Case report

Peritoneal Hydatidosis in a Non-Endemic Area: A Case Report from Brazzaville, Congo

Hydatidose péritonéale dans une région non endémique: à propos d’un cas à Brazzaville - Congo

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RÉSUMÉ

Cystic echinococcosis (CE) is not documented in Central Africa. Here, we describe the first autochthonous infection with CE in Congo, Central Africa, providing the first evidence for a new geographical area at risk for the acquisition of CE. The whole peritoneal cavity was studded with cysts. Considering the diffuse involvement, patient was successfully managed with surgery associated with medical therapy. The origin of the contamination of our patient is discussed with a focus on a possible role of bush meat.

INTRODUCTION

Cystic echinococcosis (CE) is a zoonosis caused by cestodes of the Echinococcus granulosus complex (1). Adult tapeworms inhabit the small intestine of carnivores (the definitive hosts) and produce eggs, which are passed with faeces. The intermediate host is infected by ingestion of eggs. Subsequently, a larval stage (metacestode) develops as a cyst in internal organs of this host. The metacestode produces many protoscolices, each with the potential to develop into an adult tapeworm when ingested by the definitive host. It is assumed that dogs are definitive hosts and humans the accidental intermediate host. Humans get infected by contact with infested dogs or by consuming vegetables and/or water contaminated with hydatid ova (2). Thus, geographic distribution differs by country and region depending on the presence of large numbers of nomadic or semi-nomadic sheep and goat flocks that represent the intermediate host of the parasite, and on their close contact with the final host, the dog, which mostly provides the transmission of infection to humans. People can become intermediate hosts after accidental ingestion of eggs. Then they develop cysts that cause the morbidity and mortality associated with the disorder. Liver and lungs are the most commonly affected organs (3).
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CE is known to be endemic in several areas (4). In sub-Saharan Africa, it has been reported in West African as well as the East African countries (5). In all these areas, CE is prevalent in humans among the nomadic pastoralists. However, CE is not documented in Central Africa. For this reason, any case of Cystic echinococcosis from Central Africa should be reported in order to raise interest in further epidemiologic studies and to complete the sub-Saharan picture of the current CE situation.

Here, we report a case of primitive peritoneal hydatidosis in a female patient in Brazzaville, Congo, Central Africa.

CASE REPORT

A 63 year old Congolese female presented in the emergency unit of the Centre Hospitalier Universitaire de Brazzaville, Congo, with a progressive distention of the abdomen.

There was no history of fever, nausea or vomiting, loss of appetite, acute episodes of abdominal pain, constipation or jaundice. There was no previous abdominal operation. Patient lived in the city of Brazzaville, without any exposure to dogs, cats, or sheeps.

On physical examination, patient had a pulse rate of 72 beats/minute and blood pressure of 140/70 mm of Hg, respiratory rate of 16/min, and normal body temperature. Abdomen was symmetrically hugely distended (fig 1).

Figure 1: Massive abdominal distension

On palpation, no organomegaly was noted. On percussion, note was dull. Bowel sounds were normal. Other systems were normal. Clinical diagnosis of malignant ascites was made and further investigations were planned.

Hemoglobin of 9.4 gm/dl, leucocyte count of 10,700/mm³ and no eosinophilia were noted. Liver function tests were normal. Chest X-ray was normal. Ultrasonography of the abdomen showed multiple cysts present throughout the abdomen without involving the intra-abdominal organs, including the liver (Fig 2).

Figure 2a. Ultrasound: multilocular intraperitoneal collection

Computed tomography scan of the abdomen revealed the entire peritoneal cavity distended and occupied by multiple noncalcified cysts throughout the peritoneal cavity (Figure 3). There was no lesion in any organ. The small bowel loops were clustered together posteriorly. Differential diagnosis of pseudomyxoma peritonei or peritoneal seeding of hydatid disease was made. Enzyme-linked immune sorbent assay (ELISA) test for hydatid disease was positive with a titer of more than 1:80.

Figure 2b. Ultrasound: Double wall cavity

The patient was transferred to the surgery department. At exploratory laparotomy through a midline incision, peritoneal cavity was found filled with multiple daughter cysts and a membrane (Figure 3). The area surrounding cysts was cautiously protected during all the steps of the procedure with packs immersed in hypertonic saline. About 20 litres of cysts and fluids were removed and replaced with hypertonic saline, which was subsequently removed. Total cystectomy was performed. Postoperatively, patient was put on two courses of 4 weeks of Albendazole (400 mg twice daily) given with an interval of one month. When reviewed 6 months after surgery, patient remained asymptomatic and abdominal CT scan was unremarkable.

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Despite CE described as endemic in sub-Saharan Africa (5), for Central Africa no epidemiological data exist. Furthermore, no case reports of human disease have been reported from this region of Africa. Thus, researchers generally accept that this disorder is prevalent across the whole African continent, with an area of high prevalence in east Africa (5), but not in Central Africa. In the present report, we have described the first case of human CE in Congo, Central Africa. This case has several interests.

First, our patient presented with primary peritoneal hydatidosis, an entity which represents only 2% of all abdominal hydatid diseases (6). Indeed, the overall prevalence of peritoneal involvement in cases of abdominal hydatid disease is approximately 13% and most of cases are secondary to a hydatic cyst of the liver (7). Mechanism of primary peritoneal involvement is not clear to-date. The dissemination may take via lymphatic or systemic circulation (8). Existing data are suggestive of unusual clinical presentations of cystic echinococcosis in some parts of the African continent, for which the causes are speculative as in our patient.

Second, it occurred in an area without nomadic populations. Thus, the origin of the contamination of our patient remains elusive. Indeed, our patient had no history of travelling, close contact with dogs or other animals, and used to live in a city. However, bush meat bought on the market was her major source of animal protein. This raises the question of underestimation of the diagnosis of CE in some part of Africa, particularly in Central Africa. Indeed, because of the absence of insurance coverage, the necessity for the patient to pay before any exam is done, the high cost of transport facilities, and because of limited diagnostic facilities (no ultrasound facilities, no radiographic facilities), one might suspect that CE is underdiagnosed in some parts of Sub-Saharan Africa where patients prefer to turn to traditional medicine. Furthermore, CE may be still underdiagnosed because of lack of knowledge, resources, and record keeping. Additionally central Africa is confronted to epidemics of far greater magnitude, such as HIV, tuberculosis, and malaria. Thus, CE is rightfully considered a neglected tropical disease. In 2005, the Food Standards Agency, in United Kingdom, reported that for many people in Central Africa, particularly those living in rural areas, bush meat is a major source of animal protein. In some regions, it provides up to 98% of the animal protein consumed. In cities, bush meat is a luxury item and usually commands a premium in price (10). Almost any wild animal is potentially a candidate for bush meat (10). A comprehensive overview of animals hunted in tropical moist forests in Central Africa showed that ungulates (antelopes, pigs and chevrotain) constituted 73.2% of all
hunted animals, rodents (grass cutter and porcupines) 12.2% and primates (sooty mangabey) 12.0% (10). Normally, the risks from parasites hazards (giardia, cryptococcus, T saginata, T solium, echinococcus, and toxoplasma) can be adequately controlled through basic kitchen hygiene, which includes taking steps to avoid cross contamination, and thorough cooking.

However, in Africa, in the preparation of the bush meat for cooking there is a risk of cross- contamination to other foods in the kitchen. The risk will depend importantly on the level of hygiene observed in the kitchen, the frequency of contamination of the bush meat and the amount of contamination of the bush meat.

The definitive host of Echinococcus granulosus is the dog but can also be wild animals used as bush meat. Humans are parasitized by the larval stage by ingesting eggs excreted by the definitive host.

Conventionally, the causative agent of cystic echinococcosis was regarded as one species, *E granulosus*. Available evidence suggests that several species or strains within the *Echinococcus granulosus* complex are prevalent in sub-Saharan Africa and that these strains might be associated with varying virulence and host preference (11). To date, ten different strains (G1–10) have been described. This reappraisal continues, but currently, cystic echinococcosis of people or animals, or both, can be caused by *E granulosus* (G1–3), *E felidis* (the so-called lion strain), *E equinus* (G4), *E ortleppi* (G5), and *E canadensis* (G6–10) (Thompson RCA. *E granulosus* is believed to be the cause of most human cases; *E equinus* is thought to be the only species that cannot infect people. Apart from *E felidis*, all strains use the domestic dog as an intermediate host (12). In our patient, we did not have tools to characterize the responsible strain. This would have helped us in identifying the contamination origin.

In conclusion, we have here described the first autochthonous infection with CE in Congo, Central Africa, providing the first evidence for a new geographical area at risk for the acquisition of CE. The aim of our paper is to contribute to the data for cystic echinococcosis available from sub-Saharan Africa, and to raise questions about present knowledge of the epidemiology of this disorder in the region.

REFERENCES
