SHORT REPORT



Management of malignant middle cerebral artery infarction following a cardiac stab wound – the role of early decompressive craniectomy

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Abstract

We report the presentation, investigation and management of a 22-year-old male who developed a right malignant middle cerebral artery infarct following a cardiac stab wound. This case exemplifies that early identification and timely decompression of young patients with embolic infarcts as a result of penetrating trauma can lead to a favourable clinical outcome.

Keywords: decompressive craniectomy; delayed ischaemic neurological deficit; raised intracranial pressure; surgical decompression; stroke; trauma

Clinical details

A 22-year-old Caucasian male was admitted to our level 1 trauma centre after receiving a single stab wound to his precordium. He was attended to by London's Air Ambulance service who intubated on scene, and then transferred him to our major trauma centre. He was shocked on arrival (blood pressure, 83/60 mmHg; heart rate, 145/min) with a focussed assessment with sonography for trauma (FAST) scan revealing a left haemothorax and pericardial haematoma. The patient was transferred to the operating theatre and underwent an emergency sternotomy for a presumed penetrating injury to the heart leading to tamponade. On release of the tamponade, a jet of mixed arterial and venous blood was noted ejecting from a right ventricular (RV) laceration, suggesting a possible traumatic ventricular septal defect (VSD). A vertical 4-cm laceration to the RV as well as a small laceration to posterior wall of the left ventricle (LV) were closed with Teflon-pledgeted sutures. Prior to repairing the posterior LV injury, cardiopulmonary bypass (CPB) was commenced for haemodynamic instability associated with continuing blood loss from the back of the heart. The repairs proved sound, and the patient disconnected from CPB after an intraoperative transoesophageal echocardiogram (TOE) showed no evidence of a large traumatic VSD or other intracardiac shunting.

The patient was transferred to Intensive Care with good haemodynamics, sedated and intubated. On the second day, sedation was stopped and the patient was noted to have a dense left hemiparesis. Post-operative computed tomography (CT) scans (this had not been done on admission as the patient required immediate surgical intervention) revealed an extensive area of low attenuation involving the parietal lobe with appearances suggestive of an acute infarct. A concurrent CT scan of the thorax demonstrated a 0.5-cm VSD (Fig. 1) with a repeat TOE confirming the defect's presence with no evidence of thrombus. There was no evidence of aortic or proximal carotid injuries or dissection in the area of aortic cannulation. The VSD required no intervention as it was small and near the apex, and therefore muscular in nature with good chances of spontaneous healing.

On Day 3, the patient's GCS deteriorated from 15 to 13 (E2 V5 M6). A repeat CT head scan demonstrated an increase in the degree of low attenuation within the large right MCA territory infarct as well as widespread sulcal effacement, new midline shift with subfalcine and uncal herniation to the left side (Fig. 2). The patient continued to worsen neurologically so was taken to theatre for an emergency right-sided decompressive craniectomy (DC). No perioperative complications were observed. A titanium cranioplasty was inserted 3 weeks post-discharge.

On review at three months, the patient's hemiparesis had continued to improve and no significant cognitive deficits were identified; he was mobilizing independently and had returned to part-time work. His VSD continues to be managed conservatively and is being monitored with serial echocardiograms. If the defect becomes symptomatic, he will be a candidate for percutaneous closure.

Discussion

There is significant mortality and morbidity associated with malignant middle cerebral artery infarction. Given that medical management has shown to have limited

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Fig. 1. Day 1 post-thoracotomy CT thorax demonstrating traumatic VSD (*arrow*).

success, there has been considerable interest in the use of decompressive craniectomy (DC) as an acute intervention to reduce the adverse effects of raised intracranial pressure. Although a growing volume of evidence demonstrates an overall reduction in mortality, a recent Cochrane review concluded that DC improves survival at the expense of an increased proportion of survivors with moderately severe or severe disability.¹ The current National clinical guideline for stroke² describes a subset of stroke patients who may benefit the most from emergency decompression. The guidelines suggest that timely (within 48 h of onset of symptoms) decompression should be considered in the younger population (aged under 60 years) who demonstrate both clinical and radiological evidence of a severe middle cerebral artery (MCA) insult. The patient in this report fits this subgroup and may help to explain why he had a reasonably favourable functional outcome.

We must acknowledge that the aetiology of our patient's malignant MCA infarct is not clear. A global hypoxic insult is unlikely as although the patient was haemodynamically unstable on admission, there was no documented loss of cardiac output. The authors propose that although no thrombus was identified on the perioperative transesophageal echocardiogram, a cardiac thrombus related to direct left ventricular trauma, the VSD or perioperative debris is the



Fig. 2. Day 3 post-thoracotomy CT head demonstrating evolving malignant MCA infarct.

most plausible explanation for his neurological sequelae.^{3,4} Although no obvious lung injury was noted perioperatively, given the mechanism of injury a cerebral air embolus could be considered a potential cause of the ischaemic insult.

This case is the first report in the literature of surgical hemispheric decompression for cerebral infarction following a penetrating chest injury. It demonstrates that neurosurgery has a role in the management of brain injuries that result from more than simple direct trauma.

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