

# Venous Air Embolism during Elective Craniotomy for Parasagittal Meningioma

W Mohd Nazaruddin W Hassan, MMed (Anaesth), Asmah Zainuddin, MMed(Anaesth), Saedah Ali, MMed(Anaesth)

School of Medical Sciences, Universiti Sains Malaysia, Anaesthesiology and Intensive Care, Jalan Sultanah Zainab II, Kota Bharu, Kelantan 16150, Malaysia

## SUMMARY

We report a case of a 59 year old man who developed venous air embolism (VAE) during an elective craniotomy for parasagittal meningioma resection. The surgery was done in the supine position with slightly elevated head position. VAE was provisionally diagnosed by sudden decreased in the end tidal carbon dioxide pressure from 34 to 18 mmHg, followed by marked hypotension and atrial fibrillation. Prompt central venous blood aspiration, aggressive resuscitation and inotropic support managed to stabilize the patient. Post operatively, he was admitted in neuro intensive care unit and made a good recovery without serious complications.

## KEY WORDS:

*Venous air embolism, Parasagittal meningioma, Craniotomy*

## INTRODUCTION

Venous air embolism (VAE) is the entrainment of air from the surgical site into the venous system producing a broad array of symptoms and outcomes. Traditionally, it is commonly associated with neurosurgical procedures performed in the sitting position but more have been reported to occur in non-sitting and non-neurosurgical procedures. The incidence is as high as 80% for seated posterior fossa surgery and 15 to 25% for neurosurgical procedures performed in the lateral, supine or prone positions<sup>1,2</sup>. In surgery for meningioma, this complication has seldom been reported despite the usually rich vascularization of these lesions. There are few published reports on VAE occurring in supratentorial meningioma surgeries but based on our literature search, this was most likely the first reported case of VAE in the location of the parasagittal vicinity for meningioma excision. This was also our first experience of managing VAE during craniotomy.

## CASE REPORT

A 59 year old man presented with 1 year history of left hemiparesis and bilateral eye blindness. He was diagnosed to have a parasagittal meningioma.

He was planned for elective craniotomy and tumour excision. Preoperative evaluation revealed that he was a known case of non insulin dependent diabetes mellitus (NIDDM) and newly diagnosed idiopathic thrombocytopenic purpura (ITP). He was given a course of intravenous (IV) immunoglobulin

preoperatively and was also started on IV methylprednisolone. His platelet count was slightly low (91 x 10<sup>9</sup> /L), haemoglobin (Hb) was 12 g/dl, Glasgow coma scale (GCS) was 14/15. Chest X-ray (CXR) and electro cardiogram (ECG) were normal. CT brain showed right anterior third parasagittal meningioma with uncal herniation.

The patient was monitored with invasive monitoring including invasive arterial blood pressure (BP) and central venous pressure (CVP) via triple lumen internal jugular venous catheter. The central catheter was anchored at 15 cm which was slightly deep. There were no CXR or other techniques of placement confirmation of the central catheter were done. Other standard monitors included end tidal carbon dioxide (ETCO<sub>2</sub>), pulse oximetry (SpO<sub>2</sub>) and nasopharyngeal temperature. Position was in supine with head slightly elevated about 15°. Two other large bore intravenous (IV) cannulas were inserted. After induction with fentanyl, propofol and rocuronium, anaesthesia was maintained with target controlled infusion (TCI) of propofol and alfentanil infusion. During the excision of the tumour, there was significant blood loss about 1000-1500 ml with Hb dropped from 12.0 to 8.0 g/dl. Two units of packed red blood cells (RBC) and 4 units of platelet were initially transfused and haemodynamic was still stable.

Two hours after the surgery, while the surgeons were still securing the haemostasis after tumour excision, ETCO<sub>2</sub> suddenly dropped from 34 to 18 mmHg, his ECG showed supraventricular tachycardia (SVT) followed by atrial fibrillation (AF). His blood pressure (BP) dropped to the lowest reading of 30/15 mmHg but SpO<sub>2</sub> remained 100%. A provisional diagnosis of VAE was immediately made and other differential diagnoses were also considered such as hypovolaemic shocks, acute coronary syndrome with cardiogenic shock, venous thromboembolism and tension pneumothorax. However on auscultation, air entry was equal and lungs field were clear. There was no obvious murmur. The surgeons were informed to take appropriate surgical action by securing the haemostasis, flooding the surgical field with saline and applying the bone wax if necessary. Head position of the patient was still maintained at 15° so that we did not disturb the surgeon action. Inspired oxygen concentration was increased to 100%. About 80 ml of blood aspirated from the central line. It was unsure whether air was presence or not during the aspiration because the procedure was done under the drape. His BP slightly showed

*This article was accepted: 7 October 2012*

*Corresponding Author: W Mohd Nazaruddin W Hassan, School of Medical Sciences, Universiti Sains Malaysia, Anaesthesiology and Intensive Care, Jalan Sultanah Zainab II, Kota Bharu, Kelantan 16150, Malaysia Email: nazarudin@kb.usm.my*

an improvement. TCI of propofol was discontinued and anaesthesia was maintained with sevoflurane in view of hypotension and arrhythmia. Fluid resuscitation with colloid, crystalloids, blood and blood products were still continued. CVP at that time was 15 mm Hg. BP was supported with maximum dose of dobutamine, noradrenaline and adrenaline. After an hour of resuscitation, BP was managed to be maintained between 120/60 -140/80 mmHg, HR between 110-130 beats/min. Arterial blood gases (ABG) showed mixed metabolic and respiratory acidosis (pH 7.27, pO<sub>2</sub> .123 mmHg, pCO<sub>2</sub> 52.3 mmHg, HCO<sub>3</sub> 19 mmHg. ABG also noted hypocalcaemia (calcium level: 0.79 mmol/l) and IV calcium gluconate 20 mmol was slowly given. Amiodarone was also started. After resuscitation, latest Hb from ABG was 11.3 g/dL. We were told by the surgeons that during the event, they were operating at the sinus site and not much bleeding was noted. During the resuscitation, the surgeons managed to secure haemostasis. We abandoned the closure for a while during the resuscitation and allowed them to recommence the surgical closure quickly after BP was stabilized.

Postoperatively, 12 leads ECG showed AF, ST depression at leads V2-V6 with no right heart strain changes. Bedside transoesophageal echocardiography (TEE) showed no obvious air visualised but right atrium was dilated with tricuspid regurgitation. On auscultation, there were generalised fine crepitations of the lungs but no obvious murmur heard. Postoperative CXR showed acute pulmonary oedema. IV frusemide 20 mg was given. In neuro intensive care unit (NICU), his condition was improving. All cardiac enzymes and troponin T were not raised. On day 2 postoperative, patient was extubated at GCS 13/15 (eye: 4, motor: 6 and verbal response: 3) without any inotropic supports. He was discharged from the hospital after one month with GCS of 14/15.

## DISCUSSION

Parasagittal meningioma is defined as the tumour arising from the parasagittal angle without intervening brain parenchyma between the tumor and sagittal sinus. Its attachment or invasion to the superior sagittal sinus presents unique challenges for the surgery. Radical resection of meningioma involving major dural sinuses or bridging vein may be limited, particularly when these venous channels are patent.

Major complications of parasagittal meningioma included venous thrombosis/infarction (7%), intraoperative air embolism (1.5%), and death (1.5%). Recurrence rate was 11%<sup>3</sup>. There were few reported cases of VAE in meningioma cases. Gomez-Perals et al. reported a case of VAE during convexity meningioma surgery in the supine position. The episode occurred while they were cutting the bone for the craniotomy before turning the bone flap. Aspiration of irrigant into the cut bone surfaces through several venous diploic channels in the bone edge was observed. The diagnosis was made after sudden decrease in ETCO<sub>2</sub> from 32 to 12 mmHg, hypotension, higher inspired percentage of oxygen requirements and sinus bradycardia<sup>4</sup>.

Sudden decrease in at least 5 mmHg of ETCO<sub>2</sub> within 5 min is considered significant value to be suspicious of VAE. The principles of VAE managements include prevention of further air entry, a reduction in the volume of air entrained, if possible; and hemodynamic support<sup>5</sup>. Evidence of post operative acute pulmonary oedema and right atrial dilatation on TEE can be the signs strongly suggested the complications of VAE.

## CONCLUSIONS

Craniotomy for parasagittal meningioma is potential to cause VAE even though the surgery is in supine position with just slight head elevation.

## REFERENCES

1. Albin MS, Carroll RG, Maroon JC. Clinical considerations concerning detection of venous air embolism. *Neurosurgery* 1978;3: 380-4.
2. Sindou M, Auque J, Jouanneau E. Neurosurgery and the intracranial venous system. *Acta Neurochir Suppl* 2005; 94.
3. Raza SM, Gallia GL, Brem H, et al. Perioperative and long-term outcomes from the management of parasagittal meningiomas invading the superior sagittal sinus. *Neurosurgery* 2010;67: 885-9.
4. Gomez-Perals LF, Bayo R, Lorenzana-Honrado LM, et al. Severe intraoperative air embolism during convexity meningioma surgery in the supine position. Case report. *Surg Neurol* 2002;57: 262-6.
5. Mirski MA, Lele AV, Fitzsimmons L, et al. Diagnosis and treatment of vascular air embolism. *Anesthesiology* 2007;106: 164-77.