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Case Report

Pseudomonas aeruginosa causing inflammatory mass of the nasopharynx in an immunocompromised HIV infected patient: A mimic of malignancy

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ABSTRACT

Head and neck manifestations of acquired immunodeficiency syndrome are among the most common complications of this disease. The sinonasal and oral manifestations are more common that the otologic and range from malignancies to infectious processes caused by both opportunistic and nonopportunistic organisms. We report the case of a nasopharyngeal mass of infectious etiology in a severely immunocompromised HIV infected patient. The patient was admitted with a presumptive diagnosis of infectious gastroenteritis and was found to have a nasopharyngeal mass. The mass was extending into the oropharynx and paravertebral soft tissues and was associated with extensive secretions causing near complete occlusion of the oropharynx. CT scan findings favored malignant verses infectious etiology. The surgical biopsy performed twice ruled out malignancy and the bacterial culture proved to be a pure growth of *Pseudomonas aeruginosa*. *Pseudomonas* can inhabit the nasopharynx and lower digestive tract, and is only occasionally associated with causing disease in non-susceptible patients but is a common infection in immunocompromised patients. To the best of our knowledge, and after considering the current literature, we believe this case is unique. We discuss this rare entity and its management. Clinicians should be aware of this potential life threatening condition in the HIV population and add *P. aeruginosa* infection to the differential diagnosis of an acute inflammatory nasopharyngeal mass.

Case presentation

A middle aged patient with AIDS and history of Kaposi sarcoma, presented to our emergency department with chief complaint of watery diarrhea with blood for few days associated with periumbilical abdominal pain for 3 weeks. The patient also complained of nausea, vomiting, loss of appetite and weight loss of approximately 20 lbs, however denied fevers, chills, shortness of breath, chest pain, recent traveling or antibiotic use. The patient reported noncompliance of antiretroviral medications on initiation of the diarrhea. Vital signs noted in the ED revealed a BP of 77/66, heart rate of 128 bpm, temperature 98 degrees F and the respiratory rate 18. Blood pressure responded to fluid challenge and the patient was admitted to the medical floor for management of gastroenteritis in an HIV infected patient.

On admission to the medical floor, the patient was found lying in fetal position and appeared in mild distress. The physical exam was significant for chapped lips, a whitish coating of the tongue, bilateral cervical and submandibular palpable lymph nodes. The abdomen was soft, with tenderness to light palpation over the periumbilical region and normoactive bowel sounds. Laboratory testing revealed WBC count 4250 with 50% PMNs, hemoglobin level 14.8 g/dl, BUN 26 mg/dl and creatinine of 1.6 mg/dl. Serum amylase and lipase and chest and abdominal xrays were unremarkable. CT of the abdomen revealed diffuse colitis possibly of infectious etiology, hypoproteinemia and anasarca. The patient was empirically started on metronidazole for possible *Clostridium difficile*-associated colitis and fluconazole for the treatment of oral candidiasis. The medications were started orally and then changed

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to intravenous therapy since the patient reported difficulty swallowing. The patient was found to have CD4 count 22 and CD4/CD8 ratio of 0.039. Stool was positive for *C. difficile* toxin.

By the 6th hospital day the patient remained afebrile but tachycardic, the WBC count was trending up to 19.7 with 84% PMNs. The abdominal pain subsided but the diarrhea continued. The patient appeared toxic and lethargic, his voice became muffled with labored speech. The oxygen saturation on room air was approximately 90–92%. Food remnants were seen in the mouth and a thorough examination of the oral cavity revealed a large bulging oropharyngeal mass. Antimicrobial coverage was broadened to vancomycin and zosyn and a CT scan of the neck demonstrated a mass in the nasopharynx extending into the oropharynx and paravertebral soft tissues. Also noted were extensive secretions in the nasopharynx and oropharynx with near complete occlusion. The larynx, thoracic inlet and trachea were patent. Many bilateral prominent enhancing cervical and superior mediastinal lymph nodes were also seen (Fig. 1A and B).

The CT scan findings triggered an emergency consultation with OMS and surgery and patient underwent incision and drainage of a large palatal mass. A 16 gauge needle did not yield any aspirate and blunt dissection did not yield any exudates. The mass was cultured for aerobic and anaerobic bacteria, fungi and acid fast bacilli. Palatal biopsies were taken at the dome of the lesion away from a small necrotic area. No dental etiology was associated with the mass (Fig. 2). The patient was transferred to the intensive care unit for post-operative care, placed on mechanical ventilation and

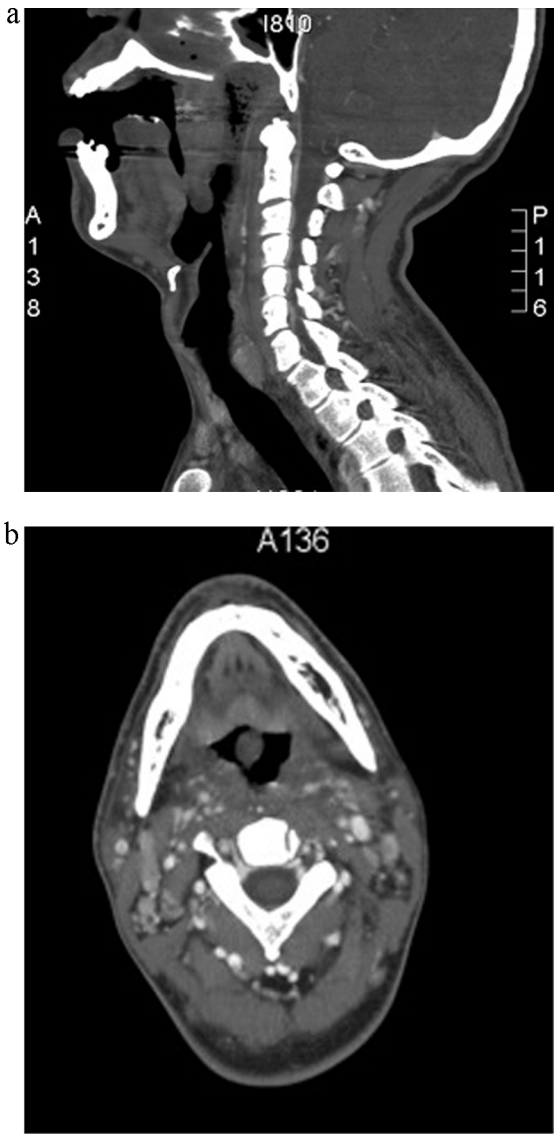

Fig. 1. (A) CT scan in sagittal view of the neck with mass in the nasopharynx extending into the oropharynx and paravertebral soft tissues. (B) CT scan in transverse view of the neck: Many bilateral prominent enhancing cervical and superior mediastinal lymph nodes.

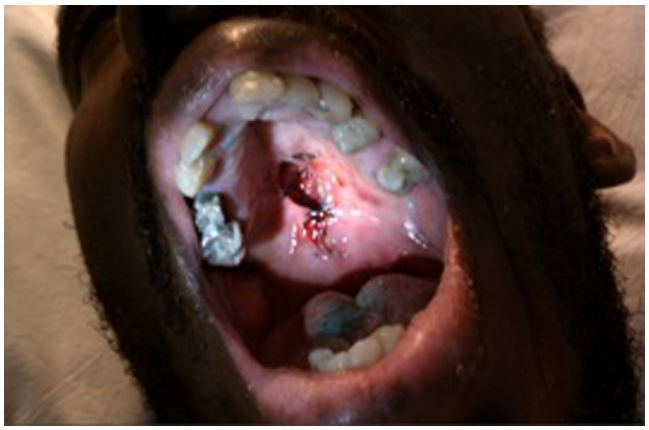

Fig. 2. Oral cavity after incision and drainage with biopsy.

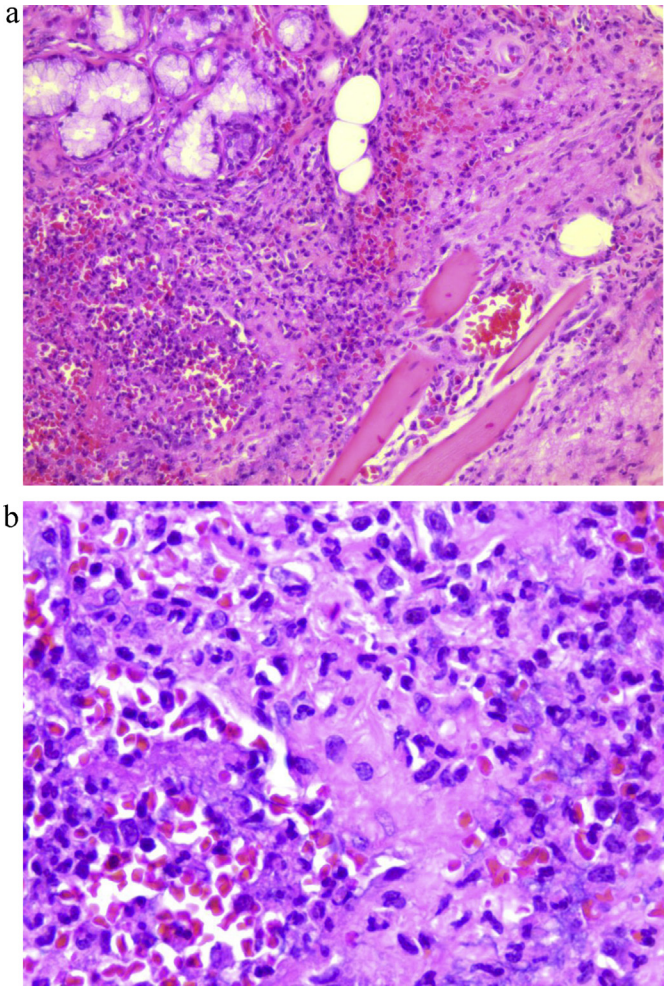

Fig. 3. (A) Nasopharyngeal mucosa showing foci of severe acute inflammation in the subjacent soft tissue and minor salivary glands (H&E, ×25) and (B) Nasopharyngeal mucosa showing severe acute inflammation with focal necrosis in the subjacent soft tissue (H&E, ×40).

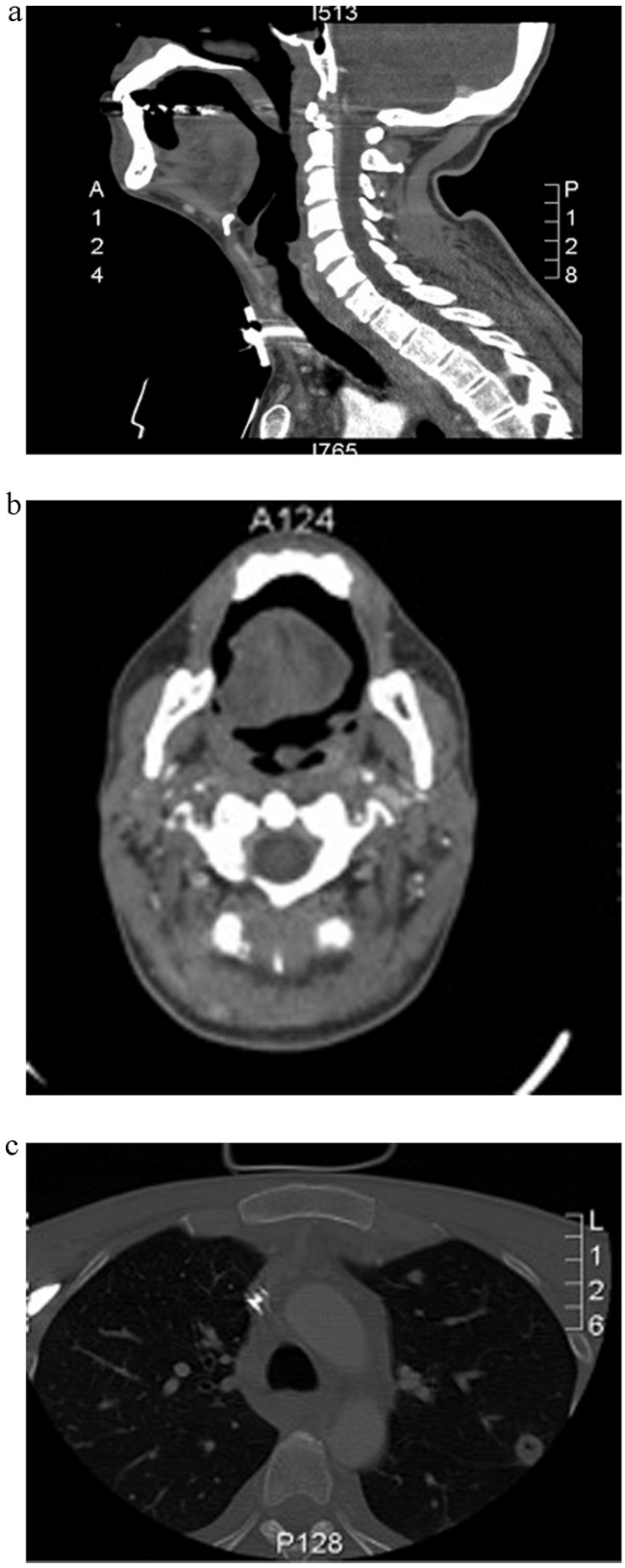

Fig. 4. (A) CT scan in sagittal view of the neck after incision and drainage. (B) CT scan in transverse view of the neck after incision and drainage. (C) CT scan in transverse view of the chest. Lesion appreciated left lobe.

advanced to CPAP and tracheostomy collar within 24 h. After the surgical procedure the WBC count trended down to 12,000, and on the 3rd postoperative day decreased to 5990.

The intraoperative specimen sent for Gram stain showed many WBC, gram negative bacilli, and the culture grew *Pseudomonas aeruginosa*. Candida glabrata also grew from the cultures. Based on culture and sensitivity findings the streamlining of the antimicrobial therapy was done on the 4th post-op day and patient was started on intravenous cefepime. The pathology showed a portion of nasopharyngeal mucosa and subjacent tissue demonstrating foci of predominantly acute inflammation, skeletal muscle and salivary glands. The overlying epithelium showed mild dysplasia (Fig. 3A and B).

On the 11th hospital day, a second biopsy was taken to rule out malignancy. The result of the second biopsy was similar to the initial pathology report, significant for foci of acute inflammation and focal necrosis. The overlying epithelium was unremarkable. No tumor was seen.

A CT scan of the neck on the 16th day of hospitalization showed a clear nasopharynx without any evidence of a mass (Fig. 4A and B). However, incidentally a lesion was found in the lung fields (Fig. 4C). The patient subsequently underwent flexible fiberoptic laryngoscopy and head and neck exam by ENT. No polyp or nasopharyngeal mass were appreciated. The nasal airways were patent and minimal soft tissue in the nasopharynx was consistent with lymphoid hyperplasia. Exam was considered unremarkable.

A CT of the chest was pursued and revealed several pulmonary soft tissue nodules in both upper right middle and right lower lobes measuring between 3 and 12 mm. Multiple small cavitating nodules also seen. The patient was placed on airborne isolation, three sputum samples sent for AFB smear and culture were negative and a follow up bronchoscopy was refused by the patient. The patient was discharged to a subacute rehabilitation facility.

Discussion

It is estimated that 40–100% of patients with HIV will have head and neck manifestations. These may be found in unusual locations and behave in a more aggressive fashion that in the HIV negative population. Lower CD4 counts and the duration of HIV infection have been significantly associated with increased prevalence of oropharyngeal conditions, with a male to female ratio of 3.4:1. [1,2]

Mass lesions arising in the nasopharynx generally cause obstruction to the passage of nasal air flow, usually in the posterior nares, resulting in nasal obstructive symptoms and with further growth nasopharyngeal masses can also extend anteriorly into the nasal cavity and present as a mass protruding through the nostrils. Associated symptoms include nasal blockage, anosmia, nasal discharge and intermittent epistaxis. If the mass extends inferiorly it can present as a mass in the oropharynx which pushes the soft palate forward and the typical presentation will include snoring or stertor or a hypo nasal quality of speech [3–7] as in our patient. In our case the nasopharyngeal mass was initially highly suspicious for a malignant process but the biopsies only showed acute inflammation and focus of necrosis.

The prevalence of nasopharyngeal manifestations in the HIV population ranges from 11% to 70% in different studies; among the malignancies the most common are the virally induced AIDS defining cancers: Kaposi sarcoma and non-Hodgkin lymphoma (NHL), especially large B-cell and plasmablastic lymphomas, and as more people are living with chronic HIV infection, the incidence of non-AIDS defining cancers including the nasopharyngeal carcinomas have been increasing. The most frequent benign lesion of the nasopharynx in the presence of HIV infection is benign lymphoid hyperplasia also referred to as nasopharyngeal lymphatic tissue hypertrophy (NHLT). Infection with HIV, Epstein–Barr virus or cytomegalovirus cause proliferation of B cells in lymphoid adenoidal tissue, occurring in 56–88% of patients in early HIV

infection and decrease in size with immune system impairment [8]. The usual presenting symptoms are nasal obstruction and recurrent serous otitis media secondary to Eustachian tube obstruction. The presence of this lesion in an otherwise asymptomatic adult patient should always raise the suspicion of HIV infection [3,7–10]. Rhinosinusitis is also very common in HIV seropositive patients and those with advanced immunodeficiency usually develop posterior sinus disease [11,12].

Several studies have shown that, in the HIV-infected population, the organisms causing acute sinonasal infection are the same as in the immunocompetent population. However, when the CD4 cell count drops below 200 in the HIV-infected cohort, more opportunistic pathogens can be seen including fungi and bacteria such as *P. aeruginosa* [5,6,8,9,11,13,14]. Patients with AIDS are at increased risk of developing serious life threatening infections and in this population *P. aeruginosa* is an important opportunistic bacterial pathogen that can cause recurrent community acquired sinus pulmonary disease even in the absence of traditional risk factors like corticosteroid use, recent antibiotic exposure, prior hospitalization, neutropenia and PCP prophylaxis with trimethoprim-sulfamethoxazole [14–18].

P. aeruginosa infect mainly those who have a weakened immune system and accounts for up to 17% of acute sinusitis and 20% of chronic sinusitis cases in the HIV infected population [19,20]. It is not clear if the sinus is acting as a nidus for infection or if the diffuse abnormality of the respiratory tract mucosa found in advanced HIV disease [21] and the impaired mucociliary transport predispose to infection with the organism [14].

We postulate that our patient had a lymphoid hyperplasia and became superinfected with *P. aeruginosa* causing a localized nasopharyngeal inflammatory mass with some obstructed oropharynx due to the accumulation of extensive secretions and debris [22] that was unable to be cleared. No cases of *P. aeruginosa* causing such an inflammatory nasopharyngeal mass could be found previously reported in the literature.

Our patient responded to the antimicrobial therapy directed to the specific bacterium isolated from the nasopharyngeal mass and the significant clinical response led us to assume that the cavitary pulmonary nodules were also caused by *P. aeruginosa*. We hypothesize that the initial source was the nasopharynx since the patient initially presented without clinical or radiological signs of pulmonary infection and the first CT scan did not reveal any lesion or cavity in the lungs. The clinical course also correlated with the literature report of patients with cavitary *P. aeruginosa* infection having a less severe disease that those without cavities [23]

In summary infections of the upper respiratory tract in patients with AIDS may present with paucity of symptoms due to poor inflammatory response and may progress to a more threatening condition with obstruction of the oropharynx like in our patient. *P. aeruginosa* is an important opportunistic bacterial pathogen that causes infection of the respiratory tract and other organs in susceptible hosts and can as well present as an acute inflammatory mass of the oropharynx.

Conflict of interest

None declared.

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