Correspondence

Diagnostic Dilemma of A Case of Chest Pain

Sir,

A 55 years man was admitted to a rural hospital with severe respiratory distress. He was hypertensive, smoker, non-diabetic and was suffering from chronic obstructive lung disease. The patient was treated with bronchodilator, antibiotic and steroid. His symptoms improved on initial treatment. But on 3rd day of admission he developed chest pain which was retrosternal in location and constrictive in nature. The pain was relieved after giving sub-lingual nitroglycerine. ECG showed normal axis with sinus rhythm. Except T-wave inversion in aVL lead, no abnormality was detected. He was referred to a tertiary care centre. However the patient chose to be admitted to a nursing home where ECG was repeated and reported as normal. Chest X-ray revealed hyperinflation of lungs due to chronic obstructive lung disease. Cardiac enzymes were within normal limits. No regional wall motion abnormality was detected by Echocardiography, LVEF was 64% and all other parameters were within normal limit. He was treated withisosorbide di nitrate 5mg four times daily, aspirin and clopidogrel combination, ACE inhibitor and bronchodilator inhaler. The patient’s chest pain improved with treatment. Then he was discharged from the nursing home in stable condition and was advised to get a coronary angiography. On the very next day, patient again developed chest pain during taking meal and was admitted in our hospital. Above investigations were repeated which depicted the same picture as before. This time chest pain relieved partially with nitrate. Coronary angiography was done and it revealed no obvious abnormality. To rule out any esophageal cause, upper GI endoscopy was performed and surprisingly it discovered an impacted tooth at the lower end of esophagus (Fig. 1). The tooth was removed endoscopically and an ulcer was seen at that site. Then the patient was managed symptomatically. After one week he was discharged with antihypertensive agent and bronchodilator. No chest pain reappeared on follow-up.

It is sometimes very difficult to distinguish between chest pain due to esophageal disease and chest pain secondary to cardiac ischaemia, as there are many similarities of cardiac and esophageal chest pain. Both may be mid and lower retrosternal in location; heaviness, squeezing, tightening or burning in nature; can be associated with diaphoresis. Both can radiate to upwards or to left neck, shoulder or arm. Eating can precipitate both angina and esophageal chest pain. To further compound the difficulty in distinguishing between angina and esophageal pain, both may be relieved by nitroglycerine1

The common esophageal disorders that may mimic angina pectoris are gastroesophageal reflux and disorders of esophageal motility, including diffuse esophageal spasm as well as “nut-cracker esophagus” characterized by high amplitude peristaltic contractions and vigorous achalasia.2

In our case, problem was that the patient was totally unaware of uprooting of his tooth. He knew it only on seeing the tooth after its removal. Chest X-ray did not give any clue probably, because of superimposition of tooth-opacity with the cardiac or vertebral shadow. The character of chest pain and its relief after taking nitrate, were all suggestive of cardiac angina. Moreover in the initial few days of treatment the patient was better as he used to take nitrate four times daily before meal. On the day of admission in our hospital, patient developed severe chest pain during taking meal. Further enquiry unveiled that he forgot to take nitrate on that day before meal. But we thought it as post prandial angina. When cardiac catheterization demonstrated angiographically absence of coronary artery disease (CAD), we reviewed our diagnostic approach and thought it prudent to exclude the possibility of an esophageal cause. It is not unusual for patients of unsuspected esophageal foreign body ingestion, to present with chest pain as the main symptom. In almost all reported cases patients have undergone extensive cardiac evaluation to exclude CAD before searching for an esophageal cause.3

So, in spite of patient presenting with typical anginal chest pain, esophageal cause of chest pain should always be kept in mind.

Fig. 1: Esophagoscopy showing impacted tooth at lower end of esophagus.
REFERENCES


H. pylori: Association with Megaloblastic Anaemia

Sir,

The article by Desai and Gupta entitled “Helicobacter pylori link to pernicious anaemia” is very interesting and timely. Megaloblastic anaemia is a common problem both in outdoor as well as in indoor patients, where they are admitted for anaemia related complications. We have also noticed that the usual causes of nutritional or systemic disorders are absent in a great majority of these patients and at time it is extremely difficult to pinpoint a probable etiology. Gastrointestinal symptoms are usually neglected unless they point towards definitive malabsorption features. We report below a young patient with megaloblastic anaemia in whom a diagnostic work-up was done as the anaemia had developed over a few weeks without a definitive etiology both systemic as well as nutritional.

A 23 years old male presented with generalised weakness for 4 weeks, blurring of vision, nausea and vomiting for 15 days. There was no history of addiction, especially alcohol intake/prolonged drug intake such as antiepileptic drugs. He came from a good family background and nutritional intake was absolutely normal. On examination patient was conscious, oriented to time, place and person, BP was 114/70 mmHg, pulse rate 126/min regular, respiratory rate 20/min and he was severely pale. CVS – loud S1 and soft systolic murmur at apex was present. Respiratory system, abdominal and nervous system examination were normal. Investigations revealed Hb of 4.0 gm%, TLC 3400/mm³, DLC – P₉₀ L₂₀ M₁0, platelet count 134000/mm³, reticulocyte count 3.2%, PCV 11.9%, RBC 1.27x10¹²/mm³, MCV 104.7 fl, MCH 31.5 pg/cell and MCHC was 33.2 g/l. Peripheral smear showed anisocytosis, microcytosis, macrocytosis, poikilocytosis and neutrophil hypersegmentation. Kidney function test, serum electrolytes, blood sugar and urine examination were normal. In liver function tests serum bilirubin (T) was 1.6 mg/dl (direct 0.03 mg/dl), SGPT 23 U/l, SGOT 64 U/l, alkaline phosphatase 36 KAU/l and total protein was 6 gm/dl (albumin 4 gm/dl). X-ray chest PA view and ultrasound abdomen were within normal limits. Stool for occult blood was positive. Iron study was normal and serum vitamin B12 level was 146 (normal 200-800 pg/ml). Fundus examination showed anemic retinopathy. Upper GI endoscopy revealed gastric erosions and rapid urease test was positive for H. pylori. Gastric and duodenal biopsies were taken to rule out atrophic gastritis and sprue. Report revealed chronic gastritis with activity and Giemsa stain was positive for H. pylori. Duodenal biopsy was suggestive of non-specific inflammation. Anti-gastric parietal cell antibodies was 49.0 U/ml (normal <10 U/ml). IFA could not be done due to cost factor. Patient was put on H. pylori kit (two Cap. Omeprazole 20 mg, two Tab. Amoxicillin 750 mg and two Tab. Tinidazole 500 mg) 1 OD for 10 days and inj. Vitcofol once a week for 4 weeks. Patient showed dramatic response to the treatment.

Our case illustrates that GI symptoms should be sought carefully and minutely in all patients with megaloblastic anaemia especially when profile indicates absence of usual causes. Upper GI endoscopy combined with investigations for H. pylori both in serum and biopsy become mandatory as indicated by these authors as well as Kaptan et al, who suggested H. pylori as novel causative agent in vitamin B12 deficiency. Similar observations have also been reported elsewhere. We agree with the view that the work-up as suggested in this article will establish diagnosis of pernicious anaemia/atrophic gastritis in a cost effective manner.

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REFERENCES


Renal Consequences of Metabolic Syndrome

Sir,

Obesity has become a major public health problem worldwide, as a result of abundant food, sedentary lifestyle and Thrifty gene phenotype. The study by Gupta et al in a large group of randomly selected adults has shown a continuous positive relationship of all markers of obesity (body-mass index, waist size and waist hip ratio) with major coronary risk factors-hypertension, diabetes and metabolic syndrome. Recent studies have shown that Metabolic Syndromes...