interval and tend to convert it into an RP interval. Antegrade block (depression of the impulse conduction to the ventricle) will prolong PR interval and tend to convert an RP into a PR interval. They reported one case with antegrade block, the PR interval measuring 0.18 sec.

Vakil\(^3\) reported two cases of atrioventricular nodal rhythm with antegrade block. One of them, an elderly, hypertensive patient with acute pulmonary edema, had a PR interval of 0.16 second. The other patient showed three different types of rhythm besides normal sinus rhythm, namely (a) uncomplicated upper nodal rhythm, (b) nodal tachycardia with two to one atrioventricular antegrade block, and (c) nodal tachycardia with instances of two to one block, first degree heart block and nodal pause. The PR interval of the case was 0.09-0.10 sec. One complex showed a PR interval of 0.20 sec. followed by retrograde P wave.

Bix\(^6\) reported three interesting cases of nodal rhythm with A-V block. One of them showed A-V nodal rhythm with A-V block in form of Wenckebach phenomenon.

The electrocardiogram of our patient showed A-V nodal rhythm with antegrade block, in which PR interval measured 0.25 seconds. This is a larger PR interval associated with atrioventricular nodal rhythm with antegrade block than we could find in any other paper.

REFERENCES

An Unusual Cause of Massive Hemothorax

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Although intestinal obstruction and strangulation as a complication of traumatic diaphragmatic hernia are well recognized and have been reported frequently since the first case was described by Pare in 1564,\(^1\) massive bloody pleural effusion has been seldom associated with strangulation. The paucity of the symptoms referred to the abdomen in this case is most unusual.

O. M., a 33 year old colored man, was admitted on December 9, 1954, complaining of shortness of breath and swollen abdomen.

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He had led an apparently uneventful existence with no serious illness until one week prior to admission when he developed a "cold" followed by cough productive of about an ounce of mucoid sputum daily. Five days prior to admission he experienced a sharp pain in the lower and lateral part of the left chest, which was aggravated by cough and deep inspiration. This condition progressively deteriorated and he developed increasing shortness of breath. He became aware of abdominal swelling in the last two days. The last bowel movement occurred two days prior to admission to the hospital. He experienced no abdominal pain or vomiting. Interrogation revealed that abdominal distension had occurred on many occasions in the past and had been relieved by infrequent bowel movements.

He was a moderate smoker but had over-indulged in alcoholic beverages for the past 10 years.

Physical examination revealed an acutely and extremely ill individual in severe respiratory distress, sweating profusely. The facial expression was anxious and apprehensive. The pulse rate was 120 per minute, regular but of low volume. Rectal temperature was 101.2° F., and respiration attained a rate of 40 per minute and was shallow in character.

Examination of the eyes, ears, nose and throat revealed an essentially normal status except for slight hyperemia of pharyngeal mucosa. There was marked deviation of the trachea toward the right. There was no evidence of clubbing of the finger tips and no lymphadenopathy was palpable anywhere in the body. There were old healed scars on the face, left arm and in the left eighth intercostal space at the scapular line. The expansion of the left hemithorax was markedly limited.

Palpation revealed diminished tactile fremitus over the entire left hemithorax. Percussion elicited flatness over the same side except at the extreme apex. The apex beat was palpable in the right fourth intercostal space two inches to the right of the sternal border. Percussion of the cardiac dullness confirmed the palpatory finding. On auscultation the heart sounds were regular, rapid and best heard over the right hemithorax. The expiratory phase of respiration was prolonged and there were diffuse expiratory wheezes over the right lung. The breath sounds were faint and distant over the left hemithorax except at the apex.

The abdomen was moderately and uniformly distended but no tenderness or rebound tenderness could be elicited. Peristalsis was hypoactive.

Rectal examination and the remainder of the physical examination were unremarkable.

Laboratory findings were red blood cells 4,600,000 per cubic millimeter, hemoglobin 11 grams per cent, hematocrit 33 per cent, white blood cells 21,800 per cubic millimeter with shift to the left. Urine was normal. Serum analysis of the b.ood was 6 Winslow's units.

An x-ray film of the chest revealed a homogeneous density involving the entire left hemithorax. There was marked mediastinal shift toward the right. The dome of the left diaphragm could not be visualized. The findings were consistent with massive pleural effusion (Figure 1).

Thirty-three hundred cubic centimeters of grossly bloody fluid was aspirated from the left pleural space. The fluid had the appearance of thin dark venous blood. The hematocrit on this fluid was 5 per cent and it contained 460,000 red blood cells per cubic millimeter. The serum analysis value of the aspirated material was 4 Winslow's units. During thoracentesis, 1,000 cc. of blood were transfused to the patient, which raised the hematocrit from 33 to 39 per cent.

He improved subjectively and objectively following thoracentesis and transfusion. A subsequent x-ray film demonstrated that the upper limit of the homogeneous density in the left hemithorax had descended to the level of the fourth costal cartilage anteriorly. A large circumscribed area of radiolucency in the left apex, which was interpreted as localized pneumothorax, was visualized. The mediastinum returned to a median position; the dome of the left diaphragm was still not demonstrable (Figure 2). X-ray examination of the abdomen showed a small and large bowel distension with fluid levels (Figure 3).

Abdominal decompression was attempted by continuous Wangensteen suction. Nothing was given by mouth but intravenous fluids were administered together with wide range antimicrobial drugs.

Surgical consultation resulted in the opinion that the abdominal distension was due to paralytic ileus. Normal saline, soap and water and warm olive oil enemas were unsuccessful and the abdominal distension was unaffected by attempts at oral and rectal decompression.

He developed a coarse tremor of the hands and lips and was incoherent and irrational. By the second hospital day he was in frank "delirium tremens."

X-ray film of the chest on the third hospital day revealed diminution of the density
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in the left chest. The left diaphragm was still not visualized but two fluid levels were seen in this area (Figure 4).

Seventy-two hours after admission, for the first and last time he vomited coffee ground material and expired 15 minutes later. The fluid aspirated from the chest was reported after death to be positive for anaerobic streptococci and E. coli on culture.

Autopsy showed abdominal distension. Old healed scars were present on the face, left arm and a third one, two inches in length, along the left eighth intercostal space in the scapular line. The gastrointestinal tract was markedly distended and covered with sero-fibrinous exudate. The descending colon was empty and collapsed below the splenic flexure.

The lungs revealed emphysematous changes in the upper lobes with emphysematous bullae in the apex of the left lung. The remainder of the left lung was atelectatic and covered by sero-fibrinous exudate and was firmly attached to the chest wall anterolaterally.

A loop composed of splenic flexure and part of the transverse colon was found in the left hemithorax. The loop was found to be edematous, hemorrhagic and necrotic. There was no peritoneal sac to this loop. The diaphragmatic opening was in the postero-lateral position of the left diaphragm. It was small with an organized plastic fibrous ring.

Microscopical examination of the strangulated loop showed destroyed mucosa with polymorphonuclear cell infiltration and fibrin deposition.

Discussion

Intestinal obstruction and strangulation are not infrequent complications of diaphragmatic hernia and are the most dangerous ones indeed. About 90 per cent of all cases of strangulated diaphragmatic hernia are traumatic. Almost all the cases reported, or 98.4 per cent are left sided for obvious anatomical reasons.

Pleural effusion accompanying strangulation of herniation through the diaphragm is not an unusual finding and has been detected clinically, radiologically, through thoracotomy, or at autopsy, on many occasions. The character of the fluid may be serous but is often serosanguinous because of the underlying physio-pathology.

FIGURE 4
Although bloody pleural effusion was described as early as 1858 as a fortuitous finding by Alderson during an autopsy of a case of strangulated diaphragmatic hernia with perforation of the stomach, massive hemothorax has been rarely encountered or reported.

We were able to find only three cases in the literature more or less comparable to the case being reported in respect to the amount and character of the pleural effusion.\(^6.7\)

Deaner et al. described a case of hemothorax with strangulated diaphragmatic hernia with opacification of the left chest on x-ray film but the amount of fluid aspirated was only 37 cc. on two occasions. On autopsy the left hemithorax contained omentum and clotted blood.

However, this case is unique and different from all the cases previously reported in the paucity of abdominal symptoms and signs except for the distension which, together with the sluggish peristalsis and the absence of pain and vomiting, had been attributed to paralytic ileus.

In all the cases reported, the abdominal symptoms of pain, discomfort and vomiting were the predominant part of the clinical picture. Although chest pain, dyspnea and evidence of pleural effusion are common with strangulated diaphragmatic hernia, they have apparently never been so overwhelmingly the outstanding part of the clinical picture as to effectively mask the abdominal component.

The history of a "cold" as the onset of the present illness, the diffuse wheezing and evidence of bronchospasm, the presence of radiolucency which was interpreted as localized pneumothorax or a bulla, the massive bloody pleural effusion, the absence of abdominal pain, vomiting or tenderness, the sluggish peristalsis, in addition to complicating delirium tremens, directed attention to the respiratory system as the site of primary pathology.

Barium enema to investigate the cause of abdominal distension probably would have given the answer and would have resulted in the correct surgical approach to the problem. The patient expired before the clue given by the report of the culture of the pleural effusion was received and before further studies could be initiated.

REFERENCES

5 Alderson: Lancet, 2:386, 1858.