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An Interesting Case of Influenza A in A Patient with Severe Neurosarcoidosis Mimicking A Neurosarcoid Flare

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Abstract

Sarcoidosis is a multisystem disease with involvement of the nervous system occurring in about 5-10 % of patients. Neurosarcoidosis most commonly affects cranial nerves but can also involve other nervous system tissues including meninges, brain parenchyma, spinal cord and peripheral nerves. This is an interesting case of a 51-year-old woman with a history of neurosarcoidosis with known involvement of brain parenchyma and cervical spine who presented with severe and symptomatic hyponatremia. She was found to have Influenza A complicated with superimposed bacterial pneumonia. In the absence of a previous history of chronic hyponatremia, endocrine or renal abnormalities we suspect the hyponatremia to be secondary to SIADH likely caused by Influenza A and bacterial pneumonia infections and less likely due to Neurosarcoidosis. We believe neurosarcoidosis is a less likely etiology in our case since our patient had Neurosarcoidosis for 4 years but severe, symptomatic hyponatremia of this sort was never reported since the time of diagnosis. The possible mechanisms of influenza A virus, the novel coronavirus (SARS-COV-2) or other viral infections causing hyponatremia are discussed in detail. Also, we believe that evaluation of altered mental status or any other neurologic dysfunction in a patient with Neurosarcoidosis should be thorough and other etiologies including infectious or endocrine causes should be highly considered. This case also highlights the importance of seasonal vaccinations especially in sarcoidosis patients to avoid such complications.

Keywords: Neurosarcoidosis; Influenza A virus; Novel coronavirus (SARS-COV-2); SIADH; Hyponatremia; Neurosarcoid lesions.

Introduction

Sarcoidosis is a multisystem disease typically affecting the lungs and mediastinal lymph nodes with involvement of the Central nervous system (CNS) occurring in about 5-10 % of patients [1]. CNS involvement can be the first manifestation of sarcoidosis in up to 50% of patients [2]. Neurosarcoidosis most commonly affects cranial nerves but can also involve other nervous system tissues including meninges, brain parenchyma, spinal cord and peripheral nerves [3]. Involvement of the spinal cord is approximated at 0.3-0.4% with predominant involvement of the cervical and thoracic spinal cord [4]. Our case is unique in-terms of severity of hyponatremia [5,6] which we suspect is secondary to SIADH likely caused by influenza A virus infection versus the presence of neurosarcoid lesions in the pituitary gland, or could be a combination of both.

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Case Presentation

A 51-year-old woman with past medical history of neurosarcoidosis with known involvement of lungs, mediastinal lymph nodes and skin who was transferred from an outside hospital for further management of symptomatic hyponatremia. Briefly, the patient presented with flu like symptoms including cough, myalgia, fatigue and was found to have influenza type A. Laboratory work was significant for hyponatremia, sodium of 121mEq/L [Reference range (RR) 135-145 mEq/L] after which she was initially treated with intravenous normal saline and a repeat sodium was noted to be worse at 116 mEq/L. Additional laboratory work obtained was significant for a serum osmolality of 238 mOsm/kg [RR: 285-295 mOsm/kg], urine osmolality 551 mOsm/kg [RR: 500-850 mOsm/kg], urine sodium 97 mmol/L [RR: 20-40 mmol/L], creatinine was noted to be noted to be 0.45 mg/dL [RR: 0.67-1.17 mg/dL) this admission, baseline creatinine was 0.3 and TSH: 0.49 IU/ml. Based on history and laboratory findings, we strongly suspected the severe hyponatremia to be secondary to Syndrome of inappropriate antidiuretic hormone (SIADH) . Hyponatremia and influenza were appropriately treated with hypertonic (3%) saline and oseltamivir respectively. MRI imaging of the brain and cervical spine were performed to rule out new sarcoid lesions at our institution which revealed several intracranial findings. There was a T2 bright lesion at the craniocervical junction corresponding to a focus of cervical neurosarcoidosis [Figure 1a, 1b]. Outside hospital imaging from two years prior was obtained which also demonstrated a similar T2 bright lesion. On contrast enhanced imaging, the patient's craniocervical junction lesion did not demonstrate significant contrast enhancement [Figure 2], whereas on imaging from two years prior, there was bright central contrast enhancement [Figure 3]. This difference may reflect a decrease in inflammatory changes within the lesion during the patient's current hospital admission. Additionally, there was thickening of the pituitary gland with leftward deviation of the infundibulum [Figure 4]. Although no large discrete mass lesion was visualized within the pituitary gland, the leftward deviation of the infundibulum was thought to be secondary to a small focus of pituitary sarcoidosis versus a microadenoma within the right aspect of the pituitary fossa. Gradually all of her symptoms including altered mental status, agitation, lethargy and confusion resolved. Neurological examination had returned to normal and she was discharged with close outpatient follow-up.

Diagnostic assessment:

Due to the relevance to our case, it is important to discuss a brief course of patients past neurosarcoidosis treatment. The patient was diagnosed with neurosarcoidosis four years ago when she initially presented to the hospital with headache and blurry vision after which MRI brain was obtained that showed brainstem medullary lesion. She also underwent lung biopsy that confirmed the diagnosis of sarcoidosis. Initial

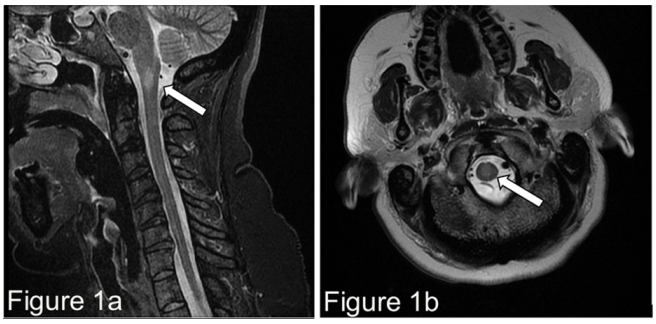

Figure 1a: Sagittal and 1b. Axial T2 weighted images demonstrating mild T2 hyperintensity at the craniocervical junction, corresponding to patient's site of cervical neurosarcoidosis.

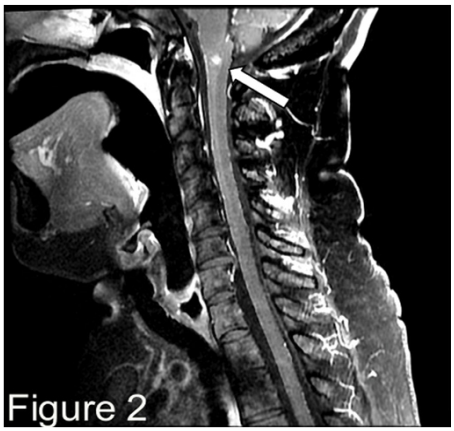

Figure 2: Sagittal T1 weighted image with contrast demonstrating focus of central contrast enhancement within the neurosarcoid lesion at the craniocervical junction. This intense central contrast enhancement was seen on patient's comparison exam from 2 years prior.

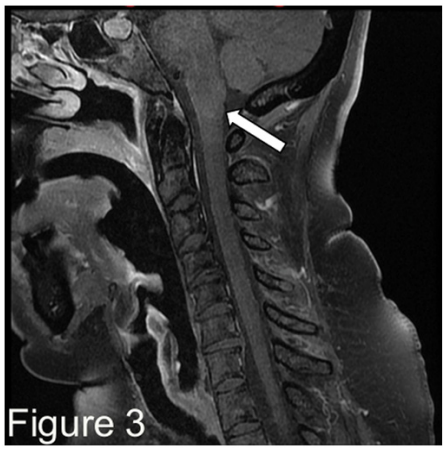

Figure 3: Sagittal T1 weighted image with contrast demonstrating interval decreased conspicuity of the focus of contrast enhancement at the craniocervical junction on our patient's imaging from her current admission. This finding suggests decreased inflammatory changes within the lesion at this point in time.

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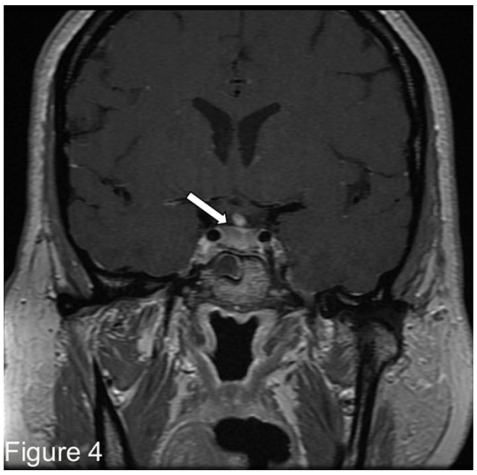

Figure 4: Coronal T1 weighted imaging with contrast through the pituitary gland demonstrated mild thickening of the pituitary gland with leftward deviation of the infundibular stalk suggesting a focus of sarcoidosis within the right aspect of the gland versus a small microadenoma.

treatment course involved high dose corticosteroids which demonstrated decreased inflammation of the brain lesion. Later on, patient received cyclophosphamide for about four months which she discontinued herself. Over the course of last few years, she continued to have severe headaches and nystagmus was manifestations of neurosarcoidosis hence she was switched from Prednisone to Methyl-prednisone along with the addition of Leflunomide. Patient reported worsening headaches and requested leflunomide to be discontinued. At that time, labs were significant for consistently low CD4 counts that showed gradual improvement during the course of time. She followed up with both the neurology and rheumatology services as outpatient with MRI brain every six months which remained stable but her Cervical spine MRI re-demonstrated a similar lesion at the craniocervical junction.

Patient was advised to slowly taper Methyl-prednisone and switch to other steroid sparing agents but she decided to stay on Methyl-prednisone while understanding the risks of long term steroid use. Since then, her overall symptoms appeared stable till this admission.

Discussion

Herein we present an interesting case of Neurosarcoidosis involving the brain parenchyma, pituitary gland and the cervical spine on monotherapy with steroids who presented with severe, symptomatic hyponatremia. She was found to have influenza A complicated with superimposed bacterial pneumonia. In the absence of chronic hyponatremia, endocrine or renal abnormalities we suspect the hyponatremia to be secondary to SIADH. The etiology of SIADH is broad and includes pulmonary disease particularly pneumonia, certain medications e.g. antidepressants, anticonvulsants, antipsychotic agents, cytotoxic agents and pain medications[7], surgical procedures, certain malignancies e.g. head and neck cancers, extrapulmonary small cell carcinomas, etc. We believe that the etiology of SIADH in our patient is likely due to Influenza A infection with subsequent super imposed bacterial pneumonia.

Diabetes insipidus is more frequently seen with neurosarcoidosis [8] and the development of SIADH in these patients is rare but a few cases have been reported in literature [9]. A few mechanisms causing SIADH have been described; one being vasculitis induced stimulation of the hypothalamic pituitary (HPA) axis causing SIADH. Hydrocephalus-induced impaired osmotic regulation as the underlying mechanism for the development of SIADH has also been described. Another mechanism noted was of Neurosarcodisosis causing SIADH was likely due to granulomas involving hypothalamic-pitituary system [9]. We believe neurosarcoidosis is a less likely etiology in our case since our patient had Neurosarcoidosis for 4 years but severe, symptomatic hyponatremia of this sort was never reported since the time of diagnosis.

Viral infections like influenza type A and more recently the novel coronavirus (SARS-COV-2) are known to cause SIADH [10,11,12,13,14]. The underlying mechanism is thought to be related to the secretion of proinflammatory cytokines such as Interleukin (IL)-2, 6, 1β, and Tumor Necrosis Factor (TNF)-α stimulating the human hypothalamic-pituitary-Adrenal (HPA) axis which have been reported to stimulate parvocellular and magnocellular Arginine Vasopressin (AVP) neurons to secrete more Anti Diuretic Hormone (ADH); hence, causing SIADH [15]. Per literature review, a study by Mastorakos et al. revealed that IL-6 activates the magnocellular ADH-secretory neurons thus leading to SIADH [16]. Lim et al. also suggested that severe inflammation causing elevated levels of IL-6 and IL-1β may activate ADH causing SIADH and resultant hyponatremia [17]. Hence, influenza A may trigger endogenous eutopic non-osmotic ADH production resulting in hyponatremia [18], as seen in our case. Another possible cause of hyponatremia, in our patient could be secondary to neurosarcoid involvement of the pituitary gland, further contributing to hyponatremia. The hypothesized mechanism is systemic vasculitis-induced stimulation of the HPA causing ADH secretion and SIADH. [19]Bacterial pneumonia is also a known cause of SIADH but the mechanism of this is still unknown. Our case is unique in terms of severe hyponatremia [5, 6]. There are only a few case reports in literature with this low or lower number. Hence, it is important to recognize infectious etiology early on in the course of SIADH before symptoms worsen so prompt and adequate management can be initiated. Our case also highlights the importance of Influenza vaccination in Sarcoidosis patients, since it can help prevent complications such as severe hyponatremia seen in our case. It is also important to note that influenza

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A vaccination in pulmonary sarcoidosis have been studied with no reported adverse effects when compared to healthy controls [20].

Conclusion

Evaluation of severe hyponatremia in a neurosarcoidosis patient should be thorough and other etiologies including viral causes should be highly considered. Viral infections play a vital role inactivating the inflammatory cascade which in turn causes SIADH. Importance of seasonal vaccinations should be emphasized in sarcoidosis patients to avoid such complications.

Ethics statement

The Subject described in this report has given written informed consent for publication of her neurosarcoidosis case.

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