Anaphylactoid Reaction Associated with Blood Transfusion during Anesthesia

– A Case of Characteristic Hemodynamic Changes –

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There are many clinical reports concerning the hemodynamic characteristic changes in anaphylactoid reaction associated with blood transfusion during anesthesia[1–5]. Omote et al.[2] described a patient whose blood pressure could not be detected by the cuff technic and whose carotid pulse was not palpable. Ogino et al.[3] experienced a case in which cardiac arrest occurred. The data of Mitsuhata et al.[5] showed that hypotension was primarily caused by peripheral vasodilation and acute reduction in intravascular fluid volume. We also recently observed a patient who had an anaphylactoid reaction associated with blood transfusion during anesthesia, which presented very characteristic hemodynamic changes. These hemodynamic changes are as follows: the radial arteries and the dorsalis pedis pulses were not palpable, whereas the carotid and femoral pulses were palpable. These are seldom observed in anaphylactoid reaction, so we report this case in detail.

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Case Report

An 80-year-old male, with a past history of pharyngo-laryngectomy for hypopharyngeal carcinoma with a blood transfusion eight months before, underwent tumorectomy for recurrence of hypopharyngeal cancer. He had no history of allergic disease. The abnormal laboratory findings were: serum potassium, 5.7 $mEq \cdot l^{-1}$; GPT, 94 $IU \cdot l^{-1}$; GOT, 62 $IU \cdot l^{-1}$; and BUN, 34 $mg \cdot dl^{-1}$. It was thought that administration of antibiotics before the operation had caused the renal and liver dysfunction.

He weighed 42 kg and was 154 cm tall. He was premedicated with atropine sulfate, 0.4 mg, intramuscularly 30 min before anesthesia induction. A spiral intratracheal tube was placed in the trachea through the tracheostoma after sedation with fentanyl, 0.2 mg intravenously. A Teflon catheter (20 G) was inserted into the dorsal artery of the left foot for drawing blood specimens and directly measuring arterial blood pressure. The course of anesthesia is shown in figure 1.

One hour and twenty-five minutes after the start of the operation, a blood transfusion was required because of moderate bleeding. The hematocrit

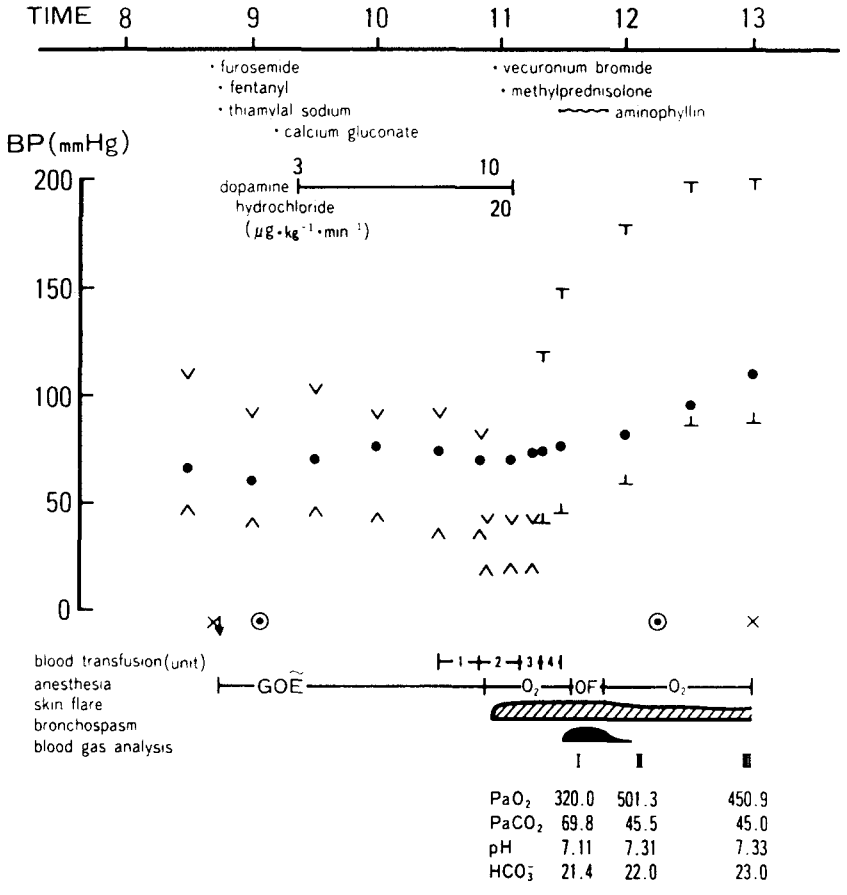

Fig. 1.

was 26.3% at the time. Twenty minutes later, transfusion of the 2nd pack of stored whole blood was started. A few minutes later, the directly measured arterial blood pressure suddenly dropped to 40/25 mmHg. The blood pressure was not detectable in the right upper arm using the cuff technic. The contralateral dorsalis pedis pulse and the bilateral radial pulses were not palpable. The skin was cyanotic at the peripheral extremities. It was considered at the time that the cause must have been massive bleeding, so inhalation of nitrous oxide and enflurane was discontinued and ventilation with 100% O_2 was started. The whole blood transfusion and the lactate Ringer's solution infusion were accelerated, and a dopamine infusion at 20 $\mu g \cdot kg^{-1} \cdot min^{-1}$ was added. However, the directly measued arterial blood pressure did not increase.

The femoral and carotid pulses were both very strong. Since the patient had a skin flare and because of the timing of symptoms, an anaphylactoid reaction associated with the blood transfusion was strongly susptected. A catheter was inserted into the strongly pulsative femoral artery and a direct arterial blood pressure of 125/45 mmHg was measured. Afterward, halothane inhalation was started with administration of aminophylline (0.6 $mg \cdot kg^{-1} \cdot min^{-1}$; total 250 mg) and methylprednisolone (2,000 mg), since bronchospasm had been induced.

Neither hematuria nor hemoglobinuria were observed.

The operation was shortened and ended one hour and twenty-five minutes after the anaphylactoid reaction had occurred. Since the patient had slight hypercapnia though sufficient spontaneous ventilation, he was admitted to the intensive care unit with CPAP respiration. A few hours after admission his blood gas data improved markedly. Next morning, he was extubated and moved to the general ward.

The blood type of this patient was B Rh(+), and no irregular red blood cell antibodies were found in his serum either before or after the operation. No agglutination was detected in a cross matching test between his blood and the administered whole blood. Antibodies against either white blood cells or platelets were not found in his serum after the operation. His postoperative IgE level (1,300 $IU \cdot ml^{-1}$) was much higher than the standard value (below 350 $IU \cdot ml^{-1}$).

Discussion

Since some skin symptoms occur in anaphylactoid reactions[6], we were convinced that an anaphylactoid reaction had occurred as soon as we saw the skin flare. It is thought that if an antigen causing an anaphylactoid reaction is administered intravenously, it causes a reaction within about five minutes. So the time-course suggested the cause of this reaction was the blood transfusion. Also, subsequent bronchospasm made the diagnosis of anaphylaxis certain.

The circulatory changes in this case report was very characteristic, considering the dissociation of pulsation between the central and peripheral arteries. The direct arterial blood pressure monitored in the dorsal pedis artery of the left foot decreased, and the blood pressure could also not be measured manually at the right upper arm. As well, the right dorsalis pedis pulse and the bilateral radial pulses could not be felt. However, the femoral and carotid arteries were pulsating strongly.

Smedegård et al.[7] induced aggregate anaphylaxis in an ovalbumin-sensitized monkey and observed that decrease of blood flow was most marked in the splanchnic organs (kidney, pancreas and spleen), and somewhat smaller in skeletal muscle, where it paralleled the decrease in mean arterial pressure. A redistribution of blood flow to vital organs (brain, heart and liver) therefore occurred.

Tonosaki et al.[8] induced anaphylactic shock in ovalbumin-sensitized mongrel dogs and studied the organ blood flows. Organ blood flows through the superior mesenteric, renal, common carotid and femoral arteries were decreased markedly just after the induction of anaphylactic shock; the blood flows through the superior mesenteric and renal arteries then gradually recovered, but the blood flows through the common carotid and femoral arteries kept decreasing. The organ blood flow distribution rate (organ blood flow/cardiac output) changed in a rather complicated fashion after induction: the rate in both the superior mesenteric and renal arteries increased slightly following a transient decrease, the rate in the common carotid artery decreased gradually following a transient increase, and the rate in the femoral artery increased markedly just after induction. Tonosaki et al. considered that blood centralization seemed to occur transiently just after induction in order to maintain the cerebral blood flow, by decreasing the rate of flow through the mesenteric artery. But actually, centralization was not clearly seen, contrary to their expectations, since the rate in the common carotid artery decreased gradually whereas the rate in the femoral artery increased as the shock ensued.

The radial and dorsalis pedis arteries were not pulsatile in our case, whereas the carotid and femoral arteries were. This suggests a protective reaction for central organs such as the heart and brain during anaphylactoid reaction. The circulatory changes in the present case were possibly reflections of such a response.

In anaphylactic shock, even if the same manner of sensitization is adopted, when the individual or animal species differs, a much wider variety of reactions occur, compared to in endotoxic or hypovolemic shock[8,9]. Pavek[10] reported that monkeys with severe anaphyalactic shock tended to have higher systemic resistance than monkeys with mild anaphylactic shock. Also, Smedegård et al.[7] observed that total peripheral resistance increased 1.5 times in anaphylactic shock in the monkey. In humans, there are some reports that systemic vascular resistance decreases[11–13] or increases[14,15]. Hanashiro et al.[14] commented that in anaphylactic shock in man, peripheral arterial resistance increased, hence there was evidence of a decrease in the effective volume available for circulation and compensatory arterial vasoconstriction. In our case, the probable reason why the radial and dorsalis pedis arteries pulses were not pulsatile whereas the carotid and femoral ones were is that marked compensatory arterial vasoconstiriction occurred, as suggested by Hanashiro et al., and apparently the shock was mild.

In summary, our patient had an anaphaylactoid reaction associated with blood transfusion during anesthesia. He showed the following characteristic hemodynamic changes: the radial and dorsal pedis artery pulses were not palpable, whereas the carotid and femoral pulses were. This apparently indicates that marked compensatory arterial vasoconstriction occurred to sustain perfusion in the central organs. These characteristic hemodynamic changes occurred when the shock level was considered to be mild.

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