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Cardio-embolic cerebellar stroke secondary to mitral valve chordae rupture as a delayed complication of a high-voltage electrical injury

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Abstract

A 41-year-old farmer sustained a high-voltage electrical injury resulting in confusion, electrical burns and paroxysmal atrial fibrillation which spontaneously reverted to sinus rhythm after a few hours. Two weeks later he presented with a sudden onset of headache, unsteadiness, horizontal oscillopsia and paraesthesia in the right side of his face. Examination revealed nystagmus to the right, right-sided limb ataxia and a tendency to veer toward the right when walking. An MRI of the brain demonstrated an acute infarct of the right cerebellar hemisphere in the territory of the right posterior inferior cerebellar artery. A transesophageal echocardiogram showed a ruptured mitral valve chordae. This is the first report of a cardio-embolic stroke secondary to mitral valve chordae rupture as a delayed complication of high-voltage electrical injury. Although many mechanisms of direct cerebral electrical injury have been speculated, a cardio-embolic origin should not be overlooked as a cause of stroke secondary to high-voltage electrical injury.

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Keywords: Electrical injury; Stroke; Cardiac valve rupture

1. Introduction

Neurological complications secondary to high-voltage electrical injury (HVEI) include spinal, peripheral nerve and cerebral injuries following lightning or accidental electrical injury. Damage is not restricted to cases where the nervous tissue lies in the electric current pathway. The neurological complications caused by electrical injury can be immediate or delayed, occurring days to months later.¹

Additionally, HVEI may cause skin burns, musculoskeletal injury or myocardial damage. Cardiac valve rupture

due to electrical injury is a rare phenomenon,² though arrhythmias, non-specific ECG changes and rarely, myocardial infarction are described at the time of injury.³ This case report combines two rare manifestations and is, to our knowledge, the first report of a cardio-embolic stroke secondary to mitral valve chordae rupture as a delayed complication of HVEI.

2. Case report

A 41-year-old right-handed farmer sustained an HVEI from a 19 000 volt power line when the tray of his dump truck came into contact with the power line. In an attempt to prevent this he grasped the electrified door handle with his right hand and was thrown clear of the truck. He was

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found shortly after wandering about aimlessly. Within a few hours his cognitive state returned to normal. The electrical injury resulted in atrial fibrillation (AF) which was confirmed on ECG and spontaneously reverted to sinus rhythm within a few hours. Electrical burns to his right hand (entry wound) and his left foot (exit wound) also occurred, requiring the amputation of three toes due to necrosis. There was no history of cardiac disorders prior to the electrical injury and no cardiovascular risk factors except for mild untreated hypertension. The patient took no medications.

Two weeks later he presented with a sudden onset of severe headache, unsteadiness, horizontal oscillopsia and paraesthesia in the right side of his face.

Examination revealed nystagmus to the right and right-sided limb ataxia. His gait was broad-based tending to veer towards the right. Cranial nerve examination, tone, power and sensation were normal. The pulse was regular and heart sounds were normal. There were no carotid or vertebral artery bruits and no cardiac failure.

The ECG remained in sinus rhythm. Laboratory investigations included normal full blood count, coagulation studies, electrolytes, troponin T, blood glucose, C-reactive protein and homocysteine. Lupus anticoagulant, anticardiolipin antibodies, anti-neutrophilic cytoplasmic antibodies, antinuclear antibody, extractable nuclear antigen antibodies and rheumatoid factor were negative.

MRI of the brain demonstrated an acute infarct of the right cerebellar hemisphere in the territory of the right posterior inferior cerebellar artery (PICA) (Figs. 1 and 2). On magnetic resonance angiography, both vertebral arteries displayed normal flow with no evidence of dissection. The left PICA was just visible but the right PICA was not well seen. A trans-oesophageal echocardiogram showed a ruptured mitral valve chordae and trivial mitral regurgitation, but no evidence of thrombi in the left atrium

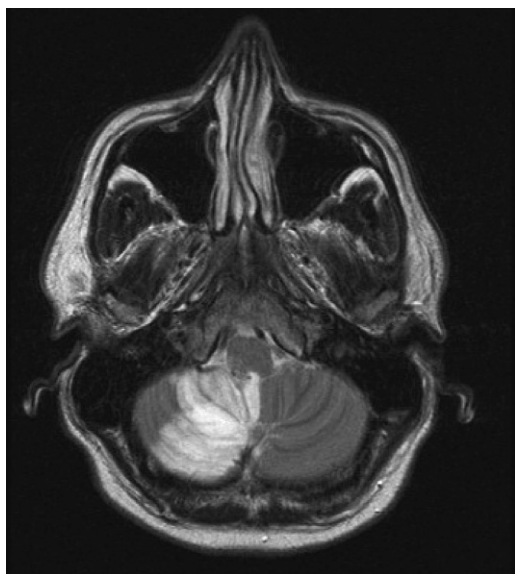


Fig. 1. Axial T₂-weighted MRI showing an increased T₂ signal involving the right cerebellar hemisphere consistent with a cerebellar stroke.

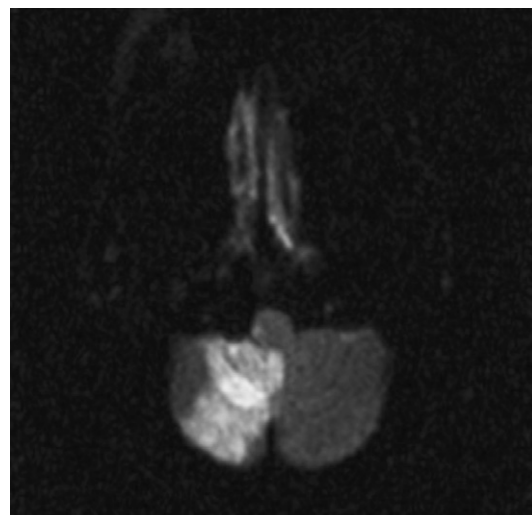


Fig. 2. Axial diffusion-weighted MRI showing acute ischaemia in the right cerebellar hemisphere.

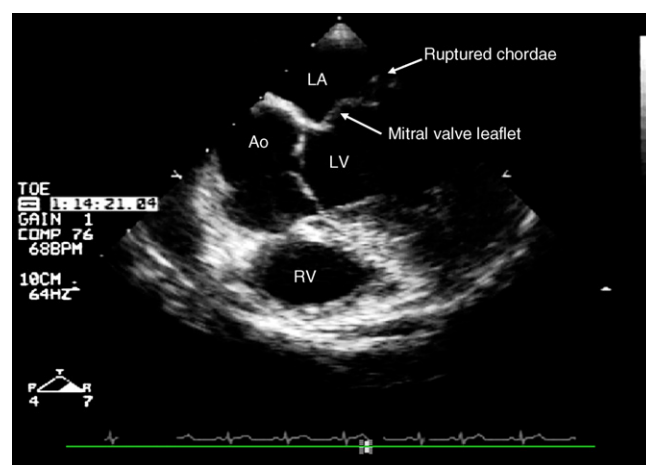


Fig. 3. Trans-oesophageal echocardiogram showing the mitral chordae rupture. LA, left atrium; LV, left ventricle; RV, right ventricle; Ao, aorta.

or left ventricle (Fig. 3). At a 3-month review the patient had made a near full neurological recovery.

3. Discussion

The patient presented with an ischemic cerebellar stroke in the territory of the right PICA presumably secondary to a mitral valve chordae rupture 2 weeks after an HVEI. We are unaware of any reports of embolic stroke as a delayed complication of HVEI. Hemiplegia and aphasia have been reported as complications after HVEI but no pathological or neuroimaging data were available at the time of description.¹ Demonstrated ischemic stroke has been described in a single case report as an immediate complication of lightning and its mechanism is speculative.⁴

In this patient, the current traversed the body from his right hand to his left foot. This route of current is one of the most frequent mechanisms of cardiac damage because the heart lies on the pathway of the current and suffers its

direct effect. The mitral valve chordae lesion was possibly caused by conversion of electrical energy into heat or the establishment of an electrical arc through the myocardium and the mitral valve. A similar mechanism was proposed by Güler et al. who reported acute aortic regurgitation due to a lacerated and necrotic valve 2 weeks after an HVEI in a patient without history of cardiovascular disease.² Interestingly, the clinical manifestations of the aortic valve rupture appeared 2 weeks after the electrical injury, the same delay as in our patient.

The cerebellar stroke in the present patient probably occurred secondary to embolism from a thrombus formed on the damaged endothelium at the site of rupture. AF was another possible mechanism; however sinus rhythm was re-established 2 h after the electrical injury, and a full 2 weeks before the stroke. A brief episode of AF in an otherwise normal heart would be unlikely to cause delayed cardiac emboli. Endothelial damage to the aortic arch or right vertebral artery is another possibility, although one would expect an ischaemic event to occur earlier.

Many mechanisms of direct cerebral electrical injury have been speculated and include complications caused

by the heating effects of the current, electrolytic effects, direct mechanical effects, electrostatic effects and vascular changes.⁵ All of these processes can result in delayed neurological sequelae. However, other factors need to be considered, as illustrated by the present case. As there was a proven rupture of the mitral valve chordae, possibly due to delayed necrosis, it is much more likely that the aetiology of the stroke was embolic, rather than being due to a delayed local neurological process.

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Interdural cavernous sinus epidermoid cyst

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Abstract

We report a patient with an uncommon interdural epidermoid tumor, located within the confines of dural layers of the lateral wall of the cavernous sinus. The tumor was resected by a basal subtemporal extradural-interdural approach. Following the surgery, the 45-year-old female patient recovered completely from her symptoms of atypical neuralgic facial pains.

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Keywords: Interdural; Epidermoid; Cavernous sinus

1. Introduction

Intracranial epidermoid tumors are relatively rare and are among the most benign intracranial tumors. These tumors have been located in several sites of the brain and spinal cord.^{1,2} We located reports of 17 cases of epidermoid

tumors in relationship to the cavernous sinus.³ Although the exact site of location of these tumors in relationship to the dural walls is not clear from all these papers, it appears that at least four tumors were ‘interdural’ as they were located within the confines of the dural layers of the lateral wall of the cavernous sinus, normally harboring only the trigeminal nerve.^{2–4} We present our experience with a patient with a ‘interdural’ epidermoid tumor and discuss our surgical strategy.

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