



SIADH as Presenting Feature in a Male with Coexisting Sarcoidosis and Systemic Lupus

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Abstract

We report a 45 year old man who presented with history suggestive of chronic meningitis. Laboratory investigations revealed lymphocytic meningitis and a syndrome of inappropriate antidiuretic hormone secretion. History, etiological workup and complications during hospitalization suggested the presence of both SLE and sarcoidosis. He improved with intravenous steroids and is doing well on follow-up.

Key Words: Sarcoidosis, systemic lupus erythematosus, coexisting, SIADH, chronic meningitis

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Introduction

Sarcoidosis is a female-predominant multisystem granulomatous illness, commoner in the Scandinavian countries [1]. A majority of patients develop respiratory system involvement [1]. Neurological involvement in sarcoidosis occurs in 5-15% of patients [2,3]. Neuropsychiatric manifestations in systemic lupus erythematosus [SLE] range from 25-70% [4]. Three percent have neuropsychiatric manifestations as the initial presentation. We report a rare case of coexisting sarcoidosis and SLE in a 45 year old male who presented with chronic meningitis and syndrome of inappropriate secretion of antidiuretic hormone [SIADH].

Case

This man had presented to us with low grade fever of 1 ½ months' duration. Headache, neck pain, projectile vomiting and blurring of vision were present for 15 days prior to admission. There was history of loss of vision in the left eye and hesitancy in passing urine for 3 days. He had suffered from large and small joint arthralgia during the preceding year for which he had been using over-the-counter analgesics. There was significant loss of weight and appetite during the preceding 5 months. He had discontinued alcohol and smoking 5 years ago. On examination, he was afebrile, pale and drowsy, with generalized muscle wasting, neck stiffness, non-reacting left pupil and hepatomegaly. Hyper-pigmented oval papules were observed on the legs [Figure1]. The ophthalmologist's opinion was that of granulomatous uveitis in the left eye. He was unwilling for skin biopsy at that time.

Pending results, the patient was empirically initiated on anti-tubercular treatment, intravenous ceftriaxone and dexamethasone. Investigations revealed hyponatremia and lymphocytic meningitis [Table 1]. Computed tomography [CT] of brain was normal [Figure1]. Workup for hyponatremia was suggestive of SIADH [Table1]. At 72 hours, ampicillin was added to his regimen, without improvement. On the 4th day, he developed a right sided parotid swelling that was treated with fluids and analgesics. On 7th day, angiotensin converting enzyme [ACE] levels, antinuclear antibody [ANA,1:1000 (420units/mL), homogenous pattern] and dsDNA titers[1:20] were available. Intravenous methyl prednisolone [1g/day x5] was initiated followed by oral prednisolone [1mg/kg] with rapid improvement in CNS symptoms and hyponatremia. On reviewing his history, he had had oral ulcers and photosensitivity during the previous year. High resolution CT chest showed bilateral lower lobe interstitial lung disease [ILD] with tree-in-bud appearance [Figure 1]. He was unwilling

for a meningeal biopsy. He was discharged on day 21. Contrast MRI brain one week after discharge revealed meningeal enhancement [Figure1]. Due to technical reasons, bronchoalveolar lavage could be performed only 10 days after discharge from hospital and was noncontributory.



Figure 1.

A-Right leg with oval hyperpigmented papules with improvement one week later,

B-CXR at discharge, revealed right hilar prominence

C-CT chest showed ground-glass appearance, tree-in-bud appearance.

D-MRI of brain with contrast, performed after discharge from hospital, revealed focal meningeal enhancement

E-X ray hands exhibited periarticular osteopenia without erosions.

Discussion

Central nervous system [CNS] features can either be induced by SLE itself or occur due to secondary complications [eg., hemorrhage] [5]. Headache, cognitive dysfunction, seizures and stroke are the commonest CNS manifestations in SLE while depression and psychosis are less common [2,5]. Aseptic meningitis is seen in 0- 5% in most series [5]. Meningeal disease, cranial neuropathies and hypothalamic pituitary dysfunction are the classic signs of neurosarcoidosis [6]. Manifestations of meningitis like fever, vomiting, headache are more common with cryptococcal meningitis in sarcoidosis when compared to neurosarcoidosis alone [3]. India ink staining and cryptococcal latex agglutination test were twice negative. Cranial nerve palsies are the commonest neurological deficits in sarcoidosis [6]. The 2nd, 5th and 8th are the nerves commonly involved [6]. Chronic meningitis is the usual cause of 6th and 8th cranial nerve palsies. Cranial nerves apart, hypothalamic, cerebral and cerebellar involvements are also seen.

Features of hypothalamic-pituitary involvement include insomnia, diabetes insipidus, SIADH, hypothyroidism, hypoadrenalism and hyperprolactinemia [7]. SIADH was also contributed by chronic meningitis in our patient. Cerebrospinal fluid [CSF] lymphocytosis and elevated protein is seen in neurosarcoidosis. Lupus with neurological involvement or tacrolimus treatment has caused SIADH in some case reports [8]. ADH levels were not measured in our patient due to its unavailability. SIADH as an initial manifestation of concurrent SLE and sarcoidosis has not been reported previously.

Serum ACE levels in neurosarcoidosis have low sensitivity rates [6]. Our patient's ACE levels were performed while patient was on steroids which can itself reduce ACE levels. Extremities usually have plaques or maculopapular eruptions like in our patient. The other dermatological manifestations in sarcoidosis are lupus pernio, nodules and erythema nodosum [1]. Coexistence of both SLE and sarcoidosis was first reported in 1945 [9]. Few other case reports are available but without similar presentations. Other autoimmune disorders associated with sarcoidosis include rheumatoid arthritis, thrombocytopenia, hemolytic anemia, Sjogren's syndrome, thyroiditis and uveitis [9]. Sarcoidosis can precede, follow or occur concurrently with SLE in a given patient.

Abnormal CXR is seen only in 30% of neurosarcoidosis [7]. ANA positivity, cryoglobulinemia and hypergammaglobulinemia are common to both SLE and sarcoidosis, while hypocomplementemia is more commonly seen in SLE [10]. Our patient's complement levels were normal. The diagnosis of such coexisting diseases relies not on immunological tests but on a combination of clinical and histopathological features. This man had two diseases that have high female predominance and had nonerosive polyarthritis, ILD, chronic meningitis, SIADH that are common to both entities. His antibody titers, oral ulcers and photosensitivity favored SLE, while granulomatous uveitis, parotitis, lymphadenopathy, ACE levels and normocomplementemia favored sarcoidosis.

In conclusion we report chronic meningitis with SIADH as the initial manifestation of coexisting sarcoidosis and SLE in a male patient, a presentation that has not been previously described in literature. In countries like ours, where tuberculosis is the commonest cause of chronic meningitis, multiple etiologies need to be considered in the differential diagnosis.

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