Case reports

Adjuvant hyperbaric oxygen treatment of acute brain herniation after microsurgical clipping of a recurring cerebral aneurysm: a case report

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Abstract
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Introduction: Acute brain herniation is a life-threatening neurological condition that occasionally develops due to severe complications following cerebral aneurysm clipping. Strategies for managing acute brain herniation have not improved substantially during the past decade. Hyperbaric oxygen treatment (HBOT) may alleviate harmful effects of cerebral hypoxia, which is one of the most important pathophysiological features of acute brain herniation and, therefore, may be useful as an adjuvant therapy for acute brain herniation. A case treated with adjuvant HBOT is reported.
Case report: A 60-year-old asymptomatic man presented with a recurring left middle cerebral artery bifurcation aneurysm with previous stent-assisted embolisation. After craniotomy for surgical clipping of the aneurysm, disturbance of consciousness and right hemiplegia occurred. Computed tomography (CT) images suggested simultaneous cerebral ischaemia and intracranial haemorrhage. Pharmacologic treatment resulted in no improvement. A CT scan acquired five days after surgery showed uncal and falcine herniation. HBOT was administered five days after surgery, and the patient's condition dramatically improved. He became conscious, and his hemiplegia improved following seven sessions of HBOT. Simultaneously, CT images showed regression of the acute brain herniation.
Conclusions: The patient had recovered completely at one year post-treatment. HBOT may be effective in the treatment of acute brain herniation following cerebral aneurysm clipping.

Introduction

Acute brain herniation is associated with high mortality and morbidity.[1] It may be caused by severe brain oedema secondary to several medical conditions, including cerebral ischaemia and intracranial haemorrhage, either of which may complicate surgery for aneurysm clipping. Although intracranial pressure monitoring and decompressive craniectomy are used to treat acute brain herniation, their efficacy remains controversial.[2]

Hypoxia is the most important pathophysiological characteristic of acute brain herniation.[1] Hyperbaric oxygen treatment (HBOT), defined as respiration of 100% oxygen at a pressure > 101 kPa, improves hypoxia in the brain tissue.[3,4] However, HBOT's utility as an acute brain herniation treatment is unknown. Herein is reported, to the best of the author's knowledge, the first case of acute brain herniation treated with adjuvant HBOT.

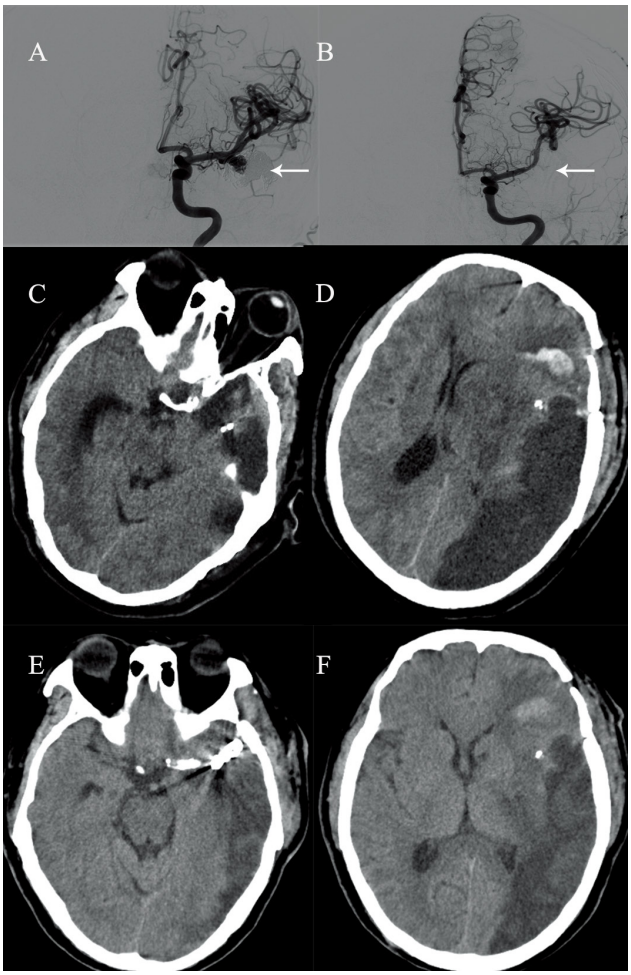
Case report

Consent for publication of this case was obtained from the patient via his immediate family.

A 60-year-old asymptomatic man was admitted to our hospital due to a recurring left middle cerebral artery (MCA) bifurcation aneurysm with a history of two stent-assisted coil embolisation procedures (Figure 1A). He had been diagnosed with a subarachnoid haemorrhage with headache four years prior and had chronic hypertension and kidney stones for 10 years.

After hospitalisation, the aneurysm was completely clipped (Figure 1B). The saccular aneurysm was 30 mm in dome diameter and 5 mm in neck diameter. Balloon dilation was performed to the M1 segment of the MCA because vasospasm was found using intraoperative digital subtraction angiography (DSA). Patency of the left superior branch of the MCA and perforating arteries was visible on

Figure 1
Digital subtraction angiography (DSA) and computed tomography (CT) images. DSA images show a recurring left middle cerebral artery bifurcation aneurysm with a history of two stent-assisted coil embolisation procedures, (A) at admission (arrow) and (B) after complete surgical clipping of the aneurysm (arrow). After attempted pharmaceutical treatment CT images acquired 5 days after surgery (C) show compression of the left cerebral peduncle and (D) a rightward shift of the cerebral midline. Following seven sessions of hyperbaric oxygen therapy CT images show (E) alleviated compression of the left cerebral peduncle and (F) an unbiased cerebral midline



intraoperative DSA images, while the left inferior trunk of the MCA was not visible (Figure 1B). Surgery lasted for 6 h and no cerebrospinal fluid drainage tube was placed.

Postoperatively, the patient suffered somnolence and right hemiplegia without sedatives, with a Glasgow Coma Scale score of 5. Head computed tomography (CT) performed 1-day post-surgery showed slight distortion of the left side of the brainstem due to simultaneous cerebral ischaemia and intracranial haemorrhage. He maintained his airway and was not intubated. Drugs including mannitol, steroids, midazolam, nimodipine, aspirin, clopidogrel, and hydroxyethyl starch were administered. However, no significant improvements occurred. CT images acquired five days post-surgery revealed a narrowed cisterna interpeduncularis, brainstem distortion, and cerebral midline shift (Figure 1C and D). Pupil diameter and light reflexes were normal.

HBOT was initiated on day 5 post-surgery along with drug administration. The multiplace hyperbaric oxygen chamber was pressurised to 203 kPa over 30 min, maintained at 203 kPa for 1 h, and then depressurised over 30 min. During the compression and decompression phases, the patient respired air; during the steady pressure period (203 kPa), he respired 100% oxygen. In total, seven consecutive daily sessions of HBOT were performed. Medical staff accompanied the patient in the chamber to monitor his electrocardiograph, blood pressure, respiratory rate, and general condition. During each HBOT session, his vital signs were stable and within the normal limits, and there were no signs of ear barotrauma or signs of cerebral oxygen toxicity.

After session one, the patient showed increased pain sensitivity. Following session three, he was conscious with mixed aphasia. After session seven, he demonstrated significant improvements in muscle strength and speech. CT images showed no midline shift and no brainstem compression (Figure 1E and F).

At the 3-month follow-up, the patient had hemiparesis and normal speech. CT angiography showed no recurrent aneurysm. At the 1- and 3-year follow-ups conducted via telephone, he had completely recovered.

Discussion

Simultaneous cerebral ischaemia and intracranial haemorrhage were the primary injuries in this patient, while secondary brain oedema led to acute brain herniation. Cerebral ischaemia was present in the region of the left inferior trunk of the MCA, while intracranial haemorrhage may have occurred due to procedure-related complications. Despite controversy regarding the effects of HBOT on cerebral ischaemia and intracranial haemorrhage,[4,5] we chose this intervention because (i) the lack of improvement after medication and risks associated with a decompressive craniotomy were of concern, and (ii) as oxygen is a limiting factor in brain injury recovery, HBOT's potential role in improving brain tissue oxygenation and metabolism was considered important.[3]

EFFICACY OF HBOT

The patient's acute brain herniation receded after treatment with medication and HBOT, resulting in symptomatic and CT image improvement. HBOT for traumatic brain injury has received considerable attention in the past several decades despite the controversy surrounding it.[6] However, few reports of HBOT being used to treat patients after

cerebral aneurysm clipping exist. One study noted that early HBOT improved the postoperative outcomes of patients with intracranial aneurysm.[7] Owing to the acute brain herniation, our patient was in more critical condition than those in the previous study; nonetheless, treatment with HBOT was associated with a good outcome. It is speculated that the HBOT protocol, specifically the timing and dosage, contributed to the good anecdotal outcome.

Regarding timing, early HBOT may help resolve brain hypoxia, which causes brain injury in acute brain herniation.[1,7] Although our patient had consciousness disturbances, his pupil diameters and reflexes were normal, indicating that he was in the early stages of acute brain herniation, prior to the occurrence of oculomotor nerve compromise. Currently, no consensus exists regarding appropriate HBOT timing.[8] Several studies initiated HBOT early after injury, while some suggested that HBOT should begin one month later, citing concerns surrounding oxygen toxicity.[8] We did not observe seizures (an oxygen toxicity sign), providing some reassurance that our early timing was appropriate.

The dose of HBOT is likewise crucial.[8] HBOT at 203 kPa elevates normal brain tissue oxygen pressure from 35 to 240 mmHg and increases brain oxygen diffusion.[9] At 203 kPa, the ability of aquaporin 4 to alleviate cerebral oedema is the strongest.[10] Here, repetitive HBOT was administered, and the patient's condition gradually improved. This might be associated with gradual elevation in cerebral tissue oxygen pressure.[11]

HBOT is considered safe when administered at pressures < 304 kPa for < 2 h.[12] The present case provides qualified support (albeit in only a single case) for the safety of HBOT at 203 kPa for the critically ill patient. We performed monitoring to ensure timely detection and intervention in the event of changes in patient condition.[12]

LIMITATIONS

As HBOT and appropriate medications were used simultaneously, both treatments may have contributed to the regression of acute brain herniation. However, the patient's abrupt improvement after HBOT and lack of improvement after earlier use of medication alone suggests that the improvements are related, at least in part, to HBOT.

Conclusion

This case suggests that HBOT may be an effective and safe treatment for critically ill patients with acute brain herniation following complicated cerebral aneurysm clipping. As this was a single case report, clinical studies with larger sample sizes are required to further elucidate a role for HBOT in this setting.

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