Case Report

Pseudocyst presenting as pseudoachalasia

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ABSTRACT

Achalasia cardia is a primary motility disorder of esophagus which is due to loss of ganglion cells in myentric plexus, etiology of which is unknown. The condition causes dysphagia due to failure of relaxation of lower esophageal sphincter. Pseudoachalasia is a similar disorder which occurs usually due to adenocarcinoma of the cardia and also due benign tumors at this level. We present a rare case of pseudocyst of pancreas extending into mediastinum which mimics as pseudoachalasia.

Keywords: Achalasia cardia, Pseudoachalasia, Oesophago-gastro-duodenoscopy

INTRODUCTION

Pseudocysts are common and well recognised complication of acute or chronic pancreatitis. They are most commonly located in pancreatic or peripancreatic region. Benign disorders causing achalasia is very rare. Pseudoachalasia is most often due to malignancy.1-3 Association of achalasia with pseudocyst is rarely reported.

We report a rare case of pseudocyst of pancreas extending into posterior mediastinum causing pseudoachalasia.

CASE REPORT

A 17-year-old male was admitted in our hospital with history of abrupt onset of non-bilious vomiting on intake of solids and liquids, retrosternal discomfort, and weight loss of 6 kgs which began 3 months before admission. Patient had past history of severe attacks of epigastric pain radiating to back along with vomiting, 3 months, 1 year and 2 years before admission for which patient was managed conservatively in nearby hospital. Patient did not give any history of alcohol abuse and smoking.

On examination, patient’s abdomen was unremarkable except for mild tenderness in epigastric region. On auscultation of chest, breath sounds were decreased in left lower zone.

Laboratory investigations were normal, chest X-ray showed mild pleural effusion on left side. Oesophago-gastro-duodenoscopy showed slightly dilated oesophagus, kissing ulcer measuring 3x2 cm seen in lower 1/3rd of oesophagus with normal surrounding mucosa, GE junction at 36 cm, sphincter did not relax, however scope could be passed beyond it with slight pressure, Achalasia cardia, stricture oesophagus. Barium swallow reported mildly dilated oesophagus. Manometry showed increased basal esophageal pressure and incomplete lower esophageal sphincter relaxation on wet swallows with absent oesophageal peristalsis without pan oesophageal pressurization.

Ultrasound of abdomen was done which showed Anechoic cystic lesion of 14.8x6.2 cm in size noted in lesser sac in relation to tail of the pancreas with few internal echoes, with wall thickness of 2 mm. Dilated main pancreatic duct of 2.9 mm at body and few calcific foci seen in pancreas.
Contrast enhanced computed tomography of abdomen revealed a peripherally enhancing cystic lesion in lesser sac with extension into posterior mediastinum reaching up to left suprarenal region and forming close relation to pancreatic tail with mass effects and splenic vein thrombosis and splenic collaterals. Mass effect of the lesion is seen in the form of the anterior displacement of thoracic oesophagus and stomach and passive atelectasis of lower lobe of left lung. Findings likely to represent pseudocyst of pancreas.

DISCUSSION

Pseudocyst is one of the most common complications of pancreatitis. Most are located in peripancreatic region. Atypical locations of pseudocyst include liver, spleen, stomach wall. Pathways for their development include hepatoduodenal, gastrohepatic and gastrosplenic ligaments.

Extra abdominal location of pseudocysts is very rare, mediastinal pseudocyst is a rare complication which was described first in 1951. Pathogenesis explained in its development include pancreatic fluid entering mediastinum via oesophageal hiatus, aortic hiatus, foramen of Morgagni or through diaphragmatic rent wherever the pathway of least resistance is or following rupture of pancreatic duct posteriorly into retroperitoneum. Kirchner et al described radiological evidence of compression or displacement of the oesophagus in 61% of cases of mediastinal pseudocysts and associated dysphagia in 19%.

Achalasia cardia is a well explained entity which is diagnosed on the basis of history, endoscopy and manometric studies. However, to rule out secondary causes computed tomography of abdomen is helpful. Mechanism of development of secondary achalasia is not well defined. In cases of malignancy tumour infiltration with nerve damage and a paraneoplastic effect has been described. Other mechanisms explained include oesophageal obstruction causing neuromuscular
contractile damage. Interruption of vagal influence by any of the mechanisms could result in secondary achalasia. In our case vagal neuropathy or non-specific response to mechanical compression of oesophagus may be the cause for secondary achalasia.

In conclusion, patients with mediastinal extension of pseudocysts may present with dysphagia, mimicking achalasia. Prior history suggestive of pancreatitis should rise the suspicion of mediastinal pseudocyst.

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