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Case Report
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Anaphylactic Shock Complicated with Hematuria and Deep Venous Thrombosis: A Case Report

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Abstract

Background: Anaphylactic shock is a common clinical emergency that shares mediators involved in the coagulation cascade and can also damage multiple systems, such as the kidneys, resulting in deep venous thrombosis and hematuria. To date, only one case of venous thrombotic events following anaphylaxis has been reported. Herein, we describe the first case of hematuria and deep venous thrombosis occurring in close temporal relation to an anaphylactic event.

Case Presentation: A 74-year-old female (153 cm, 51.5 kg) was diagnosed with, 1. Cervical spinal stenosis; 2. Cervical disc herniation at levels C3/4, C4/5, C5/6, and C6/7; 3. Spinal cord and nerve compression; 4. Chronic inflammation of both lungs; 5. Lacunar cerebral infarction; 6. Brain atrophy. She was scheduled to undergo posterior C3–7 single open-door spinal canal enlargement, laminoplasty, and mini-plate screw fixation. After anesthesia induction, the patient experienced increased airway pressure and urticaria on the anterior chest, followed by anaphylactic shock. After fluid resuscitation, corticosteroids, calcium, and adrenaline administration, the patient developed hematuria. Subsequently, 250 mL of 5% sodium bicarbonate was administered to alkalinize the urine, and blood pressure was continuously maintained. The surgery was suspended after consultation with the chief surgeon and the patient's family. Once vital signs stabilized, the endotracheal tube was removed, and the patient was safely returned to the ward. On the third postoperative day, vascular color Doppler ultrasound revealed multiple thrombi throughout the body.

Conclusion: Anaphylactic shock causes decreased renal perfusion, ischemia of the renal cortex, and damage to renal tubular epithelial cells. Immune complex deposition can lead to complement activation and subsequent intravascular injury, resulting in hematuria. The coagulation cascade shares many inflammatory mediators with conditions such as sepsis, shock, asthma, allergy, and anaphylaxis. While all these conditions, except anaphylaxis, have been linked to an increased risk of thrombosis and blood clot formation, our findings suggest that anaphylaxis may also be a contributing factor to deep vein thrombosis.

Keywords

Anaphylactic Shock, Deep Vein Thrombosis, Hematuria, Tryptase, Systemic Inflammation

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Introduction

Anaphylactic shock caused by drugs, food, or insect bites can involve multiple organ systems. Studies show that approximately 0.26% of patients are hospitalized due to allergic reactions, and there are 1–3 deaths per million people per year [1]. Interestingly, the majority of perioperative anaphylactic reactions occur during or shortly after the induction of anesthesia [2]. Hematuria and venous thrombotic events following anaphylaxis have been reported separately [3,4], but data regarding their simultaneous occurrence is scarce, and no previous case has documented both symptoms in the same clinical course.

Case Presentation

A 74-year-old female was scheduled for cervical spine surgery due to numbness and discomfort in the left upper limb. For anesthesia induction, sufentanil 20 μg, cis-atracurium 12 mg, and propofol 30 mg were administered. After the patient lost consciousness, airway pressure increased to 30 cmH_2O, tidal volume dropped to 100 mL, and urticaria with raised skin appeared on the chest. Blood pressure decreased, and the rash extended to both legs and upper arms (**Fig-1**). Blood samples were collected for tryptase measurement, and epinephrine along with other medications were administered. Following treatment, the patient developed hematuria. The surgery was suspended after consultation with the chief surgeon and the patient's family.

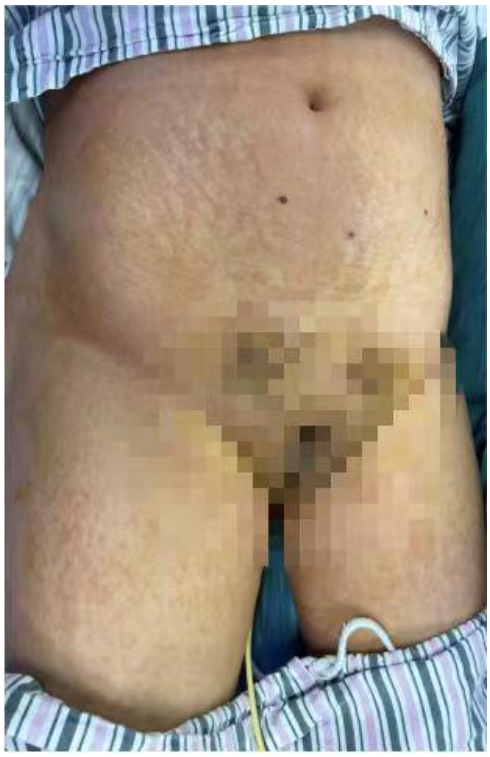

Fig-1: Urticaria

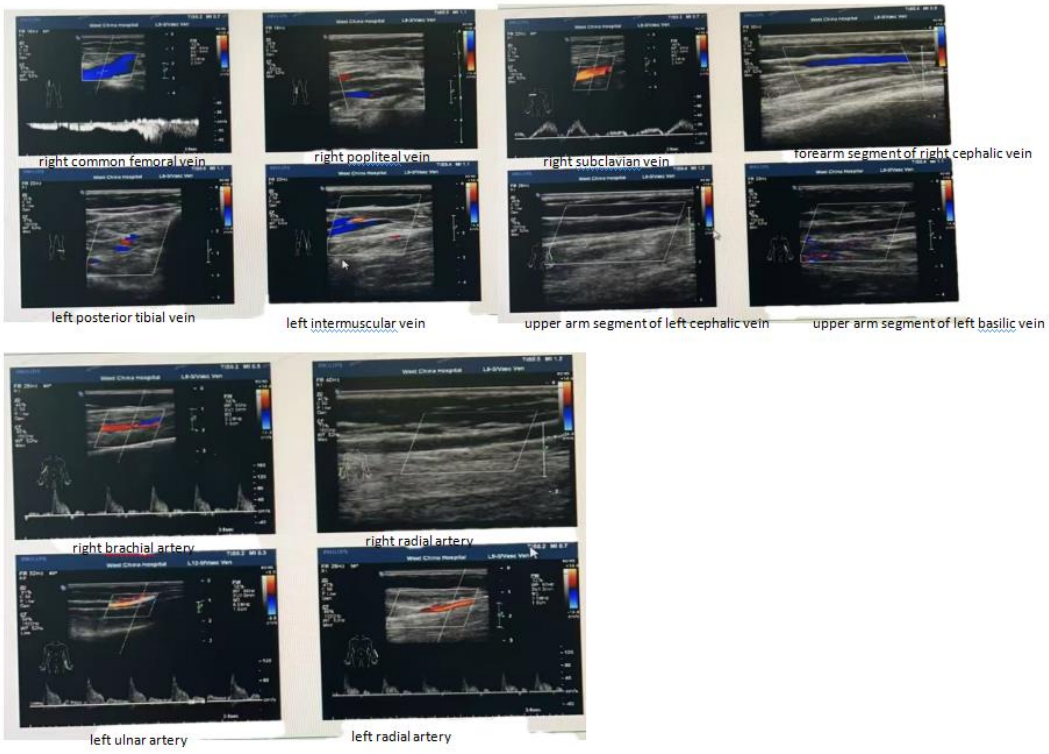

Fig-2: Intravenous Color Doppler Ultrasound on the Day After Anaphylactic Shock

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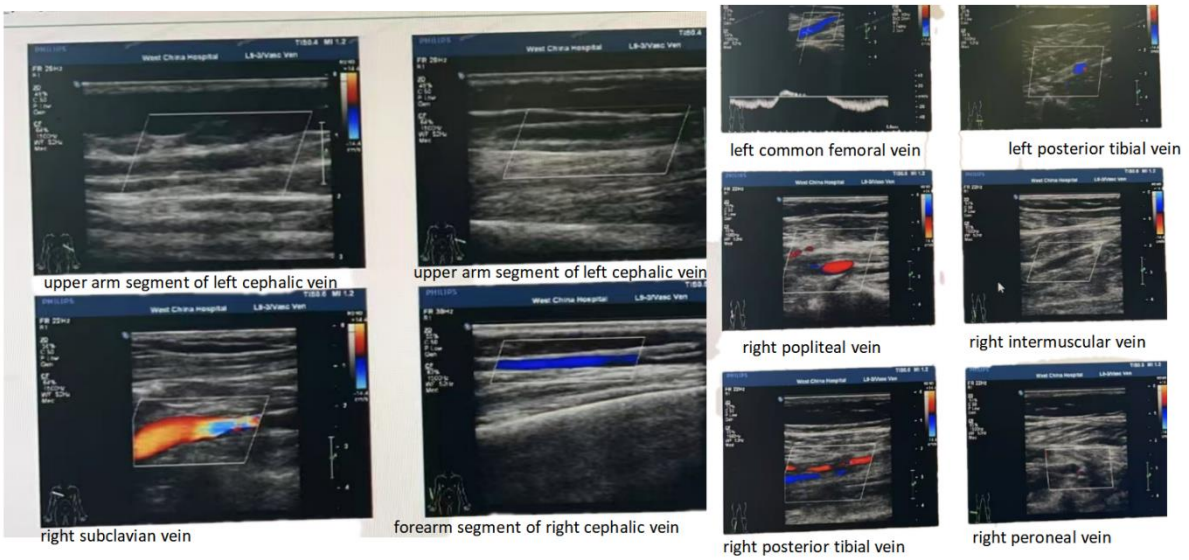

Fig-3: Intravenous Color Doppler Ultrasound on the Second Day After Anaphylactic Shock

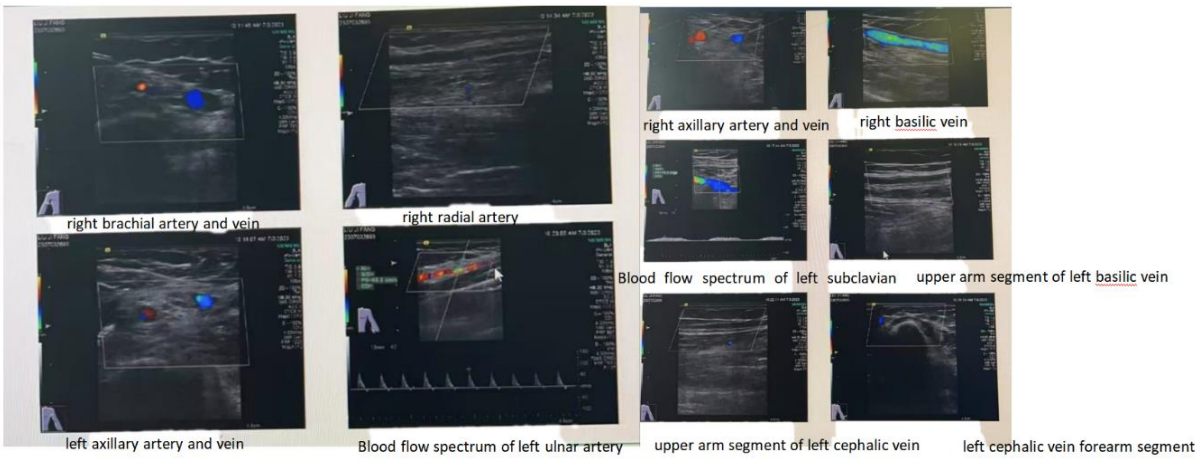

Fig-4: Intravenous Color Doppler Ultrasound on the Sixth Day After Anaphylactic Shock

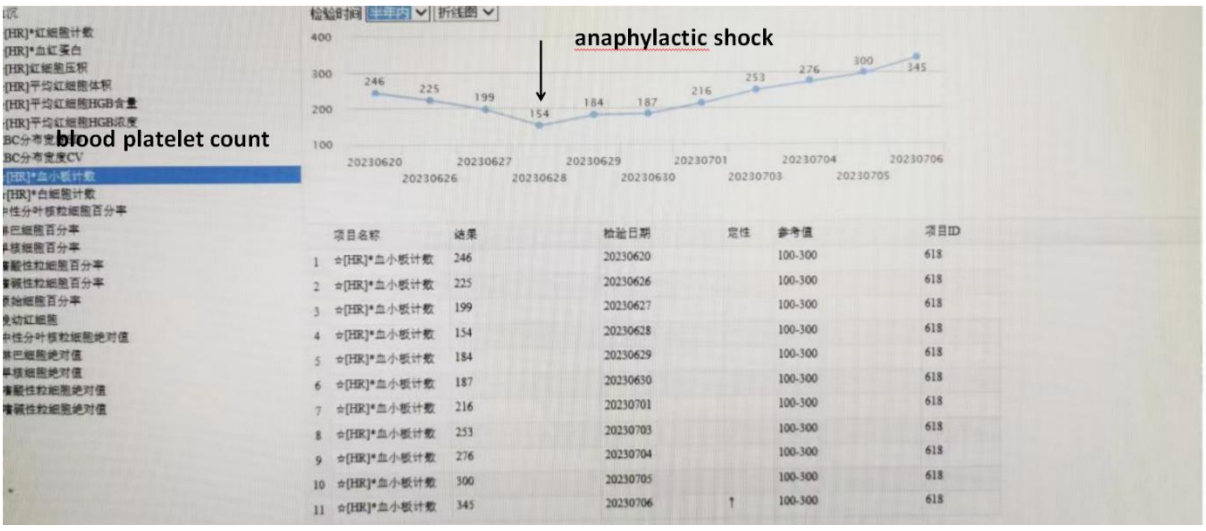

Fig-5: Platelet Changes Before and After Anaphylactic Shock

On postoperative Day 1, vascular color Doppler ultrasound of the limbs revealed thrombosis in the right popliteal vein and posterior tibial vein, thrombosis in the right radial artery, and thrombosis in the upper arm segment of both the left cephalic and left basilic veins (**Fig-2**). Anticoagulation therapy was initiated after consultation with the vascular surgery team. On postoperative Day 2, thrombosis was again observed in the left cephalic and basilic veins, as well as in the right popliteal vein, posterior tibial vein, peroneal vein, and several intermuscular veins (**Fig-3**). On postoperative Day 6, thrombosis was noted in the upper arm segment of the left basilic and cephalic veins, and in the right radial artery (**Fig-4**). Platelet consumption was observed to be consistent with the ongoing thrombotic process (**Fig-5**).

The patient was treated with low molecular weight heparin and diosmin. Investigations ruled out both genetic and acquired prothrombotic states, with the exception of suspected scratch syndrome and neoplasm. At 2-year follow-up, the patient had fully recovered. The tryptase level was 21.7 μg/L within 2 hours following successful resuscitation. The baseline tryptase value measured 24 hours after the reaction was 3.18 μg/L. The intraoperative elevation of tryptase confirmed that the patient experienced a severe allergic reaction. Based on her clinical history, skin tests for cis-atracurium, propofol, sufentanil, and chlorhexidine were negative. However, approximately 20% of severe allergic reactions occurring during the perioperative period cannot be detected by skin testing, and due to the pharmacologic nature of general anesthesia, drug provocation testing could not be performed for further diagnosis.

Discussion

Previous literature has reported that allergic reactions are often associated with coronary thrombosis [5-7], cerebral venous thrombosis [8], and pulmonary embolism [9]. However, few studies have reported a correlation between allergic reactions and deep vein thrombosis (DVT) [3], particularly in cases where hematuria and DVT occur simultaneously, as seen in this patient. Tryptase testing in our case confirmed that the patient experienced a severe allergic reaction. Unfortunately, no specific allergen could be identified. Approximately 20% of muscle relaxant-induced mast cell degranulation is mediated via MRGPRX2 receptors, which cannot be detected by conventional skin testing [10]. Although this patient had two negative intradermal skin tests for muscle relaxants, it remains possible that she was allergic to the muscle relaxant cis-atracurium.

This case strongly suggests that the allergic response triggered the release of preformed mediators from mast cells and basophils (e.g., histamine, tryptase) as well as newly synthesized mediators (e.g., glucagon, leukotrienes, platelet-activating factor). These mediators contribute to systemic responses, including immune complex deposition, activation of the complement system, intravascular injury leading to hematuria, and activation of platelet-activating factor, which in turn promotes platelet activation and aggregation. This cascade leads to the expression and release of tissue factor and plasminogen activator inhibitor-1 into the circulation [11].

Anaphylaxis is a life-threatening systemic inflammatory process that shares several mediators with the coagulation cascade pathway. The inflammatory mediators involved in sepsis, shock, asthma, and anaphylaxis overlap significantly with those that drive coagulation. This indicates a potential epidemiological link between allergic diseases and venous thrombosis. Such a connection opens the door for future studies to evaluate whether the prevention or treatment of allergic diseases might reduce the risk of thrombotic events.

Given these associations, systemic thrombosis screening following anaphylactic shock is necessary to prevent more severe complications. Early identification and management of thrombotic events in this context may significantly improve patient outcomes.

Conflict of Interest

The author has read and approved the final version of the manuscript. The author has no conflicts of interest to declare.

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