

Anaphylactoid Shock Caused by 5% Human Serum Albumin Transfusion

Takashi Tsunetoh*, Ryu Okutani*,
Mariko Masutani*, Tomoko Koshimichi*

Abstract

A 38-year-old woman underwent bilateral adnexectomy and partial omentectomy procedures for diagnosed ovarian cancer, and developed anaphylactoid shock after administration of 5% human serum albumin (HSA). She had no history of drug or food allergic reactions, nor any previous blood transfusions. Immediately after 5% HSA transfusion, blood pressure dropped to 60/35 mmHg and heart rate increased to 100 bpm. On the basis of her clinical course, we concluded that albumin had induced anaphylactoid shock. Consequently, we discontinued the albumin product transfusion and administered epinephrine, after which the symptoms improved. The concentration of plasma tryptase was remarkably elevated to $64.1 \mu\text{g} \cdot \text{l}^{-1}$ at 1 hour after the occurrence of symptoms.

Key words; anaphylactoid shock, 5% human serum albumin, tryptase

Introduction

When blood pressure (BP) drops as a result of hemorrhaging during surgery, 5% human serum albumin (HSA) is frequently used to restore intravascular blood volume. However, administration of HSA sometimes causes an anaphylaxis/anaphylactoid reaction¹⁾. Herein, we report a patient who developed

severe anaphylactoid shock immediately after transfusion of 5% HSA under general anesthesia.

Case Report

A 38-year-old woman (160 cm, 40 kg) was scheduled to undergo bilateral adnexectomy and partial omentectomy procedures based on a diagnosis of ovarian cancer. She had no history of allergic reactions to either drugs or food, nor any previous blood transfusions. General anesthesia in combination with epidural anesthesia (Th₁₂/L₁) was selected. Anesthesia was induced using fentanyl, propofol, and vecuronium bromide, and maintained with sevoflurane in oxygen and air.

One hour after the start of the operation, blood loss volume was 600 mL and Hb was $7.8 \text{ mg} \cdot \text{dl}^{-1}$ with 21% of Ht. Although 4 units of red blood cells (RBC) were transfused, Hb and Ht decreased to $5.6 \text{ g} \cdot \text{dl}^{-1}$ and 17%, respectively, due to massive blood loss, thus a rapid transfusion of 8 units of RBC was performed. Moreover, because the amount of hemorrhage exceeded 2000 ml, 6 units of fresh frozen plasma (FFP) was transfused for supply of fibrinogen. Following the transfusion, Hb and Ht returned to $12.0 \text{ g} \cdot \text{dl}^{-1}$ and 35%, respectively. Thereafter, 5% HAS (Donated Albumin-wf, Mitsubishi-Tanabe Pharma Corporation, Osaka, Japan) was transfused, as the albumin concentration in plasma had decreased to $1.8 \text{ g} \cdot \text{dl}^{-1}$. Immediately after that transfusion (total amount of 5% HSA; 10 ml), BP dropped to 60/35 mmHg (before transfusion, 100/75 mmHg) and heart rate increased

*Department of Anesthesiology, Osaka City General Hospital, Osaka, Japan

to 100 bpm (before transfusion, 70 bpm). We considered that the hypotension might have been related to hypovolemia, thus we administered norepinephrine (0.1 mg) concomitant with the rapid fluid infusion. However, the patient did not noticeably respond to these treatments and BP decreased further to 40/20 mmHg. At approximately the same time, redness began to appear over the face and precordium, though auscultation did not reveal any wheezing. Since these findings might indicate the occurrence of an anaphylactic reaction to the 5% HSA, the albumin treatment was immediately discontinued and she was administered epinephrine (0.1 mg). After the second administration of epinephrine (0.1 mg), BP rapidly increased and the symptoms started to improve. One hour later, the flushing disappeared and the parameters became stabilized (BP, 80/40 mmHg; heart rate, 95 bpm). Postoperative radiography revealed no marked changes. After it was confirmed that there was no disturbance of consciousness, the tracheal tube was extubated.

The duration of surgery was 5 hours 35 minutes and duration of anesthesia was 7 hours 5 minutes. The total fluid infusion volume was 6700 mL, while blood transfusion volume was 2160 mL, blood loss volume was 2630 mL, and urine output was 200 mL. Plasma tryptase concentration sampled 1 hour after the occurrence of anaphylactoid symptoms was $64.1 \mu\text{g} \cdot \text{l}^{-1}$ (normal range; $2.1\text{--}9.0 \mu\text{g} \cdot \text{l}^{-1}$). The postoperative course was uneventful.

Discussion

In the present patient, treatment with 5% HSA was started to compensate for blood loss during surgery. Immediately after the start of treatment and contrary to our intention, the patient entered a shock state. Redness was noted over the face and precordium, however, there were no abnormal auscultation findings. The absence of increased airway pressure and decreased SpO₂ indicated the absence of bronchospasm. Thus, we suspected anaphylactoid shock caused by administration of 5% HSA based on the clinical course.

The incidence of anaphylaxis/anaphylactoid shock during anesthesia was reported to be 1 in every 3500 to 20,000 occasions and those reactions are most frequently related to administration of muscle relaxants¹⁾. HSA contains small amounts of hypotension-inducing factors, such as prekallikrein activating factor and bradykinin^{2,3)}, and reactions occur at an extremely low frequency (0.1%)¹⁾. However, there are some reports of anaphylaxis/anaphylactoid shock caused by 5% HSA products^{4~7)}.

When anaphylaxis is suspected, measurement of plasma tryptase level is useful. Tryptase is an enzyme stored in mast cells, which is subsequently released upon mast cell activation and degranulation. It remains stable longer than histamine and is extremely useful for the diagnosis of anaphylaxis. The best cutoff point for plasma tryptase level is considered to be $8.23 \mu\text{g} \cdot \text{l}^{-1}$ ^{6~9)}. We did not measure serum haptoglobin in the present patient. However, it has been reported that severe anaphylaxis may occur after albumin transfusion in patients with ahaptoglobinemia^{4,5)}. Since the incidence of ahaptoglobinemia in Japan has been reported to be approximately 1 in 4000^{4,6,8)}, we should have suspected the presence of these factors when severe anaphylaxis following albumin or blood transfusion was revealed.

In general, the first-choice agent for treating anaphylaxis is epinephrine, with H₁ and H₂-receptor blockers, and steroids added as appropriate. Epinephrine increases cAMP stored in mast cells and basophils, and inhibits the release of inflammatory mediators from those cells¹⁾. Most importantly, since it is impossible to foresee and prevent drug-induced anaphylaxis, it is important to remain aware of its possible occurrence, and provide appropriate and prompt treatments when it appears.

Conclusion

We treated a patient who developed severe hypotension due to anaphylactoid shock immediately after beginning transfusion with 5% HSA. Although an anaphylaxis/anaphylactoid reaction to albumin products occurs at an extremely low frequency, great

caution regarding such a reaction is essential during their administration.

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