been reported to cause oesophageal injury.

The common symptoms of DIOD are dysphagia, odynophagia and retrosternal chest pain. Patients may also complain of a foreign body sensation lodged in the throat. The symptoms start variably after ingestion of medicine and typically get better within a few days of discontinuing the drug. Diagnostic endoscopy is unnecessary if the history is typical. It is reserved for cases where the history is atypical; where symptoms persist for a long duration of time, presence of a pre-existing oesophageal disorder, when there is haematemesis or when other causes of dysphagia are considered as differentials. Endoscopy is the gold standard and radiological investigations are not informative in acute drug induced oesophagitis. The common endoscopic finding is one to several discrete shallow, small ulcers. Particles of medicine can also be found at the site or ulcer formation. Our patient had kissing ulcers at the level of aortic arch and the surrounding mucosa was normal.

DIOD is self-limiting and symptoms usually improve on discontinuation of the medicine. Our patient already stopped taking doxycycline after the third day. Proton pump inhibitors or H2 receptor antagonists have no proven role in the absence of reflux oesophagitis. Topical protective agents and local anaesthetics such as liquid sucralfate or lignocaine may be of benefit for ulcer healing and pain relief. Delayed oesophageal stricture formation may require endoscopic dilatation.

DIOD is a common and largely preventable condition. A detailed history and high index of suspicion is the key to an accurate diagnosis. If left undiagnosed it can have serious consequences. The simple advice of swallowing medication with plenty of water in an upright position can prevent the consequence of erosive oesophagitis.

References

Acute Aortic Syndrome- Penetrating Atherosclerotic Ulcer with contained Rupture Managed by Endovascular Stent-Grafting

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Abstract
We present the case of a hypertensive male who came with acute onset of severe backache and hypotension. Emergency imaging revealed a penetrating atherosclerotic ulcer of descending thoracic aorta with contained rupture and bilateral hemothorax. Initially stabilised with medical management, this patient went on to undergo endovascular stent-grafting. The sequence of clinical events of this uncommon entity and the relatively novel interventional modality are reviewed.

Introduction
A acute aortic syndrome is the modern term that includes aortic dissection, intramural hematoma (IMH) and penetrating atherosclerotic ulcer (PAU).\textsuperscript{1} Classic dissection is caused by a tear in the intima, commonly preceded by medial wall degeneration or cystic medial necrosis. Hypertension is the most common risk factor, with chronic exposure of aortic wall to high shear stress. Incidence is 2.6 to 3.5 cases per 1,00,000 person-years.\textsuperscript{2} Men are affected twice as often as women, with peak incidence in the fifth and sixth decades. The following case report demonstrates a PAU complicated by rupture, eventually managed by endovascular stent grafting, and explores the role of this intervention in...
Case Report

A 62 year old male was admitted to medical intensive care unit of tertiary care centre on 5th December, 2008 with complaints of sudden onset sharp severe backache followed by sweating, giddiness and shortness of breath since two hours. He also had dull epigastric pain since ten days not responding to antacids. The patient had been referred to our institute as a case of acute pancreatitis with report of elevated serum amylase. He was a retired clerk with a smoking history of twenty pack-years and near daily consumption of 40-60 ml of alcohol since fifteen years. He was detected to have essential hypertension two years ago and was on amlodipine 5 mg daily with poor compliance. Family history was non-contributory and unsafe sexual exposure was denied.

General examination revealed pallor with cold clammy extremities. Pulse rate was 110 beats/min, all peripheral pulses were feeble but symmetrical. Patient was tachypneic and hypotensive with a systolic BP of 70 mm of Hg in both upper limbs and 78 mm of Hg in both lower limbs in recumbent position. Cardiac examination was unremarkable. Bilateral breath sounds were reduced in infra-mammary areas, with stony dull note on percussion. Abdomen was soft on palpation. Patient was conscious, oriented and anxious with no focal neurological deficit. ECG showed sinus tachycardia and left axis deviation. Chest x-ray showed haziness of the right hemithorax.

The patient was initially resuscitated from shock with IV crystalloids, inotropes and blood transfusion under central venous pressure monitoring. Baseline investigations were as follows: Hb 9 g/dl, TLC 14,000/mm$^3$, S. creatinine 2.4 mg/dl, BUN 50 mg/dl, LDL-cholesterol 136 mg/dl, random blood glucose 126 mg/dl. Serology for HIV and syphilis was negative. 2D Echocardiography showed concentric left ventricular hypertrophy with ejection fraction of 60%. Abdominal ultrasound showed bilateral grade I renal disease with cortical scarring. Grade I hypertensive retinopathy was present on fundoscopy.

In view of this clinical picture, an acute aortic syndrome was suspected as a prime differential. Diagnostic pleural tap confirmed hemothorax. Urgent CT scan of chest and upper abdomen with aortic angiography was performed using non-ionic contrast. The scan revealed that the descending thoracic aorta (DTA) at D10-D11 vertebral level was very irregular, with ‘beaking’ at its lateral aspect (Fig. 1), with surrounding intramural hematoma (Fig. 2). The entire aorta showed atherosclerotic plaques. There was bilateral moderate hemothorax with underlying passive atelectasis of both lower lobes. Pancreas was normal.

The patient was stabilized but, BP remained labile, with sudden rise to 170/100 mm of Hg. It was controlled with IV metoprolol (10mg), sedatives and analgesics. After this initial stabilisation, there was sudden deterioration on day 3, with recurrence of back pain and hypotension. He needed intubation and mechanical ventilation with further packed cell transfusions. He was hemodynamically stabilised and could be weaned off the ventilator within the next three days.

He was referred to vascular interventional radiology of...
neighbouring institute on day 7, where a definitive procedure, Thoracic Endovascular Aortic Repair (TEVAR) was performed using a 34 mm x 10 cm endovascular stent graft (TAG device, Gore) (Fig. 3). Patient was observed for five days post procedure and had uncomplicated convalescence. He was discharged on oral metoprolol, atorvastatin and advised lifestyle and dietary modifications. On follow-up at three months, he is asymptomatic, ambulatory with optimum BP control with CT chest showing patent aortic lumen with stent-graft in situ with almost total resolution of hemothorax (Fig. 4).

**Discussion**

PAU is increasingly recognised as a variant of classic false lumen aortic dissection with a high incidence of bleeding complications and rupture in up to 40% of patients. PAU itself can lead to aortic dissection or perforation. Uncomplicated de Bakey type B dissections have a 30 day mortality of 10% and may be managed medically with beta blockers, vasodilators and opioids or by stent grafting in the future. However, patients who develop ischemic complications or contained rupture often require urgent surgical repair which carries a short term mortality of 20-25%. It is in these patients that endovascular stent grafting is emerging as an attractive, safe and less invasive therapeutic modality. Rupture and leakage into the pleural space may cause hemothorax as in this case; however it is more often on the left. Diagnostic imaging includes spiral CT, MR, trans-esophageal echocardiography and aortography. CT is easily available, fast and demonstrates aortic anatomy with great clarity.

Indications for endovascular stent grafting would be persistent or recurrent pain, aortic expansion, dissection progression, end-organ malperfusion syndromes and contained rupture. The stent graft seals off the ulcer or intimal tear, induces thrombosis of the false lumen, reduces wall stress and thus provides stabilisation of the aortic segment. Follow-up includes stringent BP control at 120/80 mm of Hg and periodic imaging to detect complication or progression.

**Conclusion**

A high index of suspicion, timely imaging, hemodynamic resuscitation and definitive intervention help successfully manage acute aortic syndrome with otherwise high mortality. Endovascular stent grafting is rapidly evolving as a safe, less invasive technique in managing complicated cases and is gradually complementing or even replacing the need for surgery.

**References**