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Research Article

Random Diagnosis of Disseminated Histoplasmosis with Histoplasma Capsulatum Var. Duboisii in Senegal's Hospital: Why Did One Have to Think about it?

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Abstract

Introduction: Histoplasmosis Histoplasma capsulatum var. duboisii (H. duboisii) is a deep mycosis plagues Africa. The clinical manifestations are dominated by cutaneous involvement; however rare cases of disseminated histoplasmosis have been described. It is an unknown affection, which poses a real diagnosis and therapeutic problem.

Observation: We report the case of a 49-year-old patient with diffuse abdominal pain predominating in the right hypochondrium and epigastrium associated with constipation and vomiting for the past 1 month, all in a context of alteration of febrile general state. Clinical examination revealed moderate, firm, homogeneous, smooth and painless hepatomegaly; stage IV splenomegaly of Hackett; painless bilateral firm bilateral cervical polyadenopathies. In this precise case, histoplasmosis was not initially evoked but was revealed by a lymph node biopsy in favor of histoplasmosis with Histoplasma capsulatum var Duboisi. The evolution was marked by the death of the patient on day 17 of hospitalization.

Conclusion: African histoplasmosis remains a little-known pathology despite the few clinical cases found. The low frequency explains most often the rambling diagnostic and the fatal evolution.

Introduction

Histoplasmosis is a fungal infection related to Histoplasma capsulatum of which there are two pathogenic varieties for humans: Histoplasma capsulatum var capsulatum commonly called "Histoplasma capsulatum" and Histoplasma capsulatum var duboisii [1,2].

The duboisii variety is responsible for African histoplasmosis, which is observed in inter-tropical Africa and presents as a skin condition [2]. H. capsulatum is a dimorphic fungus in the environment, at room temperature, its growth is filamentous.

The actual frequency of the African form is unknown, most studies have been in clinical cases [3-13]. Skin lesions, which are the most common, predominate in the thorax and the face [1,2]. The disseminated forms are rare, and are most common in immunocompromised patients. Their prevalence is probably underestimated on the one hand because the diagnosis is not mentioned in first intention on the other hand because of the limited technical platform. We report here a case of disseminated histoplasmosis whose diagnosis has been posited in a fortuitous way, illustrating the difficulties of the diagnosis of this affection.

Observation

49-year-old patient, resident of Pikine, Builder by profession with no travel history and without particular history hospitalized for diffuse abdominal pain predominating in the right hypochondrium and epigastrium, associated with constipation and vomiting. The onset of symptomatology would go back to a month marked by diffuse abdominal pain with epigastric irradiation. This clinical picture evolves in a context of asthenia and anorexia. This patient also had papulo-nodular lesions on the face and thorax. On examination at the entrance, the patient had an impairment of general condition, a temperature of 38°C and a blood pressure of 13/8 mmHg. Clinical examination found consistent homogeneous hepatomegaly with smooth painless hepatic spurs at 18 cm, Hackett stage IV splenomegaly, painless, firm bilateral cervical polyadenopathy, fistulization-inducing tendency, small diffuse papulonodular lesions on the face and thorax and ascites of average abundance.

The ascites puncture returned a citrus-yellow liquid, exudative with 22.10 g /l of protein, cellularity: leukocyte 244 IU/ml, PNN: 9%, lymphocyte: 91%. On the paraclinical level, there was anemia with a normochromic normocyte 8.70 g/dl hemoglobin; The blood ionogram was normal; the HBsAg assay is negative, the tuberculin intradermal reaction (IDRt) is anergic, the Alpha Foeto-Protein (AFP) is normal, the HIV serology has not been performed. Abdominal ultrasound showed a homogeneous hepatomegaly of 21 cm nondysmorphic without parenchymal abnormality.

An expansive 78 x 57 mm splenic tumor process heterogeneously enhanced with necrotic areas suggestive of lymphoma. The computed tomography performed in addition to the ultrasound revealed an enlarged liver with no focal anomaly on the parenchyma. A 78 x 57 mm splenic tissue expansive process with necrotic areas suspected of lymphoma.

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Given this clinical picture, the diagnostic hypotheses evoked were lymphoma and tuberculosis. Histoplasmosis was not initially mentioned. The patient received a lymph node biopsy of two lymph nodes measuring between 1 and 2 cm. Sectional slices were tissue and nodular, with no areas of necrosis.

Histological examination revealed a granulomatous inflammatory infiltrate in which there were multiple large, rounded double-contoured yeast shaped bodies in the intracellular position typical of H. duboisii. Further investigations, in particular molecular biology to confirm histoplasmosis to better specify the nature of the germ could not be achieved due to a limited technical platform. The positive diagnosis was made post mortem, the patient did not receive adequate treatment. The evolution was marked by the death of the patient on day 17 of hospitalization.

Discussion

African histoplasmosis, or large-scale histoplasmosis, occurs in Africa, on either side of the equator, between 20° north latitude and 20° south latitude. About 250 cases of African histoplasmosis are reported in the literature [1].

The clinical manifestations of African histoplasmosis are either localized, mainly cutaneous and subcutaneous, bone or ganglionic, or generalized (currently 30.5% of cases) with superficial and deep, visceral and even septicemic localizations. The disease is preceded by a period of latency often long (and this explains the appearance of late forms in transplant patients). There are no evolutionary stages, but immediately an attack of one or more organs. The apparently isolated forms are probably multifocal lesions not detected because of a complete assessment [1].

The prevalence of African histoplasmosis is unknown. Most reported studies have been in the form of a clinical case. In Africa, 237 cases were recorded before 1993 [14]. Recent publications are noted notably in Mali, Burkina, Congo, Togo and Senegal [3-13] where four cases are published between 1998 and 2016 [4,8,11]. They were of various origins.

The first was a Mauritanian trader who presented the disease six years after leaving Senegal when he had resided for 15 years in the region of Thies (Western Country). The other two were a 15-year-old and a 50-year-old. They were respectively from Casamance (South) and Kaolack (Center) without notion of travel. For the fourth patient, the place of contamination is debatable.

The cases previously described in native people testify to the existence of the disease in the national territory where they could become infected. However, the long period that can separate the infestation and the onset of symptoms may also suggest a possible contamination from his country of origin (Uganda) [4]. In our patient, no notion of contagion was found.

Cases of histoplasmosis exclusively lymph node-specific have been diagnosed but after a long period of diagnostic wandering [12,15].

Adenopathies are noted in most cases reported [10,11,12,15] but their clinical remains still variable. Sometimes it is adenitis fistulized [12,15] sometimes they are non-inflammatory [10,11]. In the case of Diadie [4], these were non-inflammatory adenopathies with a caseous appearance. This initially led to ganglionic tuberculosis, but histology and culture helped to correct the diagnosis. He thus brings the first case of ganglionic histoplasmosis with a caseous appearance to macroscopy.

In our case, the clinical and macroscopic aspect pointed towards a tumoral aspect reminiscent of a lymphoma, we also evoked a ganglionic tuberculosis considering the frequency of this pathology in our region but unfortunately, the diagnosis of histoplasmosis was evoked in spite of compatibility of clinical and macroscopic manifestations. This semiotic duality should in the future encourage systematic mycological examination for exhaustive etiological research in the presence of lymphadenopathy in the tropics.

The conventional diagnosis of histoplasmosis is based on the presence of yeast-like organisms on direct examination of clinical specimens associated with a positive culture. This culture usually takes several days or even weeks before becoming positive. The detection of biomarkers thus appears to be an asset of complementary tools for a definitive positive diagnosis [2,7,17]. T. Dieng [17] in Dakar reported a case of disseminated histoplasmosis due to Histoplasma capsulatum capsulatum in a Senegalese patient infected with HIV. The diagnosis was suspected by the presence of small yeasts encapsulated in neutrophils on a blood smear, it was then confirmed using specific software in real time. PCR applied to a DNA sample extracted from the blood smear. These biomolecular techniques are unfortunately not available in our working conditions. The visceral abnormalities that the patient presented were not formally related to histoplasmosis. At first, they made one think of lymphoma. A liver and splenic biopsy would have certified the diagnosis. African histoplasmosis of the liver or spleen is rare and severe.

Evolution is most often fatal [14,15,18]. Arlet and al. reported the first case of ascites in African histoplasmosis [16].

It was exudative ascites. Hypotheses have been made concerning the origin of the peritoneal effusion. These authors believe that ascites would result from peritoneal micro-perforations or invasion by hematogenous pathways. In Mali, a second case of ascites during African histoplasmosis has been described but its nature remains unknown because it has not been studied biologically [5]. For Diadie [4] the cytopathological study of the peritoneal effusion fluid could not be done. However, this ascites is linked to a peritoneal localization of histoplasmosis or a reaction of the latter following visceral involvement. In our patient it was exudative ascites with 91% lymphocytes.

Despite the profusion of the disease, our patient had no proven immunodeficiency, however the HIV serology was not performed in our patient.

Disseminated forms have been noted in immunocompetent, HIV-negative patients [4-11,12]. The different cases described in Africa have a variable evolution. Some were favorable [5-8,13], for others the patient died on treatment [3,9].

These deaths under treatment are often linked to a lack of means for adequate care.

In Senegal the evolution has been unfavorable in all the cases described [4,8,11].

Conclusion

African histoplasmosis remains a rare and often unrecognized condition of practitioners. Its clinical polymorphism can confuse the diagnosis and thus delay its management. However, we must always think about polyadenopathies, hepatosplenomegaly and papulonodular lesions. Diagnostic delays explain the significant number of deaths in Senegal related to histoplasmosis.

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