open-lung biopsy. Additionally, IVUS may provide prognostic information based on the type and degree of these morphologic changes, such as thromboembolic lesions, concentric laminar fibrosis, thickness of vessel layers, cross-sectional area as well as atherosclerosis.\textsuperscript{8,9} These changes can be followed up over time with sequential IVUS examinations, and this information can be combined with echocardiographic and angiographic data to provide greater prognostic value. Finally, by its ability to accurately measure cross-sectional area of the vascular bed and the response to pulmonary vasodilating agents, IVUS may have potential use in the medical management of patients with pulmonary hypertension.

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Unusual Presentation of Gastric Dilatation*

Dramatic Complete Atrioventricular Block

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We report a case of gastric dilatation in a ventilated 18-year-old woman, which was revealed by the acute onset of hypotension, sinus bradycardia, and complete atrioventricular block. Hypotension and cardiac rhythm disturbances resolved with intravenous injection of atropine, but recurred a few minutes later and required a second bolus of atropine, which had a transient beneficial effect. Only gastric decompression, as soon as gastric distention was recognized, was able to restore normal cardiac rhythm and adequate blood pressure. This case highlights the seriousness of gastric dilatation and argues for the vagal mechanism of cardiac rhythm and conduction troubles complicating gastric dilatation.

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Gastric gaseous dilatation in critically ill patients is not always a harmless event. We report a case of gastric dilatation complicated by complete atrioventricular block that occurred in a patient in intensive care who had no history of heart disease.

CASE REPORT

An 18-year-old woman was admitted to our intensive care unit for recurrent seizures and unconsciousness. She had a 10-year history of epileptic seizures treated with carbamazepine (600 mg/day). The status epilepticus was related to withdrawal of medication during the previous 3 days. On clinical examination, the patient was obese, she had tonic-clonic generalized seizures, and there was no evidence of neurologic focalized features. There was no recovery of consciousness in the interval between crises. Seizures recurred a few minutes later despite the intravenous injection of clonazepam (2 mg). The patient was then treated with a continuous infusion of thiopental at a rate of 3 g/day, and mechanical ventilation was required. The baseline treatment with carbamazepine was started on the first day via a nasogastric tube.

On the fifth day, the sedation was withdrawn, and seizures had not recurred. On the sixth day, the patient developed diarrhea. There was abdominal pain with no obvious distention. The serum potassium value was 2.8 mmol/L (normal range, 3.5 to 5 mmol/L). This level was promptly corrected by parenteral adjunct therapy with potassium. The serum sodium level was 135 mmol/L and the glucose level was 6.4 mmol/L. There was no significant acid-base imbalance (pH 7.59; PaCO\textsubscript{2} 36 mm Hg; and HCO\textsubscript{3} 24 mmol/L). The PaO\textsubscript{2} was 107 mm Hg with a fractional concentration of oxygen in the inspired gas (FIO\textsubscript{2}) of 30 percent. The results of hepatic and renal function tests were normal.

Suddenly, the patient became restless and hypotensive (blood pressure, 70/50 mm Hg); she was in a cold sweat. At this moment, *from the Intensive Care Unit (Dr. Hmouda), the Internal Medicine and Infectious Disease Unit (Dr. Jemni), and the Cardiology Unit (Drs. Jeridi, Ernez-Hajri, and Ammar), Sousse Medical School, Farhat Hached Hospital, Sousse, Tunisia.

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a second blood sample showed a serum potassium level at 3.9 mmol/L, and the creatine kinase level was within the normal range. The ECG showed sinus bradycardia followed by a third-degree atrioventricular block (Fig 1). All of these symptoms were transiently relieved by intravenous injection of atropine (0.5 mg), but the same symptoms recurred 5 minutes later and were accompanied by some eructation. A second injection of atropine had the same transient effect. The chest x-ray film showed marked gaseous dilatation (Fig 2). Immediate decompression by nasogastric tube suctioning was then accomplished. All the symptoms were instantaneously relieved, and there was neither recurrence of atrioventricular block nor further need of atropine. The patient was then in sinus rhythm, with blood pressure of 130/70 mm Hg. Continuous electrocardiographic monitoring showed no evidence of conduction abnormalities or other electrical disorders but did reveal particularly a normal corrected QT interval and an absence of prominent U waves. The patient was successfully weaned from mechanical ventilation on the ninth day and was discharged from the intensive care unit to the internal medicine unit on the 11th day, where a two-dimensional echocardiogram was obtained and considered normal.

**DISCUSSION**

Our case highlights the fact that gastric dilatation can be a life-threatening complication in critically ill patients, especially when it is hidden by obesity and patients are sedated. The distention of the stomach can activate viscerocardiac reflexes via the vagus nerve, leading to a hypervagotonic state that can induce profound bradycardia and low blood pressure. The occurrence of third-degree atrioventricular block is uncommonly described as a complication of gastric dilatation. It might be explained by profound vagotonia, which depresses conduction in the atrioventricular node. The regression of symptoms after the atropine infusion strongly argues for this mechanism.

Many other potentially contributing factors to this arrhythmia should be discussed. Carbamazepine, with its anticholinergic properties, may cause arrhythmia and atrioventricular block, but no other adverse reactions due to this drug were noted. In addition, the recurrent seizures are believed to be related to withdrawal of carbamazepine, rather than to its toxic effects. The favorable outcome despite reinstitution of carbamazepine at the same dosage argues against this hypothesis. The contributions of hypokalemia or hypoxemia in this arrhythmia were excluded, since the potassium level and PaO₂ were within the normal range when atrioventricular block occurred. The absence of acid-base disturbances and other metabolic abnormalities exclude the possible contribution of metabolic factors in this arrhythmia. Cardiac ischemic hypoxia was also unlikely to occur in our patient because there were no features of circulatory failure preceding the onset of atrioventricular block. In addition, an underlying cardiac disease such as myocarditis or right coronary artery occlusion or spasm was unlikely, since the ECG, echocardiogram, and creatine kinase level were normal. Further cardiac investigation was not performed. The disappearance of this arrhythmia once gastric decompression was performed argues against all of the previous hypotheses and provides consistent proof of a cause-and-effect relation between atrioventricular block and gastric dilatation.

Other complications of gastric dilatation have been described, particularly cardiac dysrhythmias, dramatic electrocardiographic T wave changes that have completely resolved after gastric decompression, electrolyte disturbances, and acid-base imbalance. Therefore, attention should be paid to patients at high risk for developing gastric dilatation, particularly those suffering from COPD or those sedated and mechanically ventilated, those being treated by large doses of anticholinergic drugs or having electrolyte and acid-base disturbances (hypokalemia; diabetic ketoacidosis).

In conclusion, gastric dilatation must be kept in mind and suspected, particularly in the case of sudden onset of bradycardia or atrioventricular block. Immediate gastric decompression is the only relevant and lifesaving treatment.

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Left Pleural Hemorrhagic Effusion*

A Presenting Sign of Thoracic Aortic Dissecting Aneurysm

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Left hemorrhagic pleural effusion was the presenting sign of painless aortic dissecting aneurysm in two elderly hypertensive patients. Computed tomography (CT) of the chest revealed the aneurysmal dilatation of the thoracic aorta and an intimal flap connecting its descending part with the left pleural space. The patients were treated conservatively with blood transfusions and drugs directed to control blood pressure. The first reported 71-year-old patient remains in stable condition for 16 months without evidence of recurrent active aortic dissection. The second 85-year-old patient remained in stable condition for 28 days, but finally had a second fatal episode of dissection into the left pleural space. The differential diagnosis of nontraumatic left hemorrhagic pleural effusion in an elderly hypertensive patient should include dissecting aneurysm of the descending thoracic aorta and CT of the chest should be performed as the next preferable diagnostic procedure.

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CT=computed tomography

Acute dissection of the thoracic aorta is one of the medical emergencies which demands prompt medical or surgical therapy. The choice of the particular therapeutic approach depends on the site of the intimal flap. While surgical intervention is the preferred treatment for dissection of the ascending aorta, conservative management is suggested for most cases with distal dissection without aortic rupture.1 Pleural effusion, commonly on the left side, is a rare complication of the rupture of the thoracic aorta.2,3 We report two elderly patients with dissecting aneurysm of the thoracic aorta, presenting with left pleural hemorrhagic effusion.

Case Reports

Case 1

A 70-year-old man with a history of hypertension treated with nifedipine, mild COPD, and chronic renal failure was admitted to the hospital in December 1991 with a 3-day duration of dyspnea. On examination, the patient was a slightly overweight, pale, elderly man in no apparent distress. The pulse rate was 88 beats per minute; respiration rate, 26 breaths per minute; and blood pressure, 160/90 mm Hg. Increased dullness to percussion and decreased breath sounds were apparent over the base of the left lung. Laboratory studies revealed a hemoglobin level of 89 g/L (8.9 g/dl), a leukocyte count of 14.7X10^9/L, and a serum creatinine value of 247.5 µmol/L (2.8 mg/dl). An ECG was normal. The chest x-ray film demonstrated a left pleural effusion and lobulated aortic contour (Fig 1), and a diagnostic aspiration of the pleural effusion revealed hemotherax. Computed tomography (CT) of the chest revealed dilatation of the descending aorta (diameter, 6 cm) with a 5-cm intimal flap connecting with the left

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FIGURE 2. Patient 1. The CT scan shows the intimal flap separating the true lumina and false lumina which communicated with the left pleural space that contains pleural effusion.