

The Combination of Amoebiasis with Helminthiasis and Maduromycosis Complicated by Secondary Amyloidosis: A Case Report

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Abstract

A rare case of secondary amyloidosis was found in a cadaver with amoebiasis and intrathoracic complications of amoebic liver abscess related with Helminthiasis and Maduromycosis, which proved the peculiarity of Amoebiasis to combine with polyparasitoses, namely Enterobiasis, strongyloidiasis and Wuchereriasis. The authors confirmed that the progression of secondary amyloidosis, most likely hepatopathic amyloidosis, can be accelerated by longstanding course of advanced, severe underlying illness with the development of amoebic complications, especially combined with Helminthiasis and Mycosis.

Keywords: Amoebiasis; Helminthiasis; Maduromycosis; Secondary amyloidosis

Case Report

It is well-known from literature about the possibility of amoebiasis damage of any organ as well as its combination with different diseases-bacterial dysentery, Balantidiasis, Helminthiasis, which make its course more severe [1-7] Amoebiasis is often complicated by amyloidosis [2] Cases of secondary amyloidosis developed from chronic schistosomiasis when amyloid was discovered in kidneys' biopsy specimens have been described [2]. In the secondary amyloidosis amyloid can be found in any organ, especially in the kidneys, adrenal glands, liver, spleen and eyes. According to studies [1-3], liver damage due to amoebiasis is evident in 35% to 37% of cases. Burst of amoebic liver abscess through diaphragm into thoracic cavity belongs to the most severe and late complications of this disease. Pus may discharge into (1) free pleural cavity with empyema formation, (2) circumscribed pleural cavity with following implication of lung tissue, (3) lung with abscess formation or bronchus without pus in pleural cavity (bronchial fistula), (4) pericardial cavity.

Maduromycosis (mycetoma)-chronic, localized, slowly progressive subcutaneous fungal infection, typically limited to the dorsal surface of the foot and accompanied by its expansion and deformity. This disease was first described in the middle of 19th century and initially named Madura foot, after the region of Madura in India where the disease was first identified. More than 80 types of fungi can cause Mycetoma but Actinomyces are the most common cause (Actinomycetoma). This type of deep mycoses is often complicated by multiple fistulas formation, fungal granulomas, Periostitis up to Blastomatous transformation of affected area and amyloidosis of parenchymatous organs [4-7]. Considering simultaneous combination of protozoal disease (amoebiasis), Helminthiasis (Strongyloidiasis, Enterobiasis, Wuchereriasis) and deep mycosis, we decided to report our own study results with morphologic analysis of autopsy material.

A cadaver of unidentified man was found early in the morning in the market-place and forwarded to medicolegal death investigation. On autopsy: The body is that of a middle-aged well developed, poor-nourished black male. There is mild rigor mortis present. Pale mucus membranes, scleral icterus, tongue is coated. There is erythematous rash on the skin of the back. Scrotum is enlarged, edematous, its skin is saddle, and shiny, smooth, lymph nodes are enlarged with a thickened wall (lymph scrotum). Cloudy opalescent fluid is noted inside upon dissection. Testicles and epididymis are twice enlarged, seminiferous tubules are thickened. There is a large amount of serous exudate between thickened testicular membranes. Inguinal and femoral lymph nodes are enlarged to a point of a size of an average fist. Left foot is enlarged and deformed with extrusion of the arch. There are many fistulas on the skin surface; sanguineous discharge with yellow grains is coming out.

On dissection of abdominal and thoracic cavities

Colon (especially ascending) and cecum: Colon (especially ascending) and cecum are increased in diameter with irregular thickened walls; mucous membrane is hyperemic with multiple

hemorrhagic foci. Ulcerous process is spread almost through the whole colon. Multiple ulcers, flagon-like on the dissection, are located along the mucous folds, with hemorrhages at the edges. Ulcer floor consists of gray necrotic detritus. In older ulcers floor is imbibed with green intestinal contents. Brownish yellow semifluid content is in the bowel lumen. Scars on the place of healed up ulcers narrow the bowel lumen. Dirty-grayish necrotic foci are protruding from the mucous membranes surface. Histologic examination of colonic wall showed area of ulcer defects with involvement in the process of mucosa, submucosa, muscle layer, with floor made out of detritus with bacterial flora (**Figure 1A**). In the remaining colonic wall tissue on the border of necrosis there are Entamoeba clusters with tissue forms predominance (**Figure 1B**); clusters of inflammatory cells are evident around them. In the duodenum there is catarrhal inflammation with frank edema of submucosa, isolated mucosal ulcerations with penetration of helminths inside of it (**Figure 2A**), hemorrhages, eosinophilic infiltrates.

Appendix: Three adult helminths 13 mm in length have been discovered in the lumen of appendix. Appendiceal mucous membrane is edematous with multiple hemorrhages. Mucous membrane of the small bowel, especially of the duodenum, is edematous with multiple hemorrhages and superficial ulcerations. Histologic examination of the appendix revealed signs of acute appendicitis, with adult helminths in its lumen (**Figure 2B**). In inguinal and femoral lymph nodes adult helminths are discovered with formed granulomas and presence of giant cells around them (**Figure 2C**), macrophages of epithelioid cells, foci of necrosis are noted. Severe fibrosis of lymph nodes with luminal narrowing is stated (**Figure 2D**).

Liver: In the upper segment of left lobe of the enlarged liver massive merging together necrotic foci were noted. They are grayish yellow in color, with colliquation of hepatic parenchyma, liquefaction and perforation of the diaphragm, burst into pericardial sack, development of pyohemorrhagic pericarditis and pericardial cavity tamponade. About 350 ml of chocolate-brown fluid detritus was evacuated from it. There is an irregular cavity in the anteroposterior segment of the right hepatic lobe that reaches 11 cm in width. The cavity is filled with chocolate-brown fluid detritus that got into pleural cavity through liquefied diaphragm which led to empyema formation.

Lung tissue of the lower lobe of right lung was involved into purulent process. Histological examination of the liver tissue revealed a picture of chronic amoebic hepatitis is seen in liver; on the border of necrotic tissues isolated amoebic tissue forms are discovered. Passive amyloid depositions are noted between hepatic trabeculae accompanied by sinusoids with atrophy of hepatocytes and their death (**Figures 3B** and **Figure 4**).

Gallbladder: In the gallbladder cavity, there is around 40 ml of dark green thick bile, there are isolated hemorrhages on the mucous membrane. Bile ducts are patent.

Esophageal and stomach: The mucous membrane is pale; stomach mucous membrane folds are flattened.

Pancreas: Pancreas is boggy, of pale reddish color, coarse lobular.

Spleen: Spleen has a mass of 160 g with moderate scrapping; its capsule is strained, dark cherry-colored. Kidneys are equal in size, have a mass of 205 g (11 × 7 × 4 cm), increased density, pale pink in color on the cross-section. In kidneys, most capillary loops of glomerulus are substituted with amyloid. Protein dystrophy and necrobiosis of tubules epithelium is evident. Some authors think [7], that the exact combination of amyloid damage of liver and kidneys is a reliable sign that allows suspecting hepatopathic amyloidosis.

Bladder: is contracted; there is around 60 ml of cloudy urine inside.

Prostate: has a round shape, firm, yellowish-pink on the cross-section.

Testis and epidymis: Filariatosal lymphangitis and lymphadenitis takes place. In testicles, there is lymphostasis, severe edema (**Figure 2E**); vessel endothelium is in the proliferative stage, infiltrated with lymphocytes, histiocytes, and plasma cells. There is varicose dilation of lymph vessels. Signs of epididymitis and funiculitis are evidence.

Left foot: Histologic picture is heterogeneous in the tissues of foot. In tissues, taken from fistulas and diffuse purulent infiltrates, compact druses are discovered. Druses represent eosinophilic material (**Figure 3A**), with necrotic foci localized around them. In tissues around the fistulas and in the distance from them granulomatous process is represented by different proportions of epithelioid cells, plasma cells, giant cells and lymphocytes. Foci of fibrosis and hard scar tissue are noted. On culture of pathology material fungus *Actinomyces madurae* was isolated.

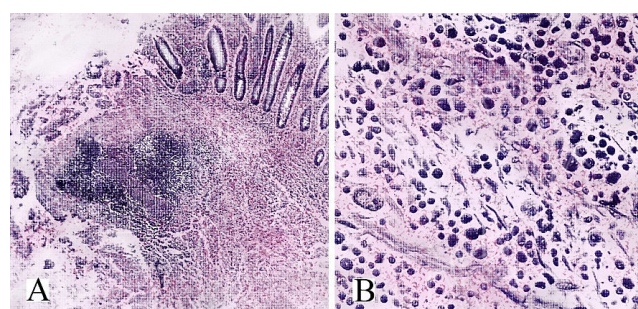


Figure 1 Morphologic features of amoebiasis. A. Ulcer defect of the colonic wall, (H&E staining, x100). B. Amoebic tissue forms on the border of necrosis, (H&E staining, x200).

In kidneys, most capillary loops of glomerulus are substituted with amyloid. Protein dystrophy and necrobiosis of tubules epithelium is evident. A picture of chronic amoebic hepatitis is seen in liver; on the border of necrotic tissues isolated amoebic tissue forms are discovered. Passive amyloid depositions are noted between hepatic trabeculae accompanied by sinusoids with atrophy of hepatocytes and their death (**Figures 3B** and **Figure 4**). Some authors think7, that the exact combination of amyloid damage of liver and

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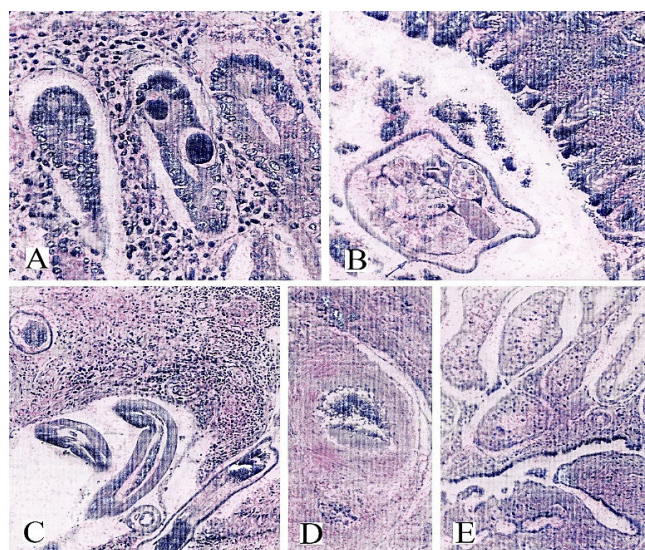


Figure 2 Morphologic characteristics of helminthiasis: Strongyloidiasis (A), Enterobiasis (B) and Wuchereriasis (C-D-E). (A) *Strongyloides stercoralis* larvae in mucosal glands of duodenum with evidence of Edematous enteritis, (H&E staining, x250). (B) Transverse cross-section of adult female helminth *Enterobius vermicularis* in the lumen of appendix, helminth has side spurs on the level of esophagus and contains a large amount of eggs, (H&E staining, x100). (D) Longitudinal and transverse cross-sections of adult *W. bancroftini* in the lymph node with granuloma formation, presence of giant cells, beginning of the necrosis of one of the parasites, (H&E staining, x100). (E) Fibrosis and luminal narrowing of the lymph node, (H&E staining, x100). (F) *Tunica albuminea* in the context of elephantiasis of scrotum, (H&E staining, x100).

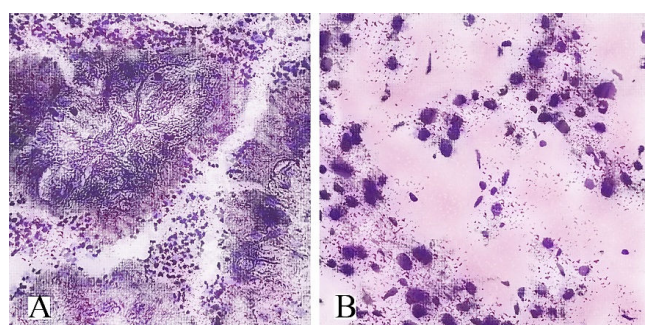


Figure 3 Morphologic characteristic of Maduromycosis (a), and secondary amyloidosis (b) in the liver. (A) Druses of *Actinomadura* in the diffuse purulent infiltrate of the foot soft tissues, (H&E staining, x200). (B) Amyloid in the form of massive depositions between the hepatic trabeculae, (H&E staining, x200).

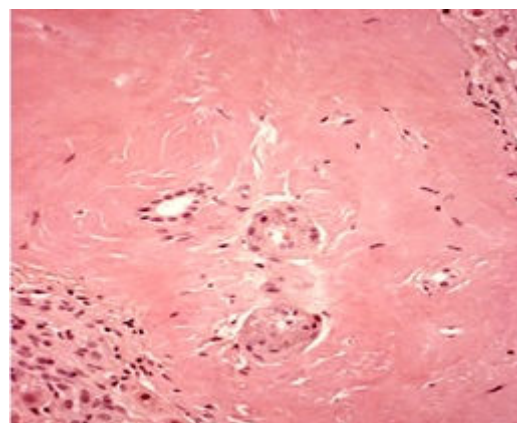


Figure 4 Amyloid deposits material in liver lesion stained with Congo red (Congo red staining, x100).

The first descriptions of amyloidosis date back from the beginning of 1840. Virchow was the first one to define amyloid substance [8]. Amyloidosis is a disease process resulting in the deposition and accumulation of fibrillar proteins and characterized by abnormal extracellular deposition of amyloid in different tissues and organs associated with dysfunction of the involved tissue or organ. The cause is still unknown. Amyloidosis is divided into: (1) primary, (2) amyloidosis associated with multiple myeloma (MM), (3) secondary. (4) hereditary-familial amyloidosis [9,10]. The progressive accumulation of amyloid deposits in normal tissues results in structural dysfunction, evolving into failure of the affected organ, most commonly the kidney, heart, liver and peripheral nervous system [11].

Primary amyloidosis is a systemic form without identifiable cause factor. Secondary amyloidosis refers to systemic amyloidosis concomitantly to chronic diseases such as tuberculosis, rheumatoid arthritis, Crohn's disease, among others [10]. The differential diagnosis between the systemic and localized form of amyloidosis may be made through α -Amyloid deposits have some characteristics: they are eosinophilic in H&E staining and present birefringence under polarized microscopic light when stained in Congo red. This is the simplest and most accepted criterion for diagnosis of amyloidosis. Under electron microscopy, these proteins have fibrous appearance [10,11].

This finding proves characteristic property of amoebiasis to combine with polyparasitoses, particularly with Enterobiasis, strongyloidiasis and Wuchereriasis. Longstanding course of advanced, severe underlying illness with developed intrathoracic complications of amoebic liver abscess combined with Helminthiasis and Maduromycosis accelerated the progression of secondary, most likely hepatopathic amyloidosis.

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