Case report - Vascular thoracic

Recovery of severe neurological dysfunction after restoration of cerebral blood flow in acute aortic dissection

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Abstract

Emergency repair has been successfully performed in acute type A aortic dissection complicated by cerebral malperfusion. Despite the lack of criteria to define irreversible brain damage, immediate surgery is often denied in case of stroke or coma. We report two patients presenting with coma and altered brainstem reflexes shortly after onset of aortic dissection, in whom aortic repair was successfully undertaken.

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1. Introduction

Non-operative management of type A acute aortic dissection carries a dismal prognosis. Neurological symptoms secondary to arch vessel malperfusion are documented in up to 20% of the cases [1]. Although stroke or coma are traditional contraindications for immediate surgery, and the criteria to define irreversible brain damage remain unclear, successful emergency repair has been reported [2–5]. We report two operations in patients presenting with coma and absent or abnormal brainstem reflexes shortly after onset of dissection.

2. Case reports

2.1. Case 1

A 58-year-old hypertensive man with a family history of aortic aneurysm was admitted to the emergency department 1 h after chest pain onset, followed by dysarthria and immediate loss of consciousness. On admission, the patient showed a Glasgow coma score (GCS) of three, anisocoria with fixed right-sided mydriasis, bilaterally absent corneal reflex, an apneustic breathing pattern and hypotension (80/50 mmHg). Mechanical ventilatory support was started and computed tomography documented type A aortic dissection with distal abdominal extension, partial arch vessel compression, and normal brain scans (Fig. 1, left panel). Although initially absent brainstem reflexes dictated an non-operative approach, subsequent re-evaluation showed neurological improvement with a GCS = 6, left upper hemiplegia with contralateral and lower left limb spontaneous movements, left Babinski sign, and right upper extremity flexion for pain localization. Tendon reflexes and pupillary reactivity were normal. Electroencephalography showed diffuse and focal right frontal abnormalities, with no epileptic foci. The patient was scheduled for immediate surgery.

Hemiarch aortic replacement was performed adopting an arch-first strategy and left femoral arterial inflow. Distal repair was accomplished during hypothermic circulatory arrest and innominate and left carotid perfusion, with left subclavian artery snaring (22 °C nasopharyngeal temperature; α-stat acid-base management). Cardiopulmonary bypass and circulatory arrest duration (antegrade cerebral perfusion) were 203 and 32 min. Weaning from bypass required only moderate catecholamine support (0.07 μg/kg/min epinephrine infusion). The patient progressively regained neurocognitive function after a period of profound coma, and was discharged from the intensive care unit 34 days after the operation.

During rehabilitation, 49 days after surgical repair, cognitive function was assessed with the P300 component of auditory-evoked potentials. The latency of the P300 component was 376 ms, whereas computed tomography documented a right-sided hemispheric stroke, (Fig. 1, middle and right panels). The patient was discharged home on the 102nd postoperative day. At 38 months follow-up, there were no late cardiovascular sequelae and the patient showed residual left upper hemiparesis with mild dysarthria and normal cognitive function.

2.2. Case 2

A 52-year-old hypertensive man with a history of alcohol abuse was transferred to our unit 3 h after onset of acute chest pain, with type A aortic dissection diagnosed at...
Fig. 1. Patient 1: Normal preoperative brain scan (a). Cognitive evoked potentials recorded from the medium frontal, central, and parietal regions, applying biaural stimulation with tone bursts of equal sound level, but different frequency and duration for target and non-target stimuli (80 dB; 2000 Hz/100 ms and 1000 Hz/10 ms). The patient was asked to report the number of target tones after each run, repeating the test three times to ensure a sustained level of attention. Recordings showed mildly-delayed and normal latency peaks of the P300 wave (376 ms) and N1 component (98 ms) in traces recorded from target (I–III) and non-target (IV–VI) stimulation in frontal, central, and parietal channels, indicating mild cognitive impairment and normal level of attention (b). Right-sided hemispheric stroke (c).

Fig. 2. Patient 2: Postoperative right-located stroke (a). Reperfused arch vessels and residual dissection, in axial (b) and sagittal (c) scans, respectively.

another hospital. The patient, who initially showed mild–moderate neurological impairment (GCS = 12), deteriorated during transfer, became unconscious (GCS = 5) and subsequently sustained cardiac arrest. On arrival after repeated cardiopulmonary resuscitation, anisocoria with left-sided mydriasis, and diffuse ST segment elevation at the electro-
cardiogram, suggesting coronary ostial involvement, were also observed. Echocardiography documented severe aortic regurgitation.

Emergency root replacement with a valved conduit was performed with open distal repair at 24 °C, employing selective antegrade cerebral perfusion. Cardiopulmonary bypass, cardioplegic arrest and antegrade cerebral perfusion times were 293, 141, and 22 min. The patient was weaned from extracorporeal perfusion with a 0.04 µg/kg/min epinephrine infusion. Neurocognitive function improved after a period of coma, and progressively returned to baseline. The only neurological motor deficit was temporary right upper hemiparesis. Computed tomograms documented a right-sided hemispheric stroke, and reperfused arch vessels despite residual arch dissection (Fig. 2). The patient was discharged from the intensive care unit 36 days after surgery.

3. Discussion

Medical therapy for type A acute aortic dissection yields unfavourable results. Although successful repair has been reported, preoperative stroke and especially coma are usually considered contraindications for immediate surgery [2–5], in spite of the absence of criteria to define irreversible brain damage preoperatively.

In the first patient, short-term delayed repair was performed after resuscitative measures in the comatose patient, and the timing of the indication was primarily based on the resumption of initially absent brainstem reflexes, whereas the second patient underwent immediate surgery. The postoperative period was temporarily characterized by profound coma, but late recovery was dramatic in apparently hopeless conditions. This suggests the possible benefits of immediate restoration of cerebral blood flow, even in case of altered or absent brainstem reflexes, and outlines the unreliability of the widely adopted Glasgow coma scale for patient stratification, as previously outlined in a small case series by our group in which the preservation of brainstem reflexes was considered a criterion to indicate emergent repair [5]. It might also be speculated that, in case of partial compression of the arch vessels, neurological dysfunction may have a higher potential for recovery. Finally, P300 peak latencies recorded with cognitive evoked potentials represent a useful tool to evaluate neurocognitive function, and are normally increased soon after open-heart operations [6]. In our first patient, the P300 latency recorded <2 months after the acute event, was only mildly increased when compared to healthy controls, and was similar to measurements after valve surgery.

Our experience stresses the potential for reversibility of dissection-induced neurological injury, and confirms a higher likelihood of a more severe ischaemic insult in right-sided territories. Extensive arch surgery was not performed because of the absence of intimal tears in the arch. However, although surgeons may be reluctant to indicate operations requiring prolonged and deeply hypothermic cardiopulmonary bypass with circulatory arrest, brain reperfusion at lower temperatures might actually represent a condition with a higher probability of neurological recovery, as recently documented in experimental settings [7]. We conclude that the resumption of initially absent brainstem activity with a substantially normal brain scan may indicate a higher probability of recovery, but its complete or partial loss does not imply irreversible brain damage. Restoration of cerebral blood flow and reversal of arch vessel compression should thus be promoted as soon as possible, and the absence of brainstem reflexes should not be considered a criterion to absolutely contraindicate immediate aortic repair.

References


eComment: Does coma state really stop from operating type A aortic dissection patients?

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We read with interest the case report by Pocar et al. who achieved positive results in the care of these patients [1]. We would like to comment on some aspects of this challenging pathology surgeons encounter.

Acute type A aortic dissection is a highly lethal disease for which prompt identification and surgery is the best hope for survival [2]. It has been reported that the major determinants of surgical outcome in acute aortic dissection patients are preoperative complications and comorbidities, such as shock, aortic rupture, severe neurologic damage, and visceral ischemia [3]. However, we have concerns about evaluating the patient’s neurological condition using Glasgow Coma Scale (GCS) score after aortic dissection. In fact, GCS score was developed to evaluate the level of consciousness in patients after head injury [4] who might have organic damage to the brain tissue and neurological system. But in case of malper-
fusion caused by aortic dissection, the pathophysiology might be different than the head injury. So, it could mislead the physician not to operate the patient presenting with a coma. Rather, operating and establishing the cerebral blood flow can revert the process, as presented by the authors. Furthermore, it has been emphasized that the primary objectives of surgical treatment of ascending aorta dissection are to prevent death of the patients from aortic rupture including cardiac tamponade and to re-establish flow in areas that have been occluded by the dissection. Hence, prompt surgical treatment could save a patient’s life. The IRAD risk prediction tool can provide an accurate prediction of mortality risk and can assist with the decision on whether or not to proceed with surgery. Nevertheless, these risk predicting models are good to guide us mainly in difficult cases and in other circumstances in which surgery may be deferred due to age and other comorbidities, but they are not beneficial in young patients, who generally all go to surgery [2].

Additionally, we would like to comment on the cannulation strategy in these patients. As it has been shown in a large study by Svensson et al. axillary artery cannulation used to establish cardiopulmonary bypass can provide continuous antegrade cerebral perfusion, which ultimately results in lower postoperative stroke and mortality rate [5].

References