CASE REPORT

A PATIENT WITH DROWSINESS AND CARDIAC ARRHYTHMIA

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Abstract:
Sick sinus syndrome (SSS) can occur due to an intrinsic defect in the sinus node or extrinsic reversible etiologies. Hyperkalemia, which is a common occurrence in the emergency care or intensive care setting, has rarely been attributed to as reversible cause of SSS. Our case describes the potential reversible association between hyperkalemia and the development of SSS. A 65-year-old elderly patient with recent cerebrovascular accident was admitted to the hospital with sudden-onset drowsiness. He had a poor Glasgow Coma Scale (GCS) score. His electrocardiogram revealed an alternating slow and fast rhythms (bradycardia-tachycardia) with right bundle branch block and left axis deviation which coincided with new-onset of hyperkalemia following sepsis-induced AKI. The patient was successfully treated for hyperkalemia and sepsis which concurrently reversed his characteristic ECG findings. Our case report adds to the literature that several reversible causes are found to be associated with the development of SSS, where manifestation of SSS can be attributed to the presence of hyperkalemia and correction of hyperkalemia can reverse the sinus node dysfunction.

Key words: Sick sinus syndrome; Hyperkalemia; Extrinsic cause;

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Case report:

A 65-year-old-male with a history of cerebrovascular accident (CVA) was admitted to our hospital with diminished appetite and sudden-onset of drowsiness since the previous night. The patient was previously admitted at another hospital for two weeks before presenting to us, where a computed tomography (CT) scan of the brain revealed a left middle cerebral artery (MCA) territory infarction. He was referred to our hospital due to a poor Glasgow Coma Scale (GCS) score (8), poor oxygen saturation (86%) and deteriorating drowsiness. He had been a smoker for the last 30 years. He was not known to have a history of hypertension and diabetes mellitus. The patient's son did not report any previous periodic fainting spells.

On general examination, his blood pressure was 140/40 mmHg, pulse rate was 52/min, and irregular, and oxygen saturation was 98% on O_2 support. He was responding to painful stimuli. Respiratory system examination revealed bronchial breath sounds in both lungs. On cardiac examination, first and second heart sounds were normally audible. On neurological examination, his muscle power in right and left limbs were 4/5 and 1/5, respectively. He was also noted to have a motor speech deficit. He had nystagmus with conjugate eye movement. His Glasgow Coma Scale (GCS) score was found to be 9 (eyes 2; verbal 5; motor 2). Pupil reactions to light were considered normal bilaterally. There was no obvious retinal abnormality identified. Nuclear cataract (NS3) was noted in both eyes.

On admission, patient's electrocardiogram showed sinoatrial nodal exit block with junctional escapes and alternating slow and fast rhythms (bradycardia-tachycardia) with right bundle branch block and left axis deviation (figure 1). Subsequent laboratory results showed pH 7.43, hemoglobin 9.2 gm%, total leucocyte count 19800/mcL, serum K+ 7.5 mEq/L, serum urea 176 mg/dL and serum creatinine 3.6 mg/dl. His liver function tests were normal. The chest radiograph showed military mottling opacity in left lower and right upper lung zones, suggestive of a bilateral bronchopneumonia (figure 2). Subsequently, a high-resolution computed tomography (HRCT) revealed bilateral pleural effusion and patchy consolidation in posterior-basal segment of both lower lobes and posterior segment of right upper lobe. Additionally, widespread and relatively homogeneous pattern of low attenuation involving entire lung was noted, suggestive of a panlobular emphysema. Echocardiography showed concentric left ventricular hypertrophy with ejection fraction of 60% and thickened, calcified aortic valve.

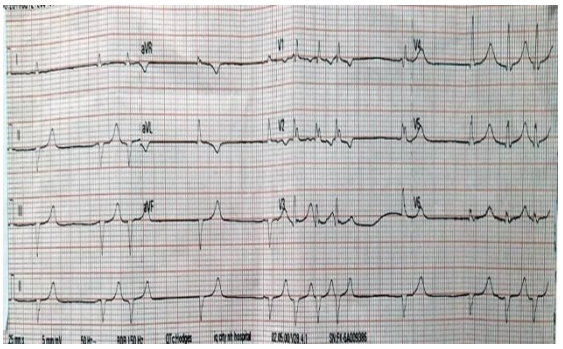

Figure 1: This ECG reveal sinoatrial nodal exit block with junctional escapes and alternating slow and fast rhythms (bradycardia-tachycardia), right bundle branch block, and left axis deviation.

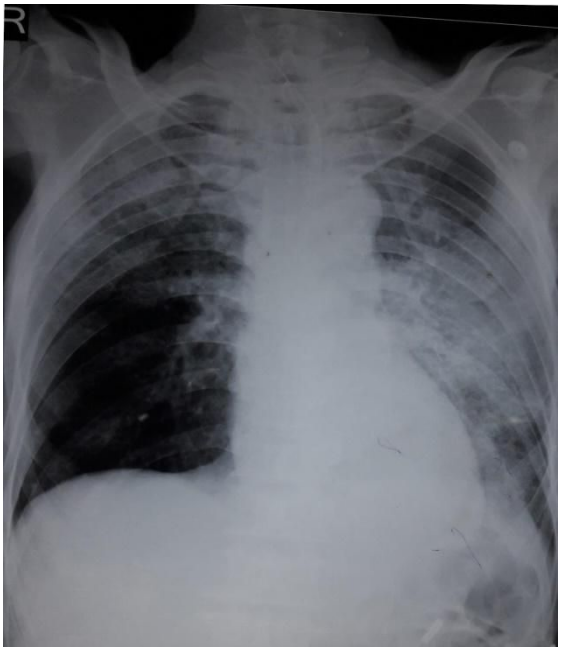

Figure 2: The chest radiograph is showing miliary mottling opacity in left lower 2/3rd and upper 1/3rd in the lungs.

Initially, following the admission, a noncontrast CT (NCCT) scan of the brain found multifocal lesion at the right parietal and the left frontoparietal region with significant edema suggesting a multiple brain metastasis. However, a further evaluation by contrast-enhanced CT (CECT) scan of the brain noted a hypodense, wedge-shaped area with adjacent dilated sulci and dilated frontal horn of the left lateral ventricle, suggesting chronic infarct in left frontal region. Additionally, the CT scan noted a subacute infarct with hemorrhagic transformation in right temporo-parietal region.

The patient's sudden drowsiness in the context of cardiac arrhythmia, acute kidney injury (AKI), electrolyte disturbance, and sepsis was resonated with metabolic encephalopathy. The patient was immediately given IV calcium gluconate. He was started on calcium polystyrene sulfonate and 6 hourly dextrose insulin infusion. Given his ongoing sepsis secondary to pneumonia, he was empirically started on IV antibiotics on admission along with other supportive management. On the same day around 8 PM serum K+ level came down to 6.8 mEq/L, and his repeat electrocardiogram showed a reversal in the characteristic tachycardia-bradycardia pattern back to sinus rhythm (figure 3). Hyperkalemia correction was continued, and subsequent measurements showed improvement in serum K+ levels from day 2 (6.1 mEq/L) to day 5 (4.4 mEq/L) and eventually, repeat electrocardiogram showed well-maintained sinus rhythm. Few days before admission, patient's electrocardiogram showed normal sinus rhythm with no evidence of elevated K+ level on blood reports.

Patients alternating slow and fast rhythms in the electrocardiogram and sudden-onset of drowsiness interpreted as sick sinus syndrome (SSS), coincided with new-onset of hyperkalemia following AKI and the characteristic ECG changes disappeared after the hyperkalemia correction.

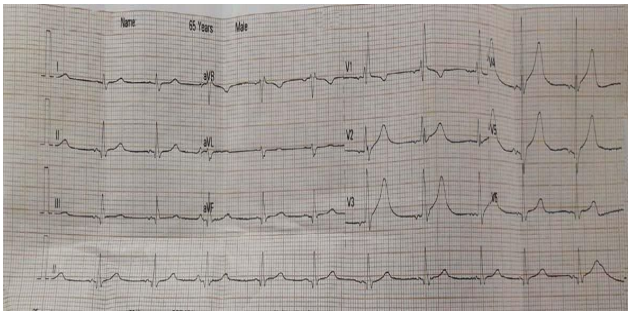

Figure 3: ECG recorded after hyperkalemia management showing resolution of sinus nodal exit block and junctional escapes.

Discussion:

The appearance of hyperkalemia and SSS suggests an association between the two but prior to our report we were unable to find any mention of a causal relationship between the two ever described in the literature. A study found that patients who encountered hyperkalemia-induced severe bradycardia may likely have an underlying SSS. [1] It is possible that the development of his hyperkalemia and SSS along with its disappearance with subsidence of hyperkalemia is coincidental and the SSS if we were able to follow up the patient, may have appeared again in him later without any development of hyperkalemia as a part of the commonest cause for SSS, which is a degenerative disease of the cardiac conduction system. [2] Usually SSS often needs permanent pacing but our patient had it transiently and was treated for his hyperkalemia. Pacemaker placement is indicated only in patients with symptomatic SSS and documented correlation between symptoms and sinus bradycardia or sinus pauses. However, in patients without symptomatic bradycardia do not require permanent pacemaker.[3]

As the patient did not have any previous periodic fainting spells, the likelihood of Stokes–Adams syndrome was considered low. Furthermore, the initial suspicion of multiple brain metastases on NCCT brain with primary malignancy in the lungs was refuted by a subsequent CECT of the brain which revealed a chronic infarction and hemorrhagic transformation. Due to the emphysematous changes in HRCT scan of lungs, high index of suspicion was then put on emphysema in the context of his chronic smoking. However, lung biopsy would have been confirmatory to rule out lung malignancy.

SSS also referred to as "Sinus node dysfunction" is a collection of disorders characterized by the inability of the heart to perform its pacemaking function. SSS has multiple manifestations on electrocardiogram including sinus bradycardia, sinus pause or arrest, and alternating patterns of bradycardia and tachycardia (bradycardia-tachycardia syndrome), which is a common electrocardiographic manifestation of sinus node dysfunction. [2] Sinus node dysfunction can occur intrinsically due to degenerative fibrosis of the sinus node. Some external reversible factors can also mimic or exacerbate SSS including certain drugs (e.g., beta blockers, calcium channel blockers), metabolic disturbances (e.g., electrolyte abnormalities, hypothermia) and autonomic dysfunction. [4]

The patient had developed this syndrome probably secondary to severe hyperkalemia following AKI as was evident by his ongoing hyperkalemia which coincided with the appearance of SSS and its reduction coincided with the disappearance of the syndrome. SSS most commonly results in cerebral hypoperfusion which may have caused sudden onset of drowsiness in this patient. Patients with this syndrome also clinically manifest as syncope, presyncope, palpitations, or dizziness. SSS has an estimated prevalence of 1 in 600 patients over the age of 65 years. Approximately 50% of pacemaker implantations in the United States occurs due to this syndrome. [2] Diagnosis of SSS is made by correlating clinical symptoms of end-organ hypoperfusion with the

electrocardiographic identification of arrhythmia. Permanent pacemaker transplantation is the only effective treatment in case of chronic symptomatic SSS which is not caused by any external reversible factors. [5]

Conclusion:

Sick sinus syndrome in this patient manifested as tachycardia bradycardia syndrome. In elderly patients, manifestation of sick sinus syndrome can be attributed to the presence of hyperkalemia. Correction of hyperkalemia may reverse sinus node dysfunction in these patients.

References:

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