# RUPTURE OF INTRACRANIAL ANEURYSM AFTER PARTIAL CLIPPING DUE TO ASPIRATION DRAINAGE SYSTEM

## - A Case Report -

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## Summary

In neurosurgical practice, extradural or subgaleal drains are commonly placed and connected to a vacuum system. Several reports have described severe bradycardia or arterial hypotension, or both, after connection of negative suction pressure to the extradural or epicranial drains following craniotomy. We encountered an unusual complication with the use of the vacuum drain after an elective aneurysmal clipping surgery. Our case is an iatrogenic intracranial hypotension leading to a clinically significant and potentially fatal complication.

**Keywords:** Equipment, vacuum drainage system; Complication, arrhythmias; intracranial hypotension; aneurysm rupture.

Many neurosurgical procedures, involving craniotomy, require the use of extradural or epicranial drains, which are connected to a vacuum device for preventing hematoma formation or drain obstruction. Severe cardiovascular disorders (bradycardia, asystole or arterial hypotension) related to their use have been published[1-3]. We report a potentially fatal and

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hitherto unreported complication with the use of the vacuum drain after an elective aneurysmal clipping surgery.

## Case Report

A 45-year-old male, weighing 60 kg, was admitted for elective clipping of left internal carotid artery aneurysm. On admission, the patient had Hunt and Hess SAH grading II. His past medical history was remarkable for hypertension since 2 years controlled with tablet Atenolol 50 mg. All preoperative investigations were within normal limits.

The patient was premedicated with Glycopyrrolate 0.2 mg intramuscularly, one hour prior to surgery. Inside the operation theatre, an 18 G cannula was placed on the dorsum of right hand. Standard monitors of electrocardiogram, non-invasive blood pressure and pulse oximeter, were attached. General anesthesia was induced with thiopentone sodium 300 mg and fentanyl 125 mcg. To facilitate tracheal intubation with cuffed oro-tracheal tube size 8.5, rocuronium bromide 60 mg was given. Anesthesia was maintained with isoflurane (MAC 0.8-1.2) in a mixture of $O_2$ and $N_2O$ (40:60) and fentanyl. Neuromuscular block was produced with vecuronium bromide 0.1-mg $kg^{-1}$ $hr^{-1}$. Controlled mechanical ventilation (Ohmeda 7000 ventilator) was adjusted to maintain end-tidal carbon dioxide pressure at 30 ± 2 mmHg. A 20 G cannula was placed in left dorsalis pedis artery for continuous monitoring of invasive blood pressure. A single lumen central venous catheter was placed in the left basilic vein. Nasopharyngeal temperature was monitored intraoperatively. As per the prevailing institutional protocol, mild hypothermia during aneurysm surgery, water mattress was used to regulate patient's body temperature.

The scalp was infiltrated with 2% lignocaine before surgical incision. A left pterional craniotomy was done with patient in supine position. Mannitol 1 gm $kg^{-1}$ was infused at the time of craniotomy. Following craniotomy the surgeons noticed a lax brain and located the aneurysm with much ease. Two attempts were made to temporarily clip the aneurysm before applying the permanent clip. The total duration of temporary clipping was 5 minutes.

The rest of the surgical course was uneventful. The duramater was closed in a watertight manner. Following bone flap replacement, a subgaleal drain (Romo Vac Set, Romsons) was placed and scalp closure done.

After scalp closure and applying dressing to the surgical wound, the bellow unit with subatmospheric pressure was connected to the subgaleal drain. Suddenly a gush of fresh blood filled the bellow unit and nearly 300 ml blood collection was noted. Suspecting intracranial bleeder vessel, the surgeons immediately decided to re-explore.

The anesthetic plane was again deepened. The sutures were opened and the surgeons noticed arterial rupture at the site of incompletely clipped aneurysm. A second larger clip was applied at the same site to stop the bleeding. The bleeding was immediately controlled.

After surgical closure for the second time the bellow unit was applied to the subgaleal drain without any negative pressure. Total blood loss was approximately 2 litres and was adequately replaced.

The patient was then shifted to the neurosurgical intensive care unit (NICU) for elective ventilation. The patient had a stormy course in the NICU, mainly due to chest infection, which developed in the following days. He was gradually weaned and extubated on the eighth postoperative day. The patient had a full score of Glasgow Coma Scale with right hemiparesis. However, the patient progressively deteriorated on account of meningitis and chest infection. He ultimately succumbed to septicemia on the fifteenth post-operative day.

## Discussion

In neurosurgical practice, some conditions require the use of drainage system, such as in brain tumors, subdural and extradural empyemas, chronic and acute subdural or extradural hematomas, and brain abscesses. These drainage systems include epidural and subgaleal drains that are connected with vacuum bottles[1]. Several reports have described severe bradycardia or arterial hypotension, or both, after connection of negative suction pressure to the extradural or epicranial drains after craniotomy[4-6].

The possible mechanism for the above complication is that application of negative pressure via extradural or epicranial drain, may lead to a sudden state of intracranial hypotension resulting in a rostral movement of the brain or brain stem nuclei responsible for the changes in cardiac rhythm or arterial hypotension, or both[1,4,7,8]. Intracranial hypotension may develop spontaneously after trauma to the head followed by CSF rhinorrhea or otorrhea, as a complication following lumbar puncture and rapid withdrawal of a large amount of CSF, or after installation of a shunt system with a low opening pressure[1].

Hernandez and coworkers[6] demonstrated a significant decrease in heart rate and intracranial pressure with application of negative pressure through the extradural drain connected to a vacuum system. But they did not find significant decrease in blood pressure. If the blood pressure remains relatively unchanged and there is a sudden decrease in intracranial pressure, the transmural pressure which is the ultimate determinant of aneurysm rupture, may suddenly increase and lead to its rupture. Rapid administration of mannitol has also been blamed for intraoperative aneurysm rupture due to sudden lowering of intracranial pressure[9].

We report a case of iatrogenic intracranial hypotension leading to a clinically significant and potentially fatal complication. It is believed that sudden intracranial hypotension may have been responsible for the rupture of partially clipped aneurysm in our case. Sudden intracranial hypotension resulted in a significant increase in the transmural pressure, as a result of which, the part of the aneurysm neck that was unclipped, ruptured. The first permanent clip was still in place when the re-exploration was done. Hence slippage of the clip causing intracranial bleeding seems unlikely. The complication occurred at the time of extubation of the patient. The systolic arterial blood pressure at that time was 160-170 mmHg. Though the pressure was not high enough to cause such complication, the fact that it was associated with simultaneous and sudden reduction in the intracranial pressure by the negative pressure in the suction device, resulted in rupture of the aneurysm. At the same time, it must also be considered that possibly the aneurysm had not been properly clipped. The transmission of the

negative pressure from the epidural or subgaleal drain to the ventricles occurs as a consequence of communication between the epidural and subdural spaces following dural closure. Communicating with the surgeon and releasing the negative pressure in the suction device can easily reverse the hemodynamic changes produced as a result of intracranial hypotension. But the complication we encountered was potentially fatal.

In conclusion, we report a case of aneurysm rupture as a result of sudden intracranial hypotension. The cause of intracranial hypotension was iatrogenic and produced by the negative pressure applied to the subgaleal drain.

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