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Case Report

Central sleep apnea in a patient with dengue encephalitis

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ABSTRACT

We report for the first time a case of Central sleep apnea in a patient with dengue encephalopathy. This is a case report of a 50 year old male who had presented with fever, body ache, headache and altered mental status. A diagnosis of dengue fever was made on the basis of IgM antibodies in serum and encephalopathy was attributed to dengue encephalitis in the absence of another etiological cause of encephalopathy. Persistent hyper somnolence and desaturation despite resolution of fever led to a polysomnographic evaluation, which revealed significant central sleep apnea. Hypersomnia, a primary complaint of excessive sleepiness is frequently seen in neurological conditions like neurodegenerative and genetic disorders, stroke, head trauma, encephalitis, and brain tumors. Some encephalitis such as bulbar poliomyelitis, Western equine encephalitis, listeria monocytogenes brainstem encephalitis, and paraneoplastic brainstem encephalitis & Japanese encephalitis have been reported to cause central apnea. This is the first reported case of Central sleep apnea associated with Dengue infection.

1. Introduction

Central apnea is characterized by a lack of central drive to breathe and is identified by cessation of airflow with cessation of respiratory efforts.[1] Dengue encephalitis is itself a rare complication of Dengue fever and incidence of respiratory disturbance in this encephalitis is not well reported as compared to other arboviral encephalitis. To our knowledge there are no reports of central sleep apnea due to Dengue encephalitis as proven by polysomnography. In this case report we present a patient with Dengue encephalitis who developed Central sleep apnea confirmed by a full laboratory based polysomnography.

2. Case report

A 59-year-old obese male (BMI 25.4) was admitted to the hospital with complaints of high-grade fever, and altered mental status. Patient was well about five days prior to admission when he developed fever associated with body aches, low back pain and anorexia. Fever was not associated with cough,

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coryza or any other respiratory symptoms. He also did not have any urinary symptoms. Over the next couple of days continuous high-grade fever was present and subsequently he was noted to be lethargic and drowsy necessitating an emergent admission via the emergency department (ED).

Review of his past medical history revealed the presence of a well-controlled Diabetes Mellitus on Metformin. He had no history of hypertension, coronary artery disease or hypothyroidism. He did not consume alcohol on a regular basis and was a half pack per day smoker. He did not have history of loud snoring, witnessed apneas or excessive daytime somnolence.

On arrival in the ED, he appeared ill and assessment revealed a temperature of 101F, tachycardia, tachypnea and blood pressure of 150/80. There was a diffuse erythematous rash, blanching with pressure prominent on the trunk and mild petechiae over the lower extremity. Auscultation of the chest was normal and oxygen saturation was 95% on room air. He was drowsy, easily arousable with no neck stiffness or any cranial nerve or focal motor deficit. Pupillary response to light was normal and consensual reflex was also normal. Sensory examination was limited due to drowsiness and the plantar response was flexor bilaterally.

His initial laboratory evaluation revealed a normal hematocrit, leucopenia, mild thrombocytopenia, elevated transaminitis and normal renal parameters. Chest radiological examination was normal with no focal consolidation. Computerized tomographic evaluation of the brain also was normal and there was no infarcts or hemorrhage. A rapid diagnostic test for dengue was positive for both IgM and IgG antibodies and patient was admitted to the wards with a working diagnosis of Dengue fever.

He was managed in the ward with closely monitored fluid resuscitation, resulting in a restoration of hemodynamics very rapidly over the first 6–8 h itself. Over the next 72 h his blood parameters also stabilized. He did not have hypotension, altered renal function, liver function and any electrolyte abnormality. However his fever and drowsiness persisted. On day 5 of admission, he became delirious and agitated and was shifted to the intensive care unit (ICU). A lumbar Puncture was performed and cerebrospinal fluid (CSF) analysis revealed normal cell count with mildly elevated protein of 60 mg/dl. EEG evaluation did not reveal any seizure activity. Fever and headache did not resolve over next 7 days and in view of persistent altered mental status a magnetic resonance imaging of the brain was obtained which did not reveal any infarcts, hemorrhage or focal hyper intensities. A repeat CSF analysis revealed a lymphocytic pleocytosis (60 cells) with elevated protein of 60 mg/dl, normal adenosine deaminase levels, with negative polymerase chain reaction to TB and herpes Simplex virus type 1 (HSV). A diagnosis of dengue encephalitis was made on the basis of encephalopathy in a patient with serologically confirmed dengue fever with absence of an alternative explanation i.e. absence of shock/renal/liver failure. Fever finally resolved after 15 days, however patient remained drowsy. Repeat CSF on day 20 revealed decreasing CSF pleocytosis (cell count 3) and decreasing protein concentration in CSF (58 mg/dl) with repeatedly normal ADA and TB & HSV PCR.

Despite resolution of fever, his drowsiness persisted and during sleep he was observed to have episodes of desaturation. In view of presence of desaturation only during sleep a diagnosis of sleep-disordered breathing was entertained. The presence of drowsiness, mild obesity and desaturation prompted a full polysomnographic evaluation.

He underwent a standard overnight polysomnography with multichannel electroencephalographic (EEG), electromyographic (EMG) and electrooculographic (EOG) recording and respiratory monitoring using a nasal thermistor. The sleep study was supervised and performed with the Alice 5 recording system (Respironics, Carlsbad, CA, USA). A single sleep technologist recorded and reported the study according to the criteria of Rechtschaffen and Kales, after which an accredited sleep physician reviewed and reported the study findings, scored the sleep study. His polysomnographic findings revealed a total sleep time of 358 min, Sleep latency of 1 min, fragmented sleep with predominant slow wave sleep and absence of REM sleep. Respiratory monitoring revealed mild snoring and a predominant central apnea pattern with the apnea-hypopnea index (AHI) was 19, the average duration of apnea was 12 s, and the longest duration of apnea was 34 s. Significant oxygen desaturation was noted with a desaturation index of 18/hour, with lowest level of 80%.

3. Discussion

40% of the world's population is at risk for dengue due to its endemicity.[2] Infection can range from asymptomatic infection to severe infection. Encephalopathy and neurologic complications like transverse myelitis, Gullian barre syndrome, acute disseminated encephalomyelitis are well reported. Encephalopathy usually occurs as a result of an indirect insult in the form of liver failure, shock, cerebral edema and deranged electrolytes. Very rarely there is a direct neurotopism of the virus causing encephalitis. Since the other arboviruses like west Nile and Japanese encepahalitis cause direct neural invasion, it is not surprising than dengue also can result in infection of the central nervous system. Thus encephalitis is a distinct clinical entity. The largest evidence for dengue encephalitis comes from Misra et al,[3] they describe 11 encephalopathic patients with confirmed dengue infection, who had extensive evaluation to exclude nonencephalitic cases and 8 of them had CSF pleocytosis suggesting a viral meningoencephalitis process. Similar cases from Solomon et al[4] from Vietnam suggest that in case of dengue encephalitis, all patients had CSF pleocytosis, focal neurologic signs or seizure and dengue confirmed in serum. A case definition of dengue encephalitis as proposed by Varatharaj[5] includes fever, headache, reduced consciousness which is not explained by liver failure, shock, electrolyte derangements and intracranial hemorrhage and which is corroborated with dengue virus or IgM in serum or CSF. In this patient a diagnosis of Dengue encephalitis was based on the case definition proposed by Varatharaj.

Encephalitis per se rarely leads to respiratory disturbance. Some encephalitis such as bulbar poliomyelitis, Western equine encephalitis, listeria monocytogenes brainstem encephalitis, paraneoplastic brainstem encephalitis and Japanese encephalitis have been reported to cause central apnea.[6] There is even a case of transient obstructive apneas with encephalitis.