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Letter to the Editor

Onsite thrombus formation may not be the only explanation of middle cerebral artery occlusion in traumatic brain injury

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We read with interest the article by Hayakawa *et al.* on an 80 years-old female who unexpectedly died after traumatic brain injury (TBI) through a car accident despite recovery from the trauma^[1]. Autopsy revealed that the index patient had suffered an ischemic stroke of the right middle cerebral artery (MCA) due to occlusion of the M1 segment by a thrombus^[1]. It was speculated that thrombus formation occurred at the site of the occlusion due to an intimal tear upstream of the thrombus as a consequence of the traffic accident^[1]. The study is appealing but raises concerns that warrant further discussion.

We disagree with the explanation for the thrombus formation^[1]. Several mechanisms of thrombus generation were not considered. The first stroke mechanism not considered in the index patient was Takotsubo syndrome (TTS)^[1]. TTS is an acute cardiomyopathy due to psychological or mechanical stress or of unknown etiology in one third of the cases each^[2]. Four subtypes of TTS are delineated, the classical, mid-ventricular, the basal, and the global subtype. TTS mimics myocardial infarction clinically, electrocardiographically, blood chemically, and echocardiographically, but coronary angiography is usually normal^[2]. In the index case TTS could have been triggered by the mechanical trauma, by the pain that was presumably caused by the trauma, or by acute stress reaction that most likely developed after such an event. We therefore should be informed about the ECGs recorded during hospitalisation, and the echocardiographies carried out during hospitalisation.

A second pathophysiological mechanism not considered is left ventricular hypertrabeculation (LVHT). LVHT is a rare congenital or acquired condition, characterised by an increased number of trabeculations inside the left ventricular myocardium. Trabeculations are most commonly seen in the apex and the lateral wall^[3]. We should know if there were any indications for hypertrabeculation in the left ventricular myocardium on autopsy.

A third mechanism not sufficiently considered is cardioembolism due to supra-ventricular- or ventricular arrhythmias. Although the history was negative for atrial fibrillation (AF), it is conceivable that the index patient had developed AF during hospitalisation triggered by the stress from the TBI^[4]. Because the prevalence of AF increases with age, advanced age of the index patient could be another causative factor. There are a number of case reports showing that TBI can trigger AF^[5]. There are also indications that intracerebral bleeding can trigger AF^[6]. There are even cases in which treatment for TBI triggered the development of AF.

We disagree with the notion that absence of a thrombus intra-ventricularly suggests formation of the thrombus occluding the right MCA at the site of the occlusion^[1]. An intraventricular thrombus could have embolised entirely without leaving behind a rest thrombus inside the ventricular cavity. It is also conceivable that only a single thrombus was generated intra-ventricularly that embolised entirely.

Vasospasm of the M1 segment after SAB is rather unlikely causative given the fact that vasospasm of the proximal MCA after SAB has not been reported before. Another argument against SAB related vasospasms as the cause of malignant stroke is that no vasospasms were reported in any other territory of any vascular bed.

A limitation of the study is that monitoring of the heart rhythm during hospitalisation was not reported.

Overall, the study carries obvious limitations that require re-evaluation and discussion. Clarifying these weaknesses would strengthen the conclusions and could improve the study.

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