Case study: Systemic complications following absolute alcohol embolisation of scalp arteriovenous malformation

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Abstract
Alcohol ablation is an accepted technique for the management of arteriovenous malformations. It is preferred due to the unique property of absolute alcohol to cause complete ablation and prevention of revascularisation. However, this technique is associated with multiple complications which may lead to patient morbidity. Here the case is presented of a female patient with scalp arteriovenous malformation who underwent alcohol ablation and developed supraventricular arrhythmia accompanied with haemodynamic instability and intravascular haemolysis in the postoperative period.

Introduction
Arteriovenous malformations (AVM) are abnormal connections between hypertrophied inflow arteries and dilated outflow veins, shunting blood through a nidus. Surgery in addition to sclerotherapy[1] has evolved as the basis of treatment for these lesions. Various materials, such as platinum or steel micro coils, purified gelatine sponges, isobutyl 2 cyanoacrylate, silicon/polyvinyl alcohol beads, and absolute alcohol, are used for obliteration of these abnormal connections. Ethanol, due to its property of protein denaturation, leads to a complete and permanent obliteration of vascular lumens.[2,3] Here a case report is presented of a patient who underwent alcohol ablation of AVM and developed paroxysmal supraventricular arrhythmia with intravascular haemolysis in the postoperative period. The management along with a review of literature is presented.

Case report
A 45-year-old female weighing 60 kg reported to the neurosurgical outpatient section with a history of boggy swelling over the back of the head for the previous two months. Initially the swelling was small in size, gradually increasing to the size of 5 cm by 3 cm, not associated with vertigo, loss of consciousness, vomiting or headache. On examination the swelling was ill defined, with a positive compression sign suggesting vascular aetiology. Computed tomography showed the swelling to be extra axial over the left tempero-occipital area. The patient was planned for preoperative embolisation of AVM followed by surgical excision.

Embolisation of the AVM was done under intramuscular (IM) sedation with injection (inj.) pentazocine 30 mg and inj. promethazine 50 mg. After cannulation of the femoral artery a superselective catheter was passed and a contrast enhanced angiogram demonstrated a high-flow AVM feeding from the posterior division of the superficial temporal (STA), middle meningeal (MMA) and occipital artery (OA). A high-flow macro fistula was noted between the right occipital artery and a large venous sac in the suboccipital region. Draining veins were seen in the suboccipital area draining into external jugular vein. Absolute alcohol was injected into the feeder artery using the intermittent pulsed spray technique in aliquots of 1 ml. A total of 55 ml of alcohol was injected. A final angiogram showed obliteration of feeders from the STA and MMA and decreased flow across the arteriovenous fistula. The patient tolerated the procedure well and was transferred to the ward.

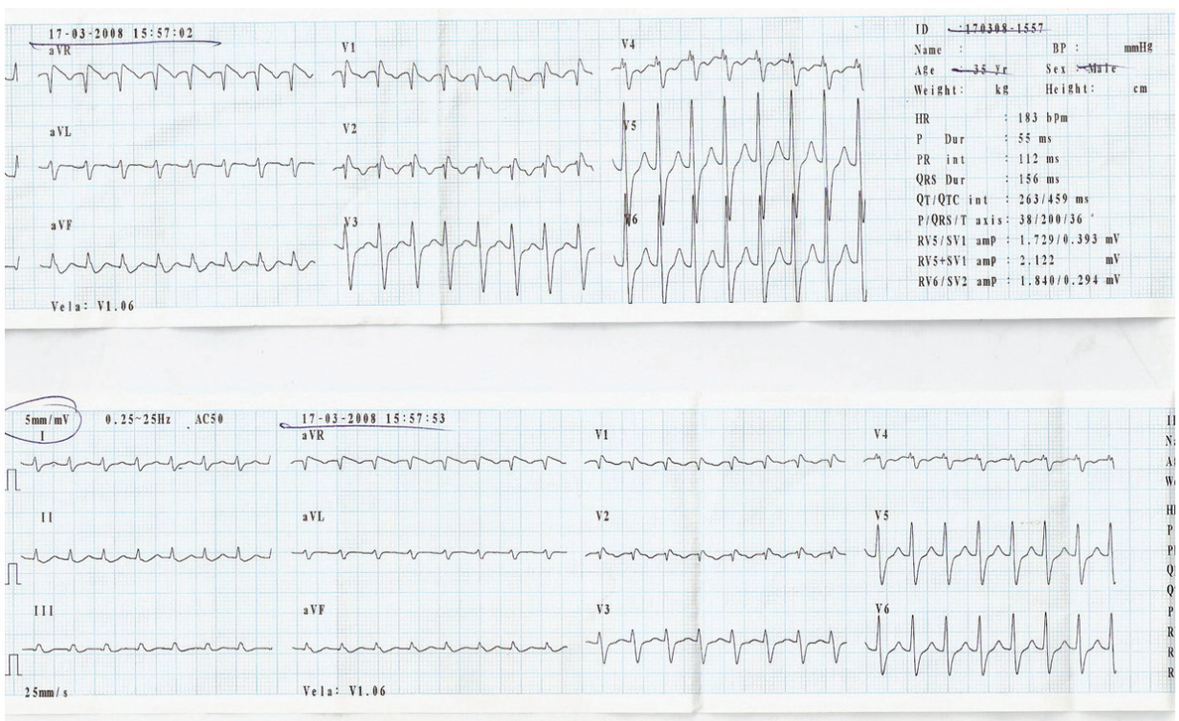
One hour after the procedure, it was noticed that the patient was hypotensive with a blood pressure of 80/60

mmHg, a tachycardia of 184/minute and a decreased level of consciousness with a Glasgow Coma Scale (GCS) score of 6/15. Anticipating complications pertaining to the alcohol injection, the patient was transferred to the intensive care unit (ICU). On arrival in ICU, the patient was found to be deeply comatose, not responding to deep painful stimuli, with a GCS score of 4/15 and a respiratory rate of twenty per minute, maintaining saturation of 96% with oxygen through a face mask at the Fio_2 of 50%. A 12-lead electrocardiogram (ECG) was obtained and a sample sent for arterial blood gas (ABG). The ECG showed the rhythm of paroxysmal supraventricular tachycardia (see Figure 1) associated with haemodynamic instability. Cardioversion with 50 J returned the rhythm to sinus. However, haemodynamic instability persisted, with a blood pressure of 70/40 mmHg. Intravenous dopamine infusion was started at 10 mcg/kg/min. The ABG report indicated the following values: pH 7.14, PCO_2 54 mmHg, PO_2 219 mmHg, HCO_3 18 mmol/l, BE -10.2, SaO_2 100%. In view of the unconsciousness, haemodynamic instability and acidosis the patient was intubated and connected to a ventilator. Blood sugar estimation done at this stage showed a value of 10 mmol/l. An intravenous dose of 50 ml of soda bicarbonate (7.5%) was given slowly. On catherisation of the bladder dark brown urine was collected, supporting the suspicion of intravascular haemolysis and haemoglobinuria. To prevent acute tubular necrosis associated with haemoglobinuria and compounded by hypovolaemia it was decided to induce forced diuresis by administering IV fluids and inj. furosemide 20 mg. The patient received about 6 000 ml of crystalloids overnight and had a urine output of 5 000 ml. By the next morning the colour of the urine had changed from dark brown to straw colour with a streak of redness. The haemodynamic parameters were slowly restored and the patient was responding to verbal commands in the morning, so the patient was extubated. A repeat ABG done 30 minutes after extubation showed pH 7.36, PCO_2 40 mmHg, PO_2 220 mmHg, HCO_3 22.6 mmol/l, BE -2.8, SaO_2 100%, Na 151 mmol/l, K 3.1 mmol/l. Blood investigations showed values of haemoglobin (Hb) 10.2 gm/dl, serum creatinine 167.96 µmol/l and normal serum electrolytes, blood sugar 5.5 mmol/l. Since there was a slight increase in the value of serum creatinine to 167.96 µmol/l the patient was monitored for serial kidney function tests for a further three days, first in the ICU and then in the high dependency unit (HDU), when the value returned to normal at 88.4 µmol/l. The patient underwent successful excision of the lesion after one week and was discharged in a satisfactory condition.

Discussion

Ethanol is an effective sclerosant for use in the treatment of vascular malformations. Its effects include protein denaturation resulting in clumping of blood cells, vessel wall necrosis resulting in thrombosis and permanent obliteration of the vessel. Ethanol is usually injected with some form of flow arrest to limit egress from the malformation due to its propensity to cause various side effects after systemic absorption.[4–9] Yakes et al reported a complication rate of 10–30% related to alcohol. [10]

Figure 1: The ECG showed the rhythm of paroxysmal supraventricular tachycardia associated with haemodynamic instability



The dose of ethanol used for ablation is the sole determinant of occurrence of these complications. Various authors[11,12] have determined that a total volume of 1 ml/kg is safe; however, haemolysis, cardiac arrhythmia and hypoglycaemia have occurred with volumes less than this recommended dose.

Acute elevation of alcohol concentration can put a patient at risk of atrial fibrillation, atrial flutter and ventricular tachycardia.[13,14] To our knowledge no specific serum level of ethanol in humans predisposes to increased risk of atrial arrhythmia in acute intoxication. The occurrence of paroxysmal supraventricular arrhythmia with ethanol is associated with electrolyte abnormalities, increaased catecholamine release, metabolic acidosis, increased oxidative stress and sleep apnoea.[15] An increased susceptibility is seen in patients with structural heart disease or subclin ical cardiac abnormalities. Alcohol-related arrhythmia might be attributed to intramyocardial catecholamine release or toxic effect of the metabolite acetaldehyde. These may have been the causes of the delayed onset of arrhythmia after the procedure in the current case. The inhibitory effect of alcohol on the sodium channels of cardiac cells is one of the proposed mechanisms of arrhythmia.[16]

A study in healthy physician volunteers showed a significant increase in pulmonary vascular resistance 30 minutes after the oral ingestion of 0.5 g/kg ethanol diluted to 15%, with return to normal values after 60 minutes.[17] Although the mechanism of ethanol-induced pulmonary vasoconstriction is not fully understood, there is some evidence that ethanol potentiates hypoxic pulmonary vasoconstriction.[17] In both animal and human models, doses of 0.5 g/kg or higher are associated with altered pulmonary and systemic haemodynamics.[18] The combined effect of an acute increase in pulmonary vascular resistance and negative inotropism can precipitate acute cor pulmonale in susceptible individuals. Patients with primary and secondary pulmonary hypertension may be especially sensitive to the pulmonary vasoconstrictive effects of large doses of intravenous ethanol.

In the absence of other plausible explanations and the temporal correlation with the injection of ethanol for ablation of AVM, we concluded that our patient's cardiovascular instability most likely was related to ethanol-induced pulmonary vasoconstriction and transient right ventricular dysfunction. No other sign of right ventricular dysfunction was found in this patient. Yakes et al[19] have postulated that pulmonary vasospasm caused by absolute ethanol results in acute pulmonary hypertension at precapillary level, which in turn increases right ventricular afterload and leads to right ventricular failure. Use of an indirect sympathomimetic to improve contractility, volume loading and the use of oxygen helped to ameliorate the detrimental effects of ethanol in this patient. Hypercarbia, acidosis and hypothermia should be aggressively treated because of their propensity to increase pulmonary vasoconstriction.[20]

To conclude, it is important that embolisation should be undertaken under monitored anaesthesia care or general anaesthesia and that anaesthesiologists should be aware of the possibility of acute haemolysis and periprocedural as well as postprocedural complications. Periprocedural monitoring and a high degree of vigilance are essential for the early detection and management of such complications.

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