3.5-5.5million/cumm) (Table 1). Sr amylase 210 U/l and sr lipase 579 U/l. Chest X-ray showed bilateral pleural effusion (left>right). Renal parameters were within normal limits and USG abdomen revealed features of acute pancreatitis with minimal peri pancreatic fluid in the lesser sac. Patient was managed with intravenous fluids and analgesics. He underwent CT abdomen on day 3 of admission which showed bulky body and tail of pancreas with non-enhancing necrotic areas, affecting more than 30%. Significant peri pancreatic fat stranding noted with ill-defined fluid collection noted in the peri pancreatic region extending to the greater curvature, the lesser curvature, perisplenic and left sub diaphragmatic regions. Filling defect noted in the retropancreatic splenic vein s/o partial thrombosis. CT severity index (CTSI) of 10 suggestive of acute necrotizing pancreatitis with acute necrotic collection (Figure 4).

Table 1: Lab parameters of the patients.

Cases	Hb (g/dl)		PCV (%)		RBC (million/cumm)	
	Day 1	Day 7	Day 1	Day 7	Day 1	Day 7
Case 1	17.9	15.5	59.5	51.4	5.64	5.48
Case 2	20.2	18.6	62.2	57.2	6.53	6.04
Case 3	18.1	12.6	53.7	41.7	5.76	4.52

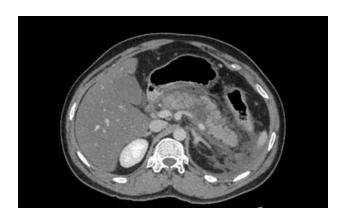


Figure 4: Bulky head and body of pancreas with areas of nonenhancement (s/o necrosis). Also note the peripancreatic collection.

Repeat Hb on day 7 was 12.6 g/dl, PCV 41.7%, RBC 4.52 million/cumm (Table 1) which showed improvement in the haematocrit value confirming our diagnosis.

### DISCUSSION

Acute Pancreatitis is one of the most common gastrointestinal disorders worldwide and continues to be a major of cause of morbidity and mortality despite improvement in intensive care, imaging and interventional techniques. Morphologically AP is of two types viz., interstitial oedematous pancreatitis and necrotizing pancreatitis. According to revised Atlanta classification, pancreatitis can be mild, moderate and severe. Early assessment of its severity helps in successful management of this dreadful disease.

Acute severe pancreatitis often features substantial total body fluid shift into the retroperitoneal space as well as peritoneal cavity, resulting in intravascular hypovolemia and decrease in pancreatic blood supply which leads to pancreatic necrosis. Pancreatic ischemia also leads to activation of inflammatory mediators. Since intravascular volume depletion can manifest as a concentrated haematocrit, it has been postulated that "Haem concentration may be a marker that reflects the deficits in pancreatic microcirculation that is purported to lead to the development of necrosis".<sup>3</sup> In the setting of acute pancreatitis, an admission haematocrit greater than or equal to 44% and/or a failure of haematocrit to drop at 24 hours have been reported as useful markers to predict subsequent necrosis.<sup>4</sup>

Relative polycythaemia is a term used to describe an elevation of the haematocrit level either caused by an acute transient state of hem concentration associated with intravascular fluid depletion or a chronic sustained relative polycythaemia caused by contraction of the plasma volume.<sup>5</sup> It also results from hem concentration due to decreased fluid intake and/or marked loss of body fluids. A contraction of plasma volume produces apparent erythrocytosis without any actual increase in the red cell mass.<sup>6</sup> Thus, early fluid resuscitation helps in restoring local pancreatic perfusion, counteract systemic hypotension and thereby preventing secondary organ failure due to fluid sequestration.

In severe haemorrhagic pancreatitis, reduced plasma volume may show a decreased haematocrit and a further decrease in Hct over 24 hr by 1% is associated with a small but a significant increase in risk of severe pancreatitis.

Portal, splenic and mesenteric vein thrombosis can be observed frequently in relative polycythaemia patients. There is no clear explanation for the cause of thrombosis. It is supposed that hyper viscosity may play a significant role.<sup>7</sup>

#### **CONCLUSION**

Acute pancreatitis is a life-threatening surgical emergency which should be managed appropriately with vigorous intravenous hydration in the initial stages to correct the haem concentration and relative polycythaemia. Decreased haematocrit during the first 24 hours of care leads to a decrease in morbidity and vigorous intravenous hydration has been shown to prevent the development of parenchymal necrosis. Also, haematocrit may be considered as a marker for acute severe pancreatitis. Hence early and adequate fluid resuscitation remains the corner stone of initial treatment of acute pancreatitis.

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