

Sudden death in a case of lateral medullary syndrome

Sir

We report a case of sudden death in a case of lateral medullary syndrome (LMS). Sudden unexpected death is an unusual event in LMS.^[1-2] A 39-year-old male, a chronic smoker, presented with vertigo, dysphagia, hoarseness of voice, and imbalance while walking of 6 hours duration. On examination, his blood pressure was 140/90 at admission and the neurological examination revealed sensory loss on the right side of the face and left half of the body with right-sided cerebellar signs, conforming to right LMS. A non contrast computed tomography (CT) scan showed an infarct in the right inferior cerebellum and a CT angiography showed non visualisation of the right vertebral with a thrombus extending into the proximal basilar artery [Figure 1]. The proximal right vertebral artery was well visualised in the CT angiography. Considering the risk of progression to complete basilar artery occlusion, the patient was taken in for an intra-arterial (IA) thrombolysis after full informed consent was obtained. A selective right vertebral artery catheterisation was done. A 5 mg bolus of r-tPA was injected over a period of 1 min. This was followed by a slow infusion of 20 mg r-tPA. However, after an infusion of 4 mg of r-tPA after the bolus, the patient had a sudden cardiorespiratory arrest. He succumbed despite resuscitative measures.



Figure 1: CT angiography [Volume rendered image, (inset-Maximum intensity projection image)] showing an occluded right vertebral artery with the thrombus extending into the proximal basilar artery (arrow)

A non contrast cranial CT scan was repeated, which did not show any hemorrhagic transformation.

Recent reports have described unexpected sudden cardiorespiratory arrest in lateral medullary infarction during convalescence after a stroke with minimal motor disability.^[3-5] Various mechanisms have been postulated for the sudden cardiorespiratory arrest in LMS including cardiac arrhythmia^[3] and ischemic penumbra affecting the cardiac and respiratory centers of the medulla.^[4] A recent neuropathological study of five patients disclosed ischemic lesions in the solitary tract nuclei of the medulla after subacute hypoperfusion of the brain during acute heart failure.^[6] It was speculated that these medullary lesions caused autonomic instability, which precipitated death. In our case, a sudden unexpected cardiorespiratory arrest occurred during IA thrombolysis in an otherwise haemodynamically stable patient. The mechanism of the arrest could not be ascertained since monitoring could not be done during the procedure. Sudden death in the presence in LMS is often puzzling and mandates close monitoring of the cardiac and respiratory functions in patients with LMS.

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