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CASE REPORT

Intraoperative Pulseless Electrical Activity Cardiac Arrest during Intracranial Aneurysm Clipping: Case Report

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Abstract

Background: Cardiac arrest is a rare intraoperative complication with few cases reported during neurosurgery. Proposed causes of PEA cardiac arrest in this setting include medications, irrigation, coronary vasospasm, and elicitation of the trigeminocardiac reflex.

Case description: Here, we report the case of a 52-year-old man who suffered an intra-operative pulseless electrical activity (PEA) cardiac-arrest while undergoing elective clipping of a right MCA bifurcation aneurysm through pterional craniotomy.

The patient was successfully resuscitated following 2 minutes of chest compressions and the administration of Adrenaline, in accordance with ALS algorithm. He made a good post-operative recovery, without evidence of any new deficit.

No biochemical, electrophysiological, or structural abnormality was identified to explain this patient's sudden intra-operative PEA arrest. We consider and discuss the alternative explanations in the literature.

Conclusions: Intra-operative cardiac arrest is a rare and potentially catastrophic event. Understanding the cause of cardiac arrest allows for prompt treatment and reversal of any precipitant, and there are specific considerations in the neurosurgical setting. Adherence to the advanced life support (ALS) algorithm may be life-saving even when the cause is unknown.

Keywords

Case report, Aneurysm, Clipping, Neurosurgery, Cardiac arrest

Introduction

Cardiac arrest is an increasingly rare intraoperative complication [1], and relatively few cases have been reported during neurosurgery [2,3]. The few recorded cases of intraoperative pulseless electrical activity (PEA) cardiac arrest during neurosurgery have been ascribed to medications, [4-6] irrigation, [7] coronary vasospasm, [8] and most commonly to the trigeminocardiac reflex [9-12].

Here, we report the case of a 52-year-old man who suffered an intra-operative PEA cardiac-arrest while undergoing elective clipping of a right MCA bifurcation aneurysm through pterional craniotomy, in the absence of an explanatory cause.

The patient consented to have the details of their case published in this anonymised case report.

Case Report

A 52-year-old man presented with an incidental finding of 12 mm right MCA bifurcation aneurysm on imaging. The scan was carried out for resection of a grade 1 myxopapillary ependymoma. Cerebral angiography confirmed the aneurysm, and the patient was discussed in the neurovascular multidisciplinary team meeting whereby elective surgery for aneurysm clipping was offered.

The patient was deemed to be low risk from an anaesthetic perspective. He had an uneventful

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intravenous induction with Propofol (150 mg), Fentanyl (100 mcg) and Rocuronium (60 mg), with Fentanyl and Propofol boluses to cover intubation and head-pin insertion. He was maintained on Sevoflurane (1.1 MAC) and Remifentanil (0.1-0.25 mcg/kg/min). No Adenosine was used.

The aneurysm was approached via pterional craniotomy with the patient supine. The Sylvian fissure was opened and the temporal M2 and frontal M2 were visualised. The aneurysm dome was dissected completely from the surrounding vessels. Frontal and temporal retractors were then inserted to prepare for placement of the clip. As the clip was placed open across the neck of the aneurysm, the anaesthetist alerted the surgical team to haemodynamic instability of the patient, noting a rapid fall in systolic blood pressure to 40 mmHg. His peripheral pulse was unrecordable and cardiac output was lost.

His electrocardiogram (ECG) showed sinus rhythm and there was no change to either rate or morphology. The plethysmograph trace was lost, followed by a drop in the carbon dioxide trace. Oxygen saturation was maintained above 96% throughout. PEA arrest was declared. The 7 mm clip was released across the neck of the aneurysm, the surgical site was covered, and the team proceeded to chest compressions, lasting 2 minutes. During this period the carbon dioxide trace increased, suggesting a loss of cardiac output rather than an embolic event. Intravenous Adrenaline (1 mg) was administered, with immediate return of cardiac output and a systolic blood pressure in excess of 240 mmHg.

The patient's blood pressure gradually returned to the normal range over the following 5 minutes. The surgical team then returned to the operative field. Occlusion of the aneurysm and the patency of the lenticulostriate perforators and the M2 divisions was confirmed using indocyanine green angiography. The rest of the operation was uneventful, with closure of the dura, bone flap and wound.

The patient made an excellent post-operative recovery and did not complain of any chest pain or breathing difficulty, nor did he have any new neurological deficit. Post-operative ECG did not find evidence of ischaemia or infarct, nor was there any evidence of an arrythmia, heart-strain, or any other cardiac abnormality. Troponin T after the event was found to be only 33 ng/L (0-14 ng/L) and stable on repeat test. Additionally, no electrolyte or haematological abnormality was identified. Post-operative computed tomography (CT) of his head showed only expected post-operative changes. The patient stayed for observation, and on the third post-operative he was discharged with cardiology follow-up.

Discussion

Common explanations for PEA arrest could not account for this patient's sudden intra-operative course. The quick reversibility of cardiac arrest with no residual deficit, and the lack of oxygen desaturation, electrolyte abnormality, or ECG changes precluded most primary cardiopulmonary explanations. Toxins may also produce PEA arrest, as in the case of the Bezold-Jarisch reflex, [13] however, the patient was not exposed to any new compounds, and the various anaesthetic agents had been introduced and in use for over an hour prior to the event.

One of the most commonly proposed mechanisms of PEA arrest in neurosurgical procedures is the trigeminocardiac reflex (TCR). This neurogenic brainstem reflex is elicited by the stimulation of trigeminal afferents, leading to increased vagal tone with consequent bradycardia, hypotension, and gastric hypermotility [14,15]. Several case reports attribute periods of bradycardia and hypotension during neurosurgery to this phenomenon [12,16-22]. The finding that the ophthalmic division of the trigeminal nerve supplies the supratentorial cerebral vasculature and dura of the rhesus macaque [23] has been proposed as a mechanism by which the TCR could be elicited during aneurysmal clipping [21]. Additionally, there are at least 2 case series' which have suggested an association between Remifentanil and elicitation of the TCR [19,22]. However, in this case, Remifentanil was used in low doses, and not newly initiated around the time of arrest. Moreover, whilst the previous literature has reported dural manipulation causing the TCR, the dura were not manipulated around the time of this patient's cardiac arrest.

Intraoperative cardiac arrest poses unique challenges, including positioning and consideration of the operative field, both of which impede the execution of standard CPR protocols. Nevertheless, there are also several advantages of this setting: The patient has IV access, their vitals are constantly monitored, they are intubated with ventilatory support, and the anaesthetic team is present. Clear communication between anaesthetic and surgical teams throughout surgery was important in facilitating a rapid response in this case. This allowed for the delivery of prompt and effective CPR, which helped to maintain tissue oxygenation while Adrenaline was prepared and administered. Shortly after administration of Adrenaline, the patient's blood pressure rose above 240 mmHg systolic. Retrospectively, it was perhaps beneficial to have the aneurysm clipped as these pressures may have precipitated intra-operative aneurysmal rupture.

Conclusion

Presumably, this patient's sudden loss of cardiac output was triggered by intracranial stimulation of the vagus nerve. However, the exact aetiology remains unclear as this case does not share many of the

features that have typically led others to infer that the TCR is responsible. The sympathomimetic effects of Adrenaline were sufficient to overcome the increased vagal tone and the decision to secure the aneurysm clip may have protected the patient from the effects of the resulting hypertension. Further research is required to more definitively establish both the cause and optimal preventative and treatment measures.

Declarations

This manuscript is original and has not been submitted elsewhere in part or in whole. The authors have no conflicts of interest or sources of funding to declare.

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