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RESEARCH ARTICLE

A PROPOSED ETIOLOGY FOR ISOLATED SMALL RIGHT TEMPORAL INFARCT AND CIRCUMFLEX CORONARY ARTERY STENOSIS OR WHAT WAS FIRST: THE EGG OR THE CHICKEN?

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ABSTRACT

The neuroimaging evidence regarding the physiological role of temporal lobe in regulating the peripheral autonomic response are rapidly replicating. However, the role of temporal cortical damage in the generation of cardiac arrhythmias are unclear. Here, we describe a unique case of isolated, small, right temporal infarct presented with significant transient autonomic symptoms. Our case helps to clarify an ongoing debate on the relationship between lesion temporal cortical location and the occurrence of cardiac arrhythmias in stroke patients and also suggest that nonspecific cardiological symptoms should be evaluated with detailed cardiological workout in stroke patients who are presented with normal ECG. To the best of our knowledge, isolated temporal infarct have not been previously reported to be associated with the occurrence of cardiac arrhythmias in stroke patients.

INTRODUCTION

The central regulation of the autonomic response involves a network of cortical and subcortical systems which are functionally connected with major autonomic regions and implicated in the generation of the peripheral cardiovascular responses.[1]This is suggested with following studies revealing that the stimulation of the insula and temporal lobe in connected with other key areas promote significant autonomic responses including significant changes in the blood pressure and heart rate.[1,2,3] Suggesting the role of central nervous system in the regulation of the cardiovascular system, several neuro imaging activation studies have demonstrated the key role of insular cortex in central autonomic processing. [1,4,5,6,7] Moreover, recent studies have already revealed that the insula is functionally linked to the anterior cingulate cortex and temporal cortices [4,8,9] as a part of a great functional network. These findings suggest that the temporal region is also involved in central autonomic processing. In respect of this, a recent fMR study has shown that middle temporal cortices in combined with other key areas play a significant role in the regulation of autonomic responses after specific normal and physiological conditions[7]. It has been already shown that cardiac arrhythmias are very common in a large portion of acute stroke patients.[4,10,11,12] The underlying responsible mechanism has been proposed that impaired central autonomic control could result with significant cardiac arhytmias. Despite previous studies showing that insular lesions led to an increased risk of cardiac autonomic imbalance, further studies have suggested that specific central lesions brain regions other than the insular cortex could also be involved in autonomic impairment. In agreement with this, recent studies have shown that parietal lobe stroke plays an independent role in predicting the long-term cardiac mortality while frontal cortex lesions are associated with the occurrence of serious cardiac arrhythmias. Acute left circumflex coronary artery (LCx) occlusion plays a major role in the pathogenesis of cardiogenic derived arrhythmia that is attributed to its major role in supplying the key rhtymogenic cardiac areas. However, it is very difficult to detect an acute left circumflex coronary artery stenosis by the standard 12-lead electrocardiogram (ECG). To the best of our knowledge, there is no stroke case in the literature suggesting that there is a link between right-sided temporal stroke and the occurrence of cardiac arrhythmia. In the present case, we report an unusual case of isolated isolated right temporal infarct with left coronary artery stenosis that is presented with tachycardia, hypertension, and atypical nonvascular headache attacks.

CASE PRESENTATION

A 48-year-old diabetic man presented with sudden tachycardia, hypertension, headache and vomiting attacks lasting approximately 10-15 minutes. There was no history of palpitations, headache or any cardiac or cerebrovascular disease. He has not hypertension and there was no other comorbid illness in his history. The headache lasted about 10 minutes located on the frontal and parietal region with no associated migrainous characters while the arterial tension was increased about 30-40 mm/Hg (150/100 mm/Hg on the admission) in the last hours. The clinical suspicion of acute myocard infarction was excluded after an acute cardiac evaluation showing normal troponin levels and standard 12-lead electrocardiogram (ECG) with sinus tachycardia (120 bpm) and left anterior hemiblock. Further cardiac evaluation through coronary digital substraction angiography revealed 90-100 % narrowing of the left circumflex optus marginal coronary artery that is supplying the sinus-node in the majority of left predominant humans (Figure 2).

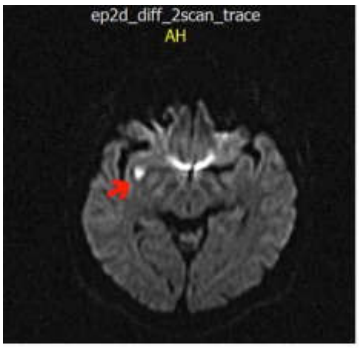

Figure 1a

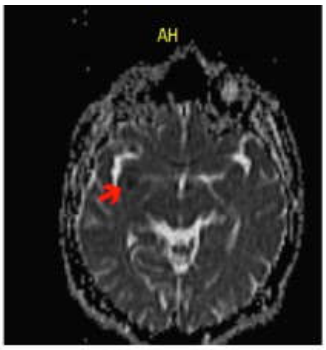

Figure 1b

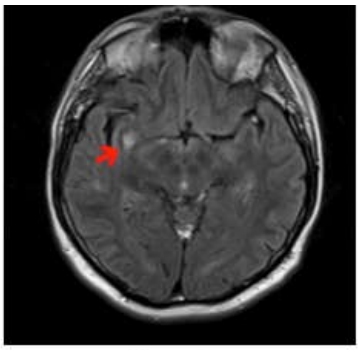

Figure 1c

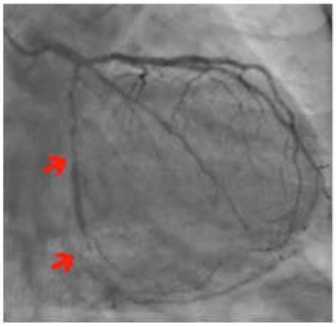

Figure 2

Figure1a,b,c Diffusion weighted MRI showed an isolated acute is chemicinfarct on the right temporal region(Figure1a). The corresponding ADC maps revealed prominent hypointensity (Figure1b) and FLAIR images show increased signal intensity on the right temporal region (Figure1c). Please see the arrows

Figure 2 Coronary digital substraction angiography revealed 90-100 % narrowing of the left circumlexoptus marginal coronary artery. Please see the arrows

His neurological examination showed that he was fully cooperative and oriented while he displayed no lateralized neurological abnormality. Detailed blood tests revealed moderately elevated HbA1c levels (7.5 %) while his lipid profile was mildly elevated. After excluding an acute cerebral hemorrhage with acute cranial CT imaging, we have immediately performed a diffusion MRI to exclude an acute ischemic infarct that showed an isolated acute ischemic infarct on the right temporal region (Figure 1 a,b,c). Cervical and cranial CT angiography, as well as the MRI-venography, have been found in normal limits. Doppler ultrasonography examination revealed mildly distributed fatty and atherosclerotic plaques on the right and left common carotid and bilateral arteriacarotisinterna without stenosis. After confirming the diagnosis of ischemic cerebral infarct, we have been focused on the cardiac investigation with long-term Holter monitoring for 72 hours and transthoracic and transeusophagialechocardiography which have been found in normal limits. Additional assessment through young stroke panel including the evaluation of the levels of Protein C, Protein S, Antithrombin III and homocysteine as well as the mutation of Factor V Leiden, MTHFR revealed no abnormality. Electroencephalopgraphic assessment in order to exclude a possible temporal lobe derived epilepsy was found to be in normal limits. We agreed with the cardiology department to initiate dual antiaggregant (acetylsalicylic acid 100 mg/day + Clopidogrel 75 mg/day) and statin (atorvastatin 40 mg/day) therapy to provide strategically effective secondary prevention both for the coronary and cerebral vasculature.

We have observed that the autonomic symptoms improved significantly after initiating the dual antiaggregant and statin therapy on day three, and his actual neurological examination was not different from the initial neurological examination on the day of discharge.

DISCUSSION

Although it has been already revealed that temporal lobe is majorly involved in the central control of autonomic functions,[6,7] it has not yet been reported its role in the context of brain-lesion-induced cardiac arrhythmias. After carefully questioning the headache history we have found no remarkable

findings suggesting a migraine attack that could be responsible for a possible migrainous infarct. We additionally have revealed significant stenosis of the left posterior circumflex coronary artery (Figure 2) that is responsible for vascular supply of the sinus node. In this context, it can be hypothesized that in some stroke patients cardiac arrhythmias might have been not the result of neurocardiological interaction but of coincident cardiac comorbidity that also could involve the ischemia of the rhytmogenic centers. Similarly, in our case, it is difficult to estimate what caused to arrhythmia symptoms, however, based on previous literature showing the role of cardiac ischemia in the generation of tacharrhythmias, it can be assumed that both cardiological and neurological factors could be connected in a feedback-forward manner to result with the cardiac autonomic impairment. Recent studies have already shown that sinus tachycardia and left hemiblockplay not as a risk factor for ischemic stroke. In the light of rapidly increasing evidence showing that longer ECG monitoring may significantly improve the detection of a possible paroxysmal atrial fibrillation, we were not able to exclude a possible cardioembolic scenario although we have revealed that the long term (72 h) ECG monitoring and echocardiographic assesments are within normal limits. Moreover recent studies have already suggested that even silent tachyarrhythmias can lead to the impairment of haemodynamic stability that may aggrevate stroke via reducing the cerebral perfusion which may in turn increase the risk of cardiac ischemia in patients with comorbid cardiovascular diseases. Taken together, beyond indicating that right-temporal hemispheric lesions may be associated with an increased risk for autonomic imbalance,[4] we suggest that a multifaceted approach including a detailed cardiac diagnostic workup is required in acute stoke patients with cardiac arrhythmias. Considering the fact that significant number of stroke patients with cardiac comorbidities may present with a normal ECG, we also provide a valuable data indicating to the important role of circumflex artery stenosis in autonomic disturbances in acute stroke patients. Although these patients could be centrally susceptible to tachyarrhythmic episodes, future studies with larger sample size are needed to understand the neurocardiological interaction between strategically located lesions in the right temporal cortex and silent sinus node dysfunction.

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