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Recumbent syncope mimicking nocturnal seizures

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RECUMBENT SYNCOPE MIMICKING NOCTURNAL SEIZURES

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CASE REPORT

A fifty seven year old man presented to the emergency at the University hospital with a 3 month history of episodes of arousals from sleep with groaning and deviation of head to his right side. These were followed by a period of "confusion" that lasted for a "couple of minutes". He had a witnessed "convulsion" in the hospital during this last visit and was loaded with one gram of phenytoin. A computed tomography (CT) scan of the head three months ago was reported as normal. Neurology was consulted for further investigations and long-term management.

His wife provided the following information: Over the past three month the patient was brought into the emergency on four occasions with stereotypical nocturnal episodes that occurred in bed within the first thirty minutes of falling asleep. A typical episode lasted 15 - 20 seconds with convulsive movements of the upper extremities and a non-versive head deviation to the right. There was no tongue bite, urinary incontinence or post-ictal paresis or paralysis. Some of these episodes were preceded by an uncomfortable sensation in his epigastrium that woke the patient from sleep. None of these episodes were followed by post-ictal confusion beyond a couple minutes. While interviewing the wife in the emergency room, the patient was witnessed to have a succession of a few clonic movements of his upper extremities lasting 10 - 15 seconds. This occurred while the patient was lying supine with his eyes closed. Although slow to respond initially, he aroused within the next minute and was able to follow 2 step commands. Review of the EKG lead showed a sinus bradycardia of 15 beats per minute lasting 15 seconds that preceded the clonic movements.

He was admitted for continuous cardiac monitoring. A routine electroencephalogram (EEG) was normal. The MRI of brain revealed a large nasopharyngeal mass extending into the right carotid sheath (figure 1) which in retrospect was present on the CT head done 3 months previously. There was no intracranial extension of the mass. Continuous cardiac monitoring for over 24 hours did not demonstrate any primary cardiac arrhythmia or other abnormality.

It was postulated that syncope was precipitated by pressure on the carotid sinus by the nasopharyngeal mass extending into the carotid sheath that increased during a certain neck position as the patient went to bed. It was unclear if this eventually precipitated an epileptic seizure, and therefore due to this uncertainty, it was decided to continue phenytoin until a more definite diagnosis was made. Biopsy of the nasopharyngeal mass showed a non-keratinizing undifferentiated squamous cell carcinoma of the nasopharynx.

Over the next 6 months patient was treated with chemotherapy and radiation treatment.

The patient responded well to the chemotherapy and radiation with an almost complete remission of the nasophayngeal mass. He however declined to come off the Phenytoin for the next three years. He did not have a syncope/seizure after starting Phenytoin and did not want to "take a chance". Three years after the diagnosis, the Phenytoin was tapered off gradually. He has been asymptomatic for the past one year.

DISCUSSION

Carotid sinus syncope has been described in patients with carotid sinus hypersensitivity in relation to neck turning, shaving, tie fitting etc. Probably the first clear clinical description of spontaneous carotid sinus syndrome with documented carotid sinus hypersensitivity was given by Wenkebach in 1914 [16].

Fig. 1: MRI brain shows the right internal carotid artery (arrow) surrounded by the invading mass (arrow heads)

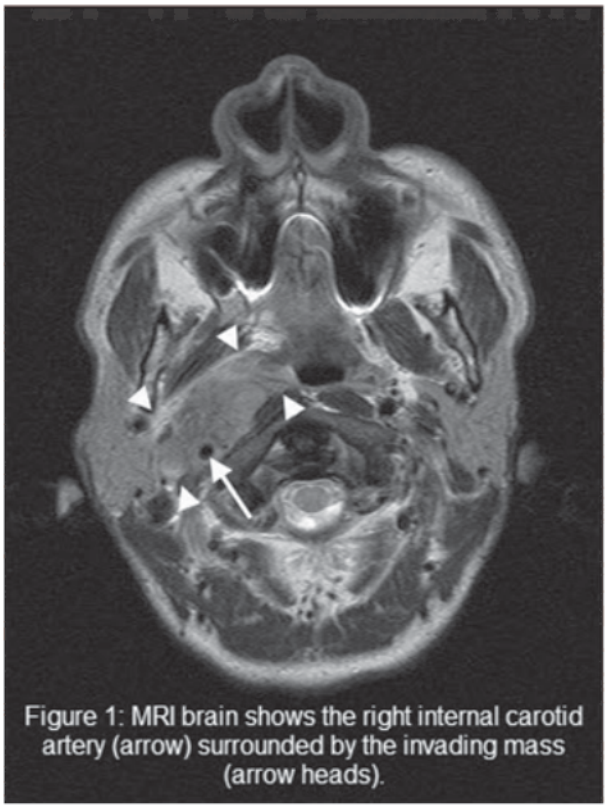

Figure 1: MRI brain shows the right internal carotid artery (arrow) surrounded by the invading mass (arrow heads).

Carotid sinus is an area of dilatation of the internal carotid artery immediately distal to the bifurcation of common carotid artery. In healthy individuals this area has abundant baroreceptors which regulate the blood pressure and pulse. The carotid sinus is innervated by the glossopharyngeal nerve, and the feedback loop has vagal efferents which can lower the heart rate, lower conduction through the A-V node and contribute to peripheral vascular dilatation. Under physiological conditions the baroreceptors in the carotid sinus are sensitive to blood pressure and therefore help regulate the blood pressure. Any external pressure on the carotid sinus, whether transient or in response to a mass lesion has the potential of inadvertently activating the vagal response which may potentially culminate in a syncope [7] Regardless of the etiology of syncope, a sustained hypoxia to the brain can lead to brief convulsive movements of the upper extremities (10) or rarely even a florid seizure (4). A correct diagnosis and identification of the underlying etiology, will minimize unnecessary commotion about the seizure, and help direct focus on the proper management. Seizures have been reported secondary to cerebral hypoperfusion in the context of cardiac arrhythmias and atrio-ventricular block [1,2,3,11]. Seizures have also been reported with vascular events like stroke [4]and aortic dissection [5]. Body movements, tongue biting and urinary incontinence can occur both with seizures due to epilepsy as well as in syncope. Even postictal disorientation can occur following both types of events; although this tends to be more severe with epileptic seizures [6]. Lempert et al conducted a study on healthy subjects in which they successfully induced syncope in 42 individuals. It was observed that myoclonus occurred in 90% and motor activity other than myoclonus occurred in 79% including head turns sometimes accompanied by ipsilateral gaze deviation. However these head turns were not tonic. Electroencephalogram (EEG) was recorded on 6 of these subjects which showed slowing of background during loss of consciousness with three EEGs demonstrating a generalized suppression of the background [8]. Syncope in the setting of a primary or metastatic tumor pressing on the carotid sinus is not a novel observation [7, 9]. Another 19 year old woman with a history of tonic posturing and loss of consciousness with upright posturing who was previously diagnosed with medically refractory epilepsy was finally diagnosed with syncope due to Takayasu's disease [10]. Individuals presenting with recurrent syncope often require extensive investigations to rule out cardiac etiologies. Those who do not present with typical symptoms can be misdiagnosed with medically refractory epilepsy.

Our patient presented with an interesting and unique learning experience for the entire team. What was initially suspected as recurrent seizures, turned-out to be a convulsive syncope triggered by pressure on the carotid sinus. An interesting take-home message of this case report is that syncope can occur in a recumbent position and that all recumbent syncopes are not necessarily related to cardiac mechanical and rhythm pathologies. If conventional investigations do not reveal a cause for the syncope, consider imaging of the structures around the carotid sinus.

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