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Case report

Pulmonary nocardiosis with a rare association of syndrome of inappropriate antidiuretic hormone secretion (SIADH) in an immunocompetant male

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Abstract

Pulmonary nocardiosis and syndrome of inappropriate antidiuretic hormone secretion (SIADH) is an interesting association that has been rarely described in the literature. SIADH may be the initial sign of central nervous system progression. Here we present one such rare association of pulmonary nocardiosis and SIADH in an immunocompetent male, successfully managed with treatment of underlying disease and fluid restriction. High index of clinical suspicion even in immunocompetant hosts and effective management of associated complications is essential for successful outcomes.

Key words: Filamentous bacilli, Hyponatremia, Immunocompetent host, Pulmonary nocardiosis, Syndrome of inappropriate antidiuretic hormone (SIADH), Trimethoprim - Sulfamethoxazole (TMP-SMX)

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Nocardia asteroides is a strictly aerobic, gram-positive, branching filamentous bacillus which is weakly acid-fast. Nocardia is a saphrophyte and can be found in soil, decomposing vegetation and organic matter and belongs to family nocardiaceae and order aerobic actinomycetales[1,2]. Nocardia species are responsible for superficial skin and pulmonary infections, dissemination to central nervous system (CNS) and other organs in patients who are immunosuppressed. Lungs are the primary sites in more than 2/3[rd] of cases[3]. Acquired through inhalation, pulmonary nocardiosis presents as subacute or chronic infection and is more frequently seen in bronchiectasis and other structural lung abnormalities as they are more susceptible to colonization by Nocardia species[4,5]. Infections in immunocompetent hosts are localized and are chronic in nature in contrast to hematogenous dissemination involving brain (multiple brain abscesses) and skin which occurs in 15 to 40% of cases and carries a high mortality[1,3,6,7].

Pulmonary nocardiosis and syndrome of inappropriate antidiuretic hormone secretion (SIADH) is an interesting association that has been rarely described in the literature. SIADH may be the initial sign of central nervous system progression. Here we present one such rare association of pulmonary nocardiosis and SIADH in an immunocompetant male, successfully managed with treatment of underlying disease and fluid restriction.

Case report

A 64 year old man presented with symptoms of cough with purulent expectoration, streaky hemoptysis, shortness of breath and fever of 2 months duration. He was not a known diabetic or hypertensive. He was negative for HIV and HBsAg status. He was diagnosed with sputum positive pulmonary tuberculosis 2 years back and received anti-tubercular therapy for 6 months.

On examination he was drowsy with altered sensorium but responding to commands. He was afebrile and tachypneic with a respiratory rate of 32/min, pulse rate of 98/min. He was hemodynamically stable and his arterial blood gases were within normal limits. Cardiac examination was normal. There was no organomegaly and CNS examination showed no focal deficits. Blood investigations revealed elevated total leukocyte counts of 16,700/mm^3. His renal, thyroid and hepatic parameters were within normal limits. He was evaluated for his drowsiness and electrolytes revealed low serum Na^+ of 125 meq/L, serum K^+ of 4.2 meq/L and Cl^- of 94 meq/L. His serum osmolality was 264 mosm/L and urine osmolality was 656 mosm/L which was consistent with the diagnosis of syndrome of inappropriate antidiuretic hormone secretion (SIADH). Further investigations revealed urine Na^+ of 190 meq/L and urine K^+ of 33.3 meq/L. Sputum examination on gram stain showed plenty of pus cells with negative status for fungal and AFB stains.

His chest radiograph showed fibrocalcific opacities in right upper zone and left perihilar and mid zone consolidation (Fig 1A). Contrast enhanced CT (CECT) of the chest revealed fibrosis in right upper lobe which is the sequelae of the old tuberculous lesion (Fig 2B). CECT thorax also revealed heterogenously enhancing mass lesion in anterior segment of left upper lobe along with adjacent patchy consolidation and reticulonodular infiltrates in the lingular segment (Fig 2A, 2C & 2D). He underwent bronchoscopy which revealed inflamed left upper lobe bronchus with thick purulent secretions oozing out. Bronchial washings cytology revealed suppurative inflammation with negative status for bacterial, fungal and AFB cultures. He underwent fine needle aspiration cytology (FNAC) of the left lung lesion which yielded pus. FNAC also revealed Gram positive filamentous structures amidst pus cells (Fig 3) which was suggestive of nocardia that was later confirmed on culture by bright yellow colonies on LJ medium.

He was diagnosed of pulmonary nocardiosis with an association of SIADH. Treatment was started with Trimethoprim - Sulfamethoxazole (TMP- SMX) 15mg/kg bodyweight along with Meropenam and fluid restriction. Sodium plasma levels improved from 125meq/L to 136meq/L after fluid intake was restricted. Once the clinical stability is achieved, he was switched to oral TMP- SMX and continued for a period of six months.

Upon clinical and radiological improvement he was discharged from the hospital. Repeat chest radiograph after one month showed clearing of left lung consolidation with residual fibrosis (Fig 1B).

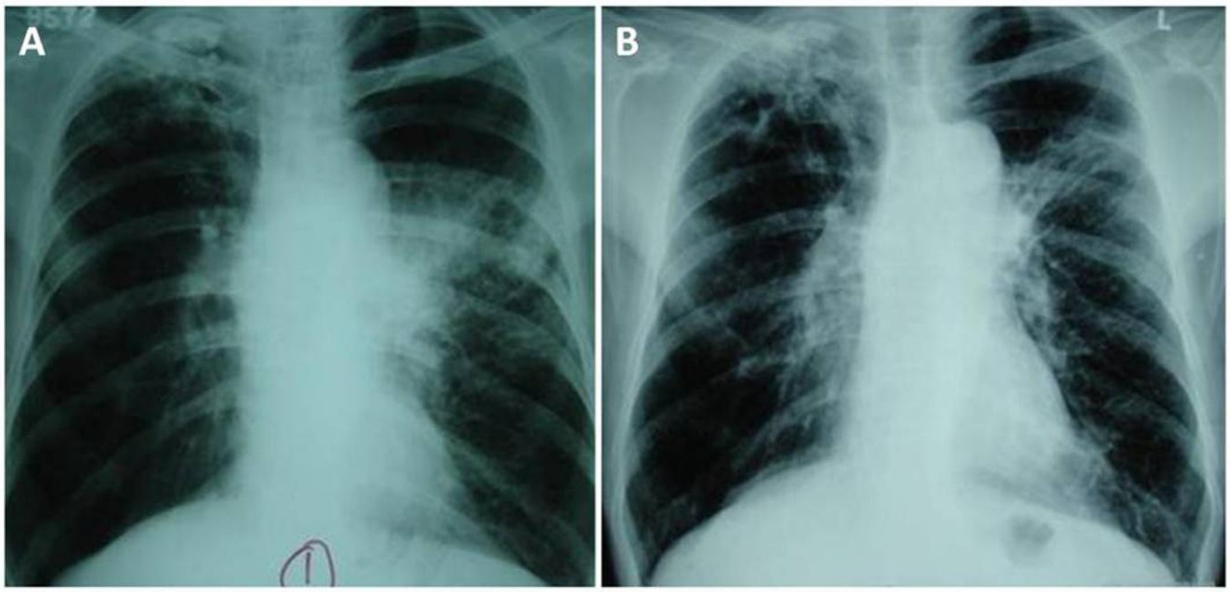

Fig 1. (A) Chest radiograph frontal view shows fibrocalcific opacities in right upper zone and left perihilar and mid zone consolidation. (B) Partial resolution of infiltrates in the left lung with residual fibrosis after treatment

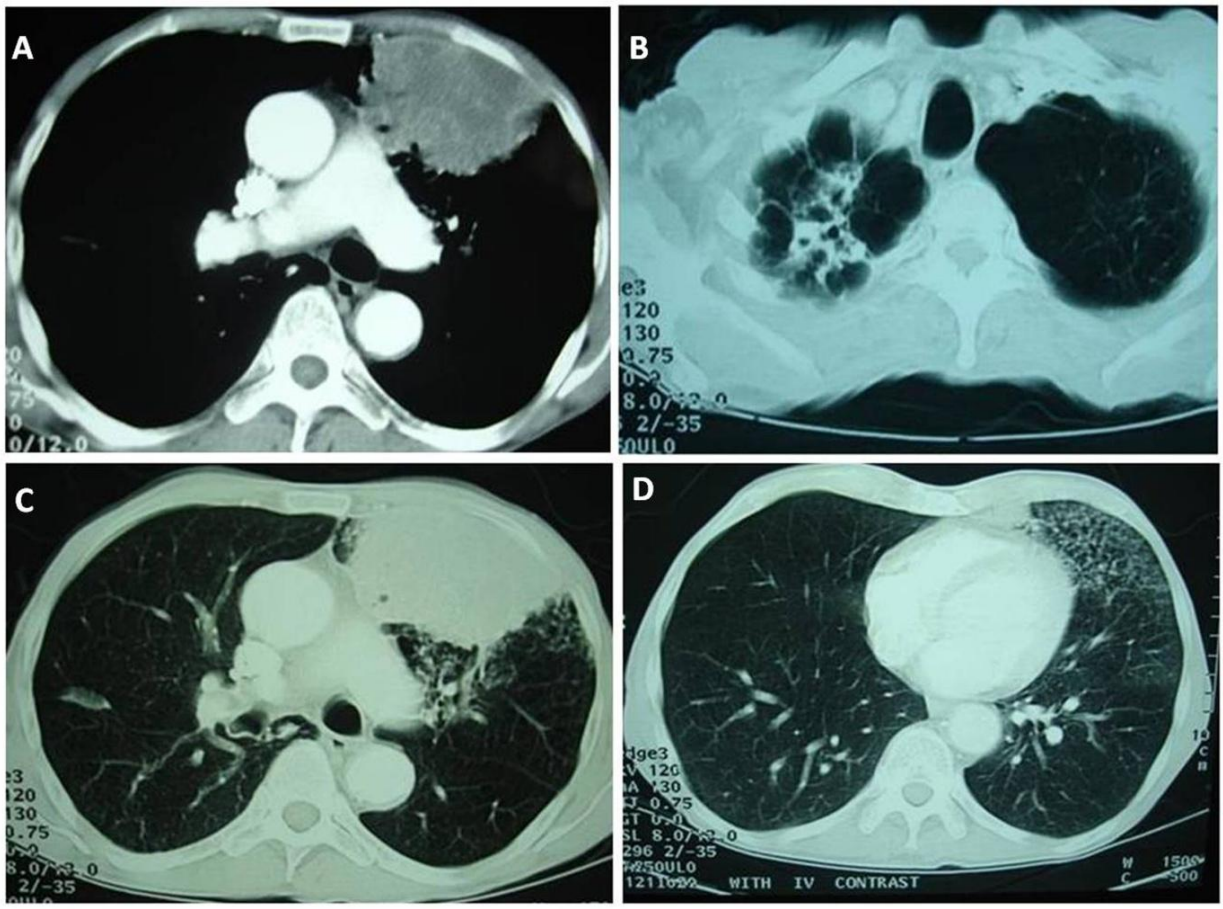

Fig 2. CECT chest. (A) Mediastinal window shows heterogenously enhancing mass lesion in anterior segment of left upper lobe. (B) Lung window shows fibrosis in right upper lobe. (C & D) Mass lesion in anterior segment of left upper lobe with adjacent reticulonodular infiltrates in lingular segment

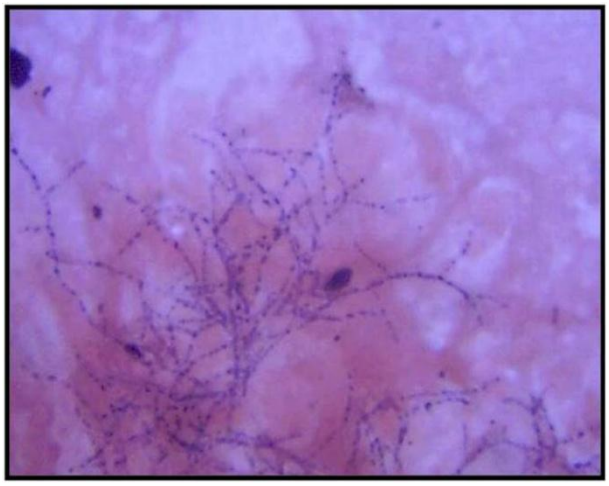

Fig 3. Smear showing branching filamentous bacteria of Nocardia species. (Hematoxylin and Eosin stain 1000x)

Discussion

The diagnosis of pulmonary nocardiosis can be delayed and is often misdiagnosed as tuberculosis, lung malignancy or invasive fungal infection due to non specific clinical and radiological presentation[7,8]. The disease in immunocompetent hosts, most commonly occurs in patients with underlying pulmonary disease like malignancy, COPD and mycobacterial infections as seen in our case[5]. Clinical symptoms include new onset cough with or without sputum production, shortness of breath, chest pain, fever, hemoptysis and weight loss[1,5]. The predominant radiological manifestations include patchy or multifocal air space consolidation along with nodules or masses with or without cavitation. Nodules may be solitary or multiple and a halo of ground glass opacity is often seen. Cavity with surrounding centrilobular nodules suggesting endobronchial spread is also reported. Other manifestations include pleural effusions and empyemas[5,9]. A study by Rossman et al[6] observed discrete nodules and wedge shaped pleural based consolidations as the most frequent radiological findings.

Differential diagnosis includes bacterial lung abscesses, tuberculosis, actinomycosis, rhodococcus, lung malignancy, aspergillosis and other opportunistic fungal infections[1]. Mycobacetria

can be differentiated from nocardiosis as they do not stain well with gram stain and modified acid-fast stain and they are microscopically different from Nocardia. Nocardia has a "beaded" acid-fast appearance on microscopy[1] where pink colored filamentous, beaded and branched bacilli appearing in modified acid-fast stained smear of respiratory secretions and specimens[2,8,10]. Typical Nocardial colonies are usually seen after 3 to 5 days, although growth may take from 48 hours to 3 weeks to appear. Colonies develop at 35–37^{0}C and can vary in color depending on the different species[3]. Nocardial colonies usually have a chalky white or cotton ball appearance because of the presence of abundant aerial filaments[2,11]. Antibiotic susceptibility varies from species to species. So species identification by culture and molecular techniques is important[12].

Sulfonamides like Trimethoprim-Sulfamethoxazole (TMP-SMX) are the treatment of choice in nocardiosis for the past 60 years[3,13]. The recommended dose of TMP-SMX is 5–10 mg/kg (trimethoprim) and 25–50 mg/kg (sulfamethoxazole) in two to four divided doses[12,14]. Other alternative antibiotics include carbapenams like meropenam, imipenam, aminoglycosides like amikacin and moxifloxacin, ceftriaxone and linezolid are promising[12].

TMP-SMX monotherapy is recommended only in immunocompetent patients with mild disease[3]. Sulfonamide monotherapy is reported to have high mortality[7], treatment failures and relapses in patients with severe and disseminated disease[3,13]. So an initial combination therapy with two or more active agents is recommended for these patients[3]. Imipenam in combination with amikacin along with sulfonamides is indicated in cases of disseminated nocardiosis, CNS involvement or severe illness[6,12].

The overall mortality in patients with pulmonary disease is in the range of 15–30%, including those who are immunocompromised[8]. So early diagnosis and appropriate therapy are essential for successful outcomes. The duration of therapy is variable and depends on the site of the lesions and the patient immune status. The duration of therapy is at least 6 months in immunocompetent patients with pulmonary nocardiosis[3,8].

Pulmonary nocardiosis and SIADH is an interesting association. SIADH is defined by the hyponatremia and hypo-osmolality resulting from inappropriate, continued secretion or action of the hormone despite normal or increased plasma volume, which results in impaired water excretion[15]. It is best defined by the classic Bartter-Schwartz criteria[16] and accounts for approximately one-third of all cases of hyponatremia[17]. SIADH may be secondary to malignancies particularly lung and pulmonary conditions like bacterial pneumonia, fungal and mycobacterial infections, asthma and cystic fibrosis. Other causes include conditions that dysregulate ADH secretion in the CNS and drugs that increase ADH secretion[15].

The only definitive treatment of SIADH is elimination of its underlying cause. The most important factors dictating the management of SIADH are the severity of the hyponatremia, its duration, and the presence or absence of symptoms[15]. As the patient was having moderate hyponatremia with less severe symptoms, fluid restriction estimated on the basis of urinary and plasma electrolyte levels was initiated and he responded well to the treatment.

In 1999, Rámila et al[18] reported the first case of pulmonary nocardiosis presenting with SIADH where in brain abscesses were found on CT. The patient was a known case of B-cell chronic lymphocytic leukemia and was treated with chlorambucil and prednisone for 6 months. Mencía Sánchez et al[19] in 2006 reported second such association in an immunocompetent old female. However there were no CNS abnormalities. As neurological manifestations are often silent and subtle, routine cranial CT scanning in all patients diagnosed as having nocardial pulmonary infection with or without neurologic signs or symptoms is recommended[13].

Conclusion

The present case is 3rd such association to be reported in the world. Our case is an immunocompetent male with isolated pulmonary nocardiosis with no CNS involvement. The patient responded well to the treatment and survived. High index of clinical suspicion even in immunocompetent hosts and effective management of associated complications is essential for successful outcomes.

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Conflict of interest: None

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