Fluctuating serum amylase levels in a patient with pancreatic ascites

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Introduction

Gross calcification of the pancreatic head in adolescence is a rare condition which raises the possibility of hereditary chronic pancreatitis (HCP) even in the absence of a family history [1]. HCP leads to premature autocatalytic activation of trypsinogen to trypsin. This results in imbalance between intra-pancreatic proteases and their inhibitors, progressing to exocrine and endocrine pancreatic failure with 35%-54% lifetime risk of pancreatic cancer [1,2]. Spontaneous pancreatic ascites usually occurs following pancreatic duct disruption. About 50% of these patients can have leaking pancreatic pseudocysts which causes significant morbidity and mortality [3, 4].

Case report

A 14-year old previously healthy boy, presented with intermittent epigastric pain and vomiting of four months duration. He developed progressive abdominal distension and pain with general ill health, warranting hospitalisation. He did not have a significant family history of pancreatic disorders. He was pale, tachycardic, tachypnoeic and had gross ascites on examination. Initial investigations showed white cell count of $15x10^6$/µl with 83% neutrophils, initial serum amylase was 1400 u/l (normal range 40 – 140). The ultrasound scan (USS) showed a large pancreatic pseudocyst, extensive pancreatic head calcification and moderate to gross ascites. Contrast CT abdomen confirmed these findings (Figure 1). The child had features of exocrine pancreatic insufficiency; loss of weight, steatorrhoea and low serum albumin of 24 g/l (normal range 35 – 55). Glycaemic control was normal suggesting that the endocrine portion was intact. Total parenteral nutrition was commenced as he could not tolerate oral food and to rest the pancreas. A peritoneal drainage catheter was placed as the child was having progressive abdominal distension. Peritoneal fluid analysis showed marked elevation of amylase (22935 u/l), there were no malignant cells.

Denegrade repeated serum amylase levels done during the initial course of the illness showed marked fluctuation. This was associated with clamping and unclamping of the peritoneal drainage system. When the catheter was unclamped the serum amylase level was 285 u/l. When it was clamped the serum amylase level increased up to 1240 u/l and again dropped to 340 u/l on unclamping. Endoscopic retrograde pancreatico cholangiography (ERCP) was performed after initial resuscitation. ERCP showed a markedly dilated main pancreatic duct with minimal side branches with a demonstrable leak between the middle and distal thirds of the pancreatic duct (Figure 2). The guide wire was passed beyond the leaking site and pancreatic duct was stented with 7Fr/10cm microvasive plastic stent (Figure 3).

Few days following endotherapy there was reduction in the peritoneal drainage output and serum amylase. Repeat USS abdomen two weeks following endotherapy showed interval changes of the pseudocyst.

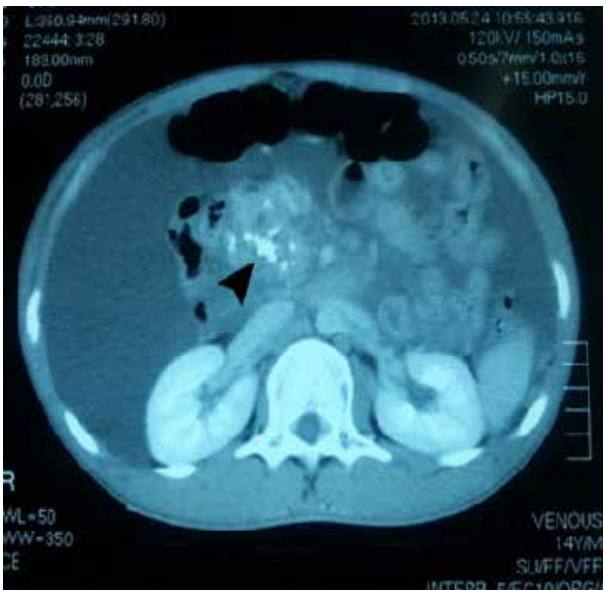

Figure 1. **CECT Abdomen shown pancreatic head calcification (arrow head)**

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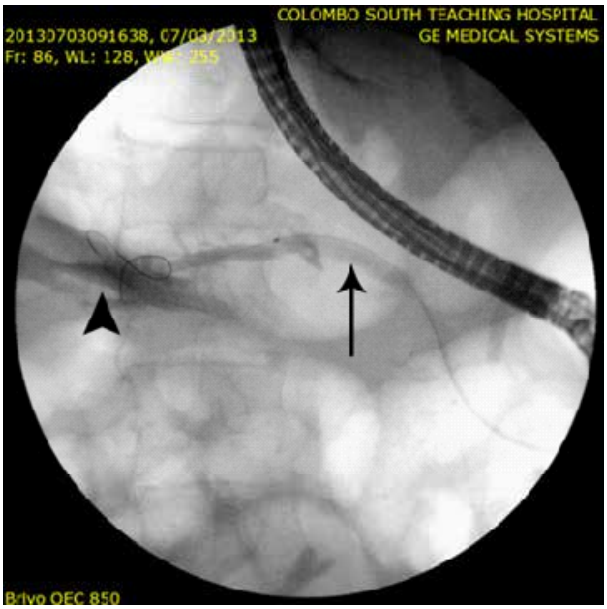

Figure 2. **ERCP showing proximal dilated pancreatic duct (arrow) and distal leaking site (arrow head)**

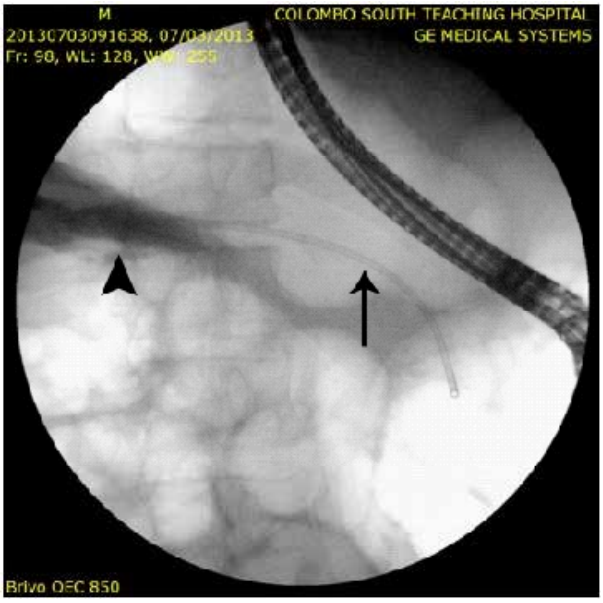

Figure 3. **Pancreatogram showing placement of plastic stent (arrow) beyond the leaking site (arrow head)**

Discussion

Approximately 95% of spontaneous pancreatic ascites are associated with chronic pancreatitis [3]. The exact pathophysiology for duct or pseudocyst rupture is unknown. Localised pancreatic necrosis with an element of increased intraductal pressure due to downstream obstruction would be a likely mechanism for duct rupture. Extensive calcification in the head of the pancreas causing duct distortion and blockage leading to very high intra-ductal pressure towards distal pancreas could be a possible cause of spontaneous duct disruption. Increased pressure within the pseudocyst, local erosion of the cyst wall by autodigestion or ischaemia, and increased abdominal pressure are likely to have contributed to the pseudocyst leakage. In this case, extensive calcification in the head of the pancreas could have caused spontaneous duct disruption. Leakage from the large pseudocyst may have contributed to the gross ascites with high amylase. In this patient the pancreatogram demonstrated a disrupted distal pancreatic duct with free leakage of contrast into the peritoneal cavity with no enhancement of the pseudocyst.

The rise in serum amylase levels could be secondary to absorption of amylase through peritoneum into the circulation, rather than due to continuing acute inflammatory process of the pancreas. This may explain why serum amylase levels fluctuated with clamping and unclamping of the peritoneal drainage and why levels reduced after endotherapy. Though rising serum amylase is well explained in the literature, fluctuation of serum amylase in pancreatic ascites in relation to clamping and unclamping is not reported [5].

Earlier this condition was treated conservatively with pancreatic rest, octreotide, paracentesis and diuretics. When these measure failed surgery is performed resecting the leaking part of pancreas or in cases with concomitant pseudocyst by cysto-gastrostomy or cysto-jejunostomy [3]. With the endotherapeutic measures there is an improvement in morbidity and mortality [3].

Conflicts of interests

There are no conflicts of interest.

References

1. Jonas R, Hans B, Joachim M, Niels T. Hereditary chronic pancreatitis. *Orphanet J Rare Dis* 2007; 2: 1.
2. Wikipedia. Hereditary Pancreatitis. Available at: http://en.wikipedia.org/wiki/Hereditary_pancreatitis [Accessed 19 January 2014].
3. Richard AK. Management of Pancreatic Ascites. *Gastroenterol Hepatol* 2007; **3**: 362-4.
4. Ganesh PC, Suvarna D, Bhat G. Endoscopic treatment as first-line therapy for pancreatic ascites and pleural effusion. *J Gastroenterol Hepatol* 2009; **24**: 1198-202.
5. Runyon BA. Amylase levels in ascitic fluid. *J Clinical Gastroenterol* 1987; **9**: 172-4.