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CASE REPORT

Acute Cardiopulmonary Decompensation Following Ethanol Sclerotherapy under General Anesthesia

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Ethanol sclerotherapy is a therapeutic method commonly applied to address low-flow vascular malformations. However, numerous complications, including hemodynamic instability, may develop after ethanol injection. We present a case who experienced cardiopulmonary instability during intraoperative percutaneous ethanol injection under general anesthesia and spontaneously recovered 5 min after ethanol injection. The toxic effects of the treatment were associated with the injection volume and may be related to ethanol-induced pulmonary vasoconstriction and transient right ventricular dysfunction. Prompt and proper management, including providing supportive treatment, decreasing pulmonary vascular resistance with pulmonary vasodilators, and improving right ventricular function, should be carefully prepared to minimize the progression of cardiovascular collapse.

Key words: Ethanol sclerotherapy, acute cardiopulmonary decompensation, general anesthesia

INTRODUCTION

Percutaneous intralesional injection of liquid sclerosing agents, also known as sclerotherapy, is a therapeutic method commonly applied to address low-flow vascular malformations. Ethanol is a safe and effective sclerosing agent.[1] However, the toxic effects of ethanol sclerotherapy are associated with the injection volume, and catastrophic complications may develop if the ethanol injection dose exceeds 1.0 mL kg^{-1}; these complications may include hemodynamic instability, seizures, respiratory depression, rhabdomyolysis, and hypoglycemia.[2,3] Herein, we report a case who experienced cardiopulmonary instability during intraoperative percutaneous ethanol injection under general anesthesia and spontaneously recovered 5 min after ethanol injection.

CASE REPORT

The ethics committee of the Tri-Service General Hospital approved this case report. (TSGHIRB No: A202105197). Our case is a 32-year-old female, with a height of 144 cm, weight of 44 kg, and no history of systemic disease. The patient has had vascular malformation on the left side of her face since childhood. The patient visited our hospital for the first time in 2019, and brain computed tomography revealed several enhanced lesions with venous engorgement and phlebolith formation involving the left face, left masticator space, and left orbital cavity, complicated with multiple hemangiomas. The patient received sclerotherapy with 95% ethanol injection for her facial lesions four times between the years 2019 and 2020. This time, the plastic surgeons performed local ethanol injection on her facial lesions. No intracranial or large vessel lesions were intervened during the whole procedure. General anesthesia was induced with propofol, fentanyl, and rocuronium and maintained by using target-controlled infusion (Fresenius Orchestra Primea; Fresenius Kabi AG, Bad Homburg, Germany) with propofol at an effect-site concentration of 3–4 mcg mL^{-1} in oxygen–air delivered through mechanical ventilation at a flow rate of 0.3 L min^{-1}. After injection of 6 mL of 95% ethanol, the patient developed bradycardia (40 beats min^{-1}), hypoxemia (peripheral oxygen

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saturation; SpO_2 82%), elevated airway pressure (from 15 cmH_2O to 41 cmH_2O), decreased end-tidal CO_2 (from 40 mmHg to 22 mmHg), and undetectable noninvasive blood pressure. The procedure was temporarily interrupted, and atrial catheterization was immediately performed for intensive hemodynamic monitoring. The patient's initial arterial blood pressure was revealed to be 75/50 mmHg. Blood gas analysis showed mixed respiratory–metabolic acidosis with pH 7.297, partial pressure of oxygen of 122 mmHg, partial pressure of carbon dioxide of 48 mmHg, bicarbonate of 23.7 mmol L^{-1}, and base excess of 2.4 mmol L^{-1} (fraction of inspired oxygen; FiO_2 88%). Approximately 5 min after the onset of cardiovascular instability, rapid infusion of 200 mL of normal saline was prescribed, and stable hemodynamics was spontaneously achieved. Sclerotherapy was restarted. A total volume of 13 mL of 95% ethanol was injected into the patient over the course of the procedure. The patient regained consciousness with spontaneous respiration, and her endotracheal tube was subsequently removed. The patient was transferred to the postanesthetic care unit. No complaints of pain were reported, and no neurologic sequelae were observed.

DISCUSSION

Ethanol produces various cardiopulmonary manifestations.[4] A previous clinical research demonstrated significantly elevated systolic and diastolic pulmonary arterial pressure (PAP) and pulmonary vascular resistance in six healthy physician volunteers 30 min after oral ingestion of 0.5 g kg^{-1} ethanol diluted to 15% (gram of solute per 100 mL of solution) with fruit juice.[5] Another prospective study including 14 female and 16 male patients with arteriovenous malformations divided patients retrospectively into two groups treated with and without vascular occlusion techniques; this study concluded that bolus injection of ethanol is strongly correlated with PAP elevation, especially in those treated without vascular occlusion techniques, and that ethanol blood levels are strongly related to mean PAP.[6] Mitchell *et al*.[2] conducted ethanol embolization procedures in 92 cases with vascular malformations and proposed that the upper limit of the ethanol injection volume should not exceed 0.1 mL kg^{-1} over a 5-min interval between injections to avoid cardiovascular collapse. To evaluate the systemic toxic influence of ethanol, Bisdorff *et al*.[7] retrospectively analyzed the data of 71 consecutive patients with venous malformation receiving ethanol sclerotherapy and found that adverse cardiopulmonary effects are initiated by an absolute ethanol dose of 0.24 mL kg^{-1}. The mechanism of ethanol-induced pulmonary vasoconstriction or vasospasm is unclear. Some scholars believe that ethanol induces hemolysis, which affects nitric oxide metabolism in the pulmonary circulation and suppresses nitric oxide.[8] Ethanol may induce myocardial contractile dysfunction and cardiac arrhythmia by impairing of calcium signaling in myocardiocytes;[9] the alcohol and its metabolite, acetaldehyde, also exert direct toxicity effects.[10] The pathophysiology has been attributed to direct toxicity to the cardiac conduction system or pulmonary arterial vasospasms leading to cardiovascular collapse.[2] We believe that the acute decompensation experienced by our case is related to ethanol-induced pulmonary vasoconstriction and transient right ventricular dysfunction. Although the total dose of ethanol injection was 13 mL, only the first 6 mL ethanol injection was responsible for the toxic effect. The first ethanol injection volume, at ~0.13 mL kg^{-1}, was much less than the upper limit of 1.0 mL kg^{-1} body weight, the devastating hemodynamic changes reported may have resulted from the sudden-bolus effect of ethanol on the systemic circulation because the hemangiomas decreased in size after four cycles of ethanol sclerotherapy. Fortunately, our patient quickly recovered without any sequelae.

CONCLUSION

Acute pulmonary vasoconstriction and right ventricular dysfunction should be considered if hemodynamic instability occurs immediately after percutaneous ethanol injection under general anesthesia. Prompt and proper management, including providing supportive treatment, decreasing pulmonary vascular resistance with pulmonary vasodilators, and improving right ventricular function, should be carefully prepared for such cardiopulmonary conditions to minimize the progression of cardiovascular collapse. Hypercarbia, acidosis, and hypothermia, which may predispose pulmonary vasoconstriction, should be corrected and aggressively treated. Pulmonary artery catheterization or transesophageal echocardiography may be helpful if a large volume of ethanol injection is required.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given her consent for her clinical information to be reported in the journal. The patient understands that her name and initials will not be published, and due efforts will be made to conceal identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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