Brain Tumor is a Rare Cause of both Bradycardia and Seizure

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ABSTRACT

The association between brain tumors and cardiac asystole has rarely been reported in the medical literature. This potentially life-threatening symptom has usually been observed to arise from left temporal lobe brain tumors. Yet previously published papers have shown that cardiac asystole and bradycardia, as manifestations of epilepsy, originate from the frontal lobe of the brain. Although syncope is a common presenting symptom of a brain tumor, bradycardia and complete atrioventricular (AV) block, as the first signs of a brain tumor, have been only sporadically documented in the literature. We report here on a patient with recurrent complete AV blocks that were followed by syncope as an expression of seizures that may have arose from a brain tumor; this tumor was most likely a meningioma in the right frontal lobe. The patient required the subsequent placement of a permanent pacemaker. In conclusion, cardiac asystole may be a potentially life-threatening symptom of frontal lobe lesion of the brain. The frontal lobe may play a role in the autonomic regulation of cardiovascular responses. (Korean Circulation J 2007;37:449-452)

KEY WORDS : Brain tumor ; Heart block ; Seizures.

Introduction

A patient with a brain tumor can present with a diversity of symptoms or signs, including seizures, headache, nausea, vomiting, focal neurological deficit and an altered mental function.1) It is often difficult to differentiate between the cardiac and neurological etiologies of syncope. To complicate matters, primary cardiac abnormalities can cause secondary neurological signs and vice versa. The causes of syncope range from benign to lethal conditions. The distribution of the causes of syncope in the population of the Framingham heart study(7814 participants) was cardiac causes 9.7%, seizure disorders 5.0%, vasovagal causes 21.2%, orthostatic causes 9.3%, medication 6.7%, unknown causes 36.3% and other causes 11.8%.2) Bradycardia and cardiac asystole sometimes lead to life-threatening syncope for which implanting a demand pacemaker may be useful.3-6) We report here on a patient with recurrent and transient complete AV blocks that were followed by syncope as an expression of seizures, and these seizures may have arose from a brain tumor in the right frontal lobe.

Case

A 78-year-old right-handed man, who was without any relevant medical history and he was not taking any medication except aspirin, was admitted to the emergency room of our hospital with loss of consciousness. He’d had brief paroxysmal episodes that were characterized by behavioral arrest with loss of consciousness during eating breakfast.

On arrival at the hospital, the patient had an alert mentality, a blood pressure of 170/100 mmHg and a regular pulse rate of 71 bpm. The laboratory examinations did not reveal any abnormalities. Further neurological examination revealed no abnormality of the cranial nerves. The initial electrocardiography (ECG) showed a normal sinus rhythm and no prolonged QT interval (0.414 second). A chest X-ray demonstrated no abnormality. Blood analysis showed no abnormalities, including the electrolytes and the CK-MB and Troponin-I levels. The thyroid function tests were also within normal limits. Contrast computed tomography (CT) of the brain showed a 1.4×2.0 cm sized brain mass in the right...
frontal lobe. Magnetic resonance imaging (MRI) of the brain revealed a 1.4 × 2.0 cm sized, slightly high signal-intensity mass that was most likely a meningioma in the right frontal lobe (Fig. 1). The patient was admitted to the Department of Neurosurgery for further evaluation.

The EEG recordings revealed no definite epileptic discharges. On the 7th hospital day, he had a seizure and then lost consciousness again. During the syncope event, the ECG showed a progressive decrease of the heart rate to 28 bpm and complete AV block (Fig. 2A), and the blood pressure decreased to 60/30 mmHg. Cardiac massage was initiated and some minutes later patient recovered an alert mentality with a normal sinus rhythm of 78 bpm (Fig. 2B) and a blood pressure of 90/60 mmHg.

Some 6 hours later, he had another seizure and then he experienced complete AV block (Fig. 2C) with a blood pressure of 60/50 mmHg, but he did not lose consciousness. Some minutes later, the patient completely recovered spontaneously with a normal sinus rhythm and blood pressure. The patient was transferred to the Department of Cardiology for the further evaluation. Transthoracic echocardiography revealed good regional wall motion with

![Fig. 1](Image)

**Fig. 1.** Brain MRI revealed a 1.4 × 2.0 cm sized slight high signal mass (white arrow) in the right frontal lobe on T2-weighted axial image. MRI: magnetic resonance imaging.

![Fig. 2](Image)

**Fig. 2.** The ECG showed a complete AV block during the first syncope event in the hospital (A), recovered the normal sinus rhythm after the first syncope (B), redeveloped a complete AV block (C) and setted ventricular rate over 60 bpm by permanent pacemaker (D). ECG: electrocardiography, AV: atrioventricular, bpm: beats per minute.
a left ventricular ejection fraction of 67%. A Holter test revealed frequent APCs and rare VPCs, but it didn’t reveal complete AV block. The coronary angiogram showed no definite abnormality. We still noted intermittent seizures with complete AV block for 5-10 seconds until the 12th hospital day. Thus, we decided to implant a permanent pacemaker. During the following days after permanent pacemaker implantation (the VVI type), we set the ventricular rate over 60 bpm, and this resulted in no bradycardia and no complete AV block or seizure (Fig 2D). The patient did not experience any seizure and syncope attacks during the 20 days after implantation of the permanent pacemaker.

Discussion

Many previously published papers have shown that cardiac asystole and bradycardia, as manifestations of syncope or seizure, originate from the temporal lobe. 4-13 Several studies have suggested a frontal lobe influence on the autonomic cardiovascular regulation. 12-13

We report here on a patient with a complete AV block that was followed by syncope as the first sign of a right frontal brain tumor. Although we didn’t perform simultaneous ECG/EEG recordings during a period of bradycardia, we believe that the origin of the bradycardia was a brain tumor in the frontal lobe. Electrical stimulation of the cingulated gyrus and orbitofrontal cortex has produced changes in the heart rate, and cases of ictal bradycardia related to orbitofrontal lobe seizures have been reported on. 14-16 The insular cortex, the central nucleus of the amygdala and some structures of the hypothalamic (paraventricular nucleus, the lateral hypothalamic area and dorsomedial nucleus) belong to the central autonomic network, which controls the pre-ganglionic sympathetic and parasympathetic visceromotor outputs. The mesial, temporal and frontal areas are interconnected to the central autonomic network so that ictal discharges arising from or spreading to these regions are more likely to induce autonomic changes. 17

Many different regions of the nervous system are involved in the cardiovascular control (brain stem, thalamus, hypothalamus, amygdale and insular cortex). Pathology in these regions can give rise to various types of cardiac dysfunction. For instance, cortical stimulation studies in humans have shown depressor responses and bradycardia upon stimulation of the left insular cortex, whereas the converse applied for the right insular cortex. 18 In humans undergoing temporal lobectomy with amygdalo-hippocampectomy for epilepsy, bradycardia and hypotension have occurred during manipulation of the amygdale and hippocampus. 19

Critchley et al. 20 conducted functional MRI experiments, and they reported changes in the heart rate for both cognitive and motor performance that was related to the strength of activation in the anterior cingulated cortex. In particular, the activity observed in the anterior cingulated cortex was related to sympathetic modulation of the heart rate, and this was dissociable from cognitive and motor-related activity.

In conclusion, our case confirms that cardiac asystole may be a potentially life-threatening symptom during the partial seizures that were caused by right frontal brain tumor. We can also hypothesize that seizures originating in the fronto-mesial structures may disrupt a complex neural system involving the fronto-temporal-insular regions, and these regions have been implicated in the autonomic regulation of the cardiovascular responses.

REFERENCES

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