CASE REPORT

Histoplasmosis: An elusive re-emerging chest infection

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Abstract

An immunocompetent patient presenting with disseminated histoplasmosis and superior vena cava obstruction. Features at presentation were in keeping with tuberculosis. Histology of bronchoalveolar lavage specimen clinches the diagnosis of histoplasmosis.

Key words: Disseminated histoplasmosis, immunocompetent, superior vena cava obstruction

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Case Report

A 28-year-old Nigerian poultry farmer presented to MMSH Kano in February 2009, on account of 2 year's history of progressive dyspnea on exertion, weight loss, dry cough, neck swelling, decreased appetite, and fatigue.

There was no history of syncope or dysphagia. No history of contact with chronic cough. He had never smoked cigarettes. He was diagnosed earlier at Peripheral Hospital as sputa negative PTB; hence he was given 6-month-course DOTS treatment with little or no improvement.

Clinical examination revealed a young man wasted 48 kg, cervical lymph nodes, dyspnoic, tachypnoic at rest. HR was 120 beats per minute, RR 28 breaths per minute, and superficial vascular distention over the neck, chest, and upper abdomen (nonpulsatile). There was no stridor, but coarse basal crackles in the chest posteriorly. Abominal examination revealed hepatoslenomegaly – an impression of *superior vena cava syndrome*? Cause was entertained. Urgent chest X-ray and abdominal ultrasound were ordered,

The results showed the following.
Abdominal ultrasound and CT abdomen showed evidence of multiple abdominal masses.

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Table 1: Haematology and chemistry profile

FBC/ESR	LFTs
Hb 13.0 g/dl	Albumin 26 g/dl
WBC 8.0 × 10^9/l	Bilurbin – 6.04 mm/l
Platelets 410 × 10^9/l	Alp Phosph – 307 mg/dl
ESR 23 mm/h	SGOT – 733 iu/l
U/E/Cr	SGPT – 502 iu/l
Na – 134 mm/l	ALT – 46 iu/l
K – 4.7 mm/l	
Ch – 106 mm/l	
HCO_3 – 23 mm/l	
Creatinine – 86 umo/l	
Others	
Fasting sugar – 4.7 mm/l	
Mantoux test – 10 mm	
Urine vanyl mandelic acid – normal	
Serum ACTH levels – normal	

Provisional diagnosis of disseminated tuberculosis was entertained based on the following:

History, chest involvement, deranged LFTs [Table 1], hepatosplenomegaly and abdominal masses on CT, and

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cervical lymph nodes. Other differential diagnosis considered include fungal infection, lymphoma, thymoma, metastatic carcinoma, pheochromocytoma. Hence, further investigations were requested. A fine needle aspiration biopsy (FNAB) of cervical lymph node showed reactive changes.

Routine blood cultures were done, and no growth was found. Sputum acid fast bacilli (AFB) on three separate occasions were negative. Serology was negative for cryptococcus and HIV. Bone marrow aspiration showed mild reactive changes only. FNAB fungal staining showed yeast cells on microscopy

Broncoscopy and alveolar lavage were rewarding, as it clinches the diagnosis of disseminated histoplasmosis. Culture yields histoplasma capsulatum

He was discharged home on fluconazole 200 mg b.d. for 1 month, seen at follow-up chest clinic after 3 months, doing well, a febrile gaining weight, 53 kg in May 2009

Discussions

Superior vena cava syndrome encompasses a constellation of symptoms and signs resulting from obstruction of the superior vena cava. The increased venous pressure in the upper body results in edema of the head, neck, and aims, often with cyanosis, plethora, and distended subcutaneous vessels may be a consequence of obstruction of the superior vena cava (intrinsic or due to extrinsic compression), compression of the heart by a large mass in the chest, or both.[1] The superior vena cava carries blood from the head, arms, and upper torso to the heart; it carries approximately one-third of the venous return to the heart. When the superior vena cava is obstructed, blood flows through a collateral vascular network to the lower body and the inferior vena cava or the azygos vein. It generally takes several weeks for the venous collaterals to dilate sufficiently to accommodate the blood flow of the superior vena cava.[2,3] In humans with obstruction of the superior vena cava, the cervical venous pressure is usually increased to 20 to 40 mm Hg (normal range, 2–8 mm Hg).[4,5,6] The severity of the symptoms depends on the degree of narrowing of the superior vena cava and the speed of the onset of the narrowing. Compression of the superior vena cava may result from the presence of a mass in the middle or anterior mediastinum, consisting of enlarged right paratracheal lymph nodes, lymphoma, thymoma, an inflammatory process, or an aortic aneurysm. Infectious causes e.g. tuberculosis and syphilis accounted for the majority of cases and malignant conditions accounted for more than 90% of cases approximately in pre HIV era.

Our initial suspicion was rather that of a malignant cause of SVC obstruction; however earlier ancillary investigations were not in keeping with any of the differentials. It is customary for practising physicians to commence anti-TB medications as therapeutic trial if patients present with high index of clinical suspicion despite negative sputa testing. The time frame for the therapeutic trial is not well stated in the Nigerian national guidelines for TB treatment.

The occupational history and duration of the exposure to high inocula of poultry roost was overlooked. This could explain the occurrence of disseminated disease in the absence of immunosuppression. In addition this patient was also HIV negative; hence the diagnosis was remotely considered. Histoplasmosis is one the neglected cause of SVC. Few sporadic cases of histoplasmosis have been reported in northern Nigeria.

These cases are often seen in immunocompromised patients. However, it has been fairly common in our series to diagnose this infection authochronous to Kano State, especially in patients with initial suspicion of tuberculosis with, or without immunosuppression. Histoplasma capsulatum is a dimorphic fungus that remains in a mycelial form at ambient temperatures and grows as yeast at body temperature. Infection causes histoplasmosis, transmitted via inhalation and deposition in alveoli.[7] Most individuals with histoplasmosis are asymptomatic. Those who develop clinical manifestations are usually immunocompromised or are exposed to a high quantity of inoculum. Histoplasma species may remain latent in healed granulomas and recur, resulting in cell-mediated immunity impairment; however in our patient no signs of immunosuppression were demonstrated. The host defense includes the fungistatic properties of neutrophils and macrophages. The lymphocytes are crucial in limiting the extent of infection. Susceptibility to dissemination is increased markedly with impaired cellular host defenses Clinical manifestations of histoplasmosis appear with continued exposure to large inocula. The initial pulmonary infection may disseminate systemically, with hematogenous spread, and produce extrapulmonary manifestations. Hematogenous spread to regional lymph nodes may occur through the lymphatics or the liver and spleen. Progressive disseminated histoplasmosis is rare in adult hosts who are immunocompetent. Systemic spread usually occurs in patients with impaired cellular immunity and typically involves the CNS, liver, spleen, and rheumatologic, ocular, and hematologic systems. Progressive disseminated histoplasmosis occurs in 1 case per 2000 cases in adults who are immunocompetent.[8] It is recommended that patients with severe infection should be treated with amphotericin B; once the patient is stable, amphotericin B may be changed to itraconazole and should be continued for 1 year.[9]

The choice of fluconazole in our patient, though not gold standard of treatment, was based on the affordability and availability in our low resource setting.

This patient responded well to fluconazole and presently been followed up at the hospital chest clinic. Histoplasmosis should be suspected in patients within certain epidemiologic setting presenting with pneumonia and mediastinal or hilar lymphadenopathy, masses suggestive of malignancy on chest X-ray, pulmonary nodule, cavitary lung disease suggestive of tuberculosis or pericarditis with mediastinal lymphadenopathy. The population at risk includes farmers, poultry keepers, especially when cleaning chicken coops, pigeon roosts, and bat-infested barns or lofts, construction workers, especially those who work around old buildings with roosting birds, landscapers, archeologists, and geologists. Most of risks of severe infection can occur in infants and very young children and older adults. The risk of disseminated histoplasmosis increases with age. HIV-positive or those with AIDS, people receiving chemotherapy or long-term treatment with corticosteroid such as prednisone, people who have had organ transplants and are taking anti-rejection medications.

Conclusion

Histoplasmosis should be considered as an important differential diagnosis of DTB, particularly in individuals from endemic areas with or without immunosuppression. Clinical features may be similar to disseminated tuberculosis.

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