Digestive Chagas disease with concomitant lipoid pneumonia*

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A 50-year-old woman with chagasic esophageal achalasia and megacolon presented with nonproductive cough, chest pain and dyspnea. A chest X-ray showed bilateral opacity suggestive of lobar pneumonia. Open lung biopsy revealed lipoid pneumonia resulting from aspiration of mineral oil from a mineral oil-based laxative that the patient had been taking regularly for the last three years. The authors discuss concomitance of chagasic megacolon and esophageal achalasia with lipoid pneumonia and make recommendations regarding the use of mineral oil-based products by these patients.

.Key words: Pneumonia, aspirtion/etiology. Pneumonia, lipid/etiology. Esophageal achalasia/pathology.

Chagas disease/complications.

INTRODUCTION

A 50-year-old woman presented with nonproductive cough, chest pain and dyspnea. Thirty years prior, the patient had presented signs of intestinal subocclusion and, after a laparotomy, was diagnosed with megacolon. On that occasion, indirect hemagglutination for Chagas disease was positive. The patient remained free of symptoms until 15 years later, at which time she began to experience episodes of constipation that spanned increasingly greater periods of time, often as long as 10 days. She then started using laxatives on a daily basis and giving herself weekly enemas.

CASE REPORT

Three years prior to the most recent treatment, after an even more prolonged episode of constipation, the patient was hospitalized for an intestinal lavage. During hospitalization, she received, per oral, mineral oil, a practice she maintained after discharge. With the use of 40 to 50 mL of this substance daily, the patient was able to evacuate her bowels every 3 to 4 days with resorting to enemas. Four months prior to seeking treatment again, she began to present dyspnea upon heavy exertion, and mild, sporadic chest pain upon drawing a breath, as well as a

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Submitted: 17 August 2003. Accepted, after review: 20 November 2003.

dry cough, mainly in the morning. She was admitted under suspicion of heart disease. A chest X-ray revealed alveolar opacities with poorly-defined borders in the posterior segment of the upper lobe of the right lung and in the lower lobe of the left lung, as well as a small opacity in the left lung base (Figure 1). On this occasion, the patient presented no signs or symptoms of infection or consumption, which are common in cases of tuberculosis and pulmonary mycosis. The patient was therefore submitted to a computed tomography scan of the chest, which revealed a ground-glass pattern with poorly-defined borders, involving both lower lobes but more pronounced in the right lobe. Esophageal achalasia, with an air-fluid level, was also observed (Figure 2). The predominance of right-sided lesions, together with the esophageal achalasia, suggested aspiration. Since the ground-glass pattern revealed an inflammatory process of light intensity, it was not considered compatible with suppurative pneumonia.

An open-lung freeze biopsy was recommended and performed, and the histopathological analysis showed partitioning of the alveolar walls with lymphatic ectasia and pneumocyte hyperplasia.

Within the alveolar space, great numbers of histiocytes, with microvascularization within the cytoplasm, were observed.

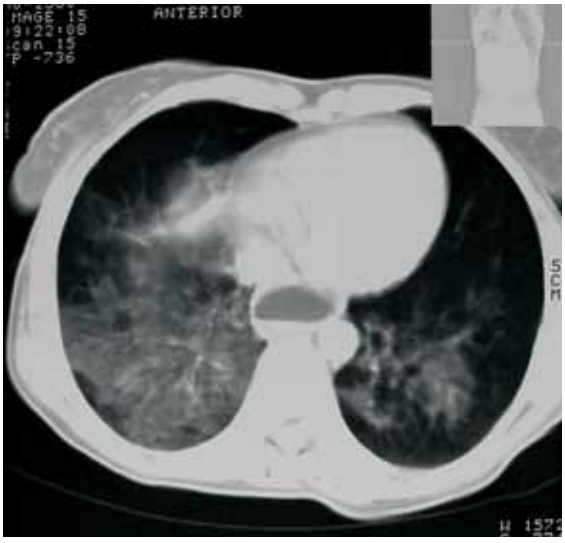

Figure 2 – Computed tomography of the chest showing a ground-glass pattern with poorly defined borders involving both lower lobes, more pronounced in the right lobe

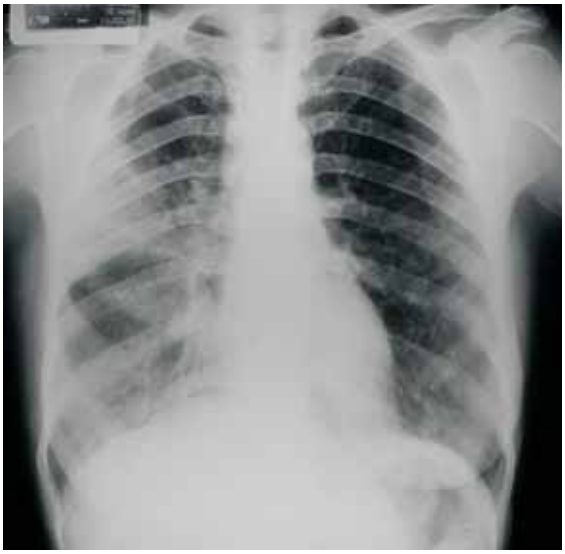

Figure 1 – Anteroposterior chest X-ray showing alveolar opacity with poorly defined borders in the posterior segment of the lower right lobe and a small opacity in the left lung base

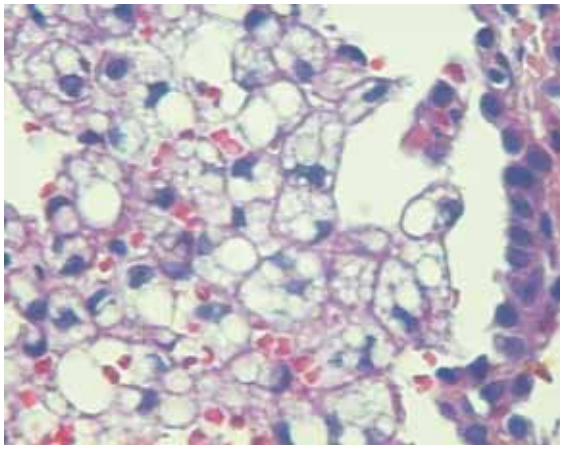

Figure 3 – Alveolar space filled with numerous macrophages containing a great quantity of lipid vacuoles in their cytoplasm

DISCUSSION

Chagas disease is caused by a protozoan parasite known as *Trypanosoma cruzi*. The initial infection may pass unnoticed or may manifest as either a mild or severe form of the disease, the latter being more common in endemic regions. Undetected infection is the more common scenario. In such individuals, the disease may evolve slowly and gradually into one of the chronic forms of the disease, which typically present cardiac or digestive abnormalities. The digestive form manifests as dysphagia in the presence of esophageal achalasia and, in the presence of megacolon, as severe intestinal constipation.[4-6]

The basic pathogenic phenomenom that determines the digestive form of the disease is denervation within the walls of the autonomic nervous system myenteric plexuses, especially the parasympathetic plexuses.[7]

In Brazil, chronic chagasic colopathy is a condition that is frequently seen, especially in the elderly, and results in progressively more severe intestinal constipation and alternating periods of diarrhea. In the later stages, after megacolon has been established, patients may remain constipated for extended periods, requiring the aid of laxatives and high colonics.[4]

Chagasic esophagopathy is another common alteration seen in cases of the digestive form of the disease. This condition mainly affects individuals older than 30 and manifests as dysphagia, primarily when dry, hard or cold foods are consumed. Such patients tend to ingest greater quantities of liquid at mealtimes in order to facilitate deglutition and relieve the symptoms.[4]

Esophageal dysfunction progresses through accumulation of ingested material in the superior portion of the esophagus, facilitating the aspiration of this material. This condition is more common in individuals in whom the cough reflex has been lost or suppressed due to neuromuscular disturbances, or due to the use of sedatives or other drugs. In such cases, aspiration pneumonia becomes established.[8-10]

Mineral oil commonly inhibits airway protective responses, such as glottal closure and cough, and is also capable of retarding mucociliary clearance. Mineral oil aspiration may be imperceptible, creating a condition that is easily confused with pneumonia. The substance is relatively inert and is not metabolized by tissue enzymes. It consists of a mixture of long-chain saturated hydrocarbons derived from petroleum. When aspirated, mineral oil emulsifies and appears as drops, or vacuoles, of free lipids in the macrophage cytoplasm. Therefore, an area of alveolar and interstitial accumulation of lipid-filled macrophages develops, surrounded by lymphoplasmacytic infiltrate. Over time, this area evolves into fibrosis, and oil-filled giant cells encircle the site, forming a structure known as a paraffinoma. The appearance of this type of lesion in imaging exams may lead to a diagnosis of pulmonary neoplasm. However, mineral oil aspiration can present a variety of radiological findings. Those most commonly seen in lipoid pneumonia are ground-glass opacities, interstitial abnormalities and pulmonary consolidations, any and all of which may appear in isolation or concomitantly in the same patient.[2,3,11,12]

In light of the possibility that these complications may occur, it is recommended that individuals at risk for bronchoaspiration avoid the use of mineral oil. In addition, it is strongly suggested that mineral oil be prescribed for short periods (less than one week) only since chronic use increases the risk of aspiration. The authors of this report propose that, in Brazil, mineral oil not be prescribed for individuals with chagasic megacolon since there is a risk that, in such patients, its use will become chronic. This warning carries additional weight in cases of intestinal Chagas disease involving esophageal achalasia.[13,14]

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