

When Thrombosis and Hemorrhage Coexist: A Case of Pediatric Septic Pulmonary Embolism

Tromboz ve Kanama Birlikte Görüldüğünde: Pediatrik Septik Pulmoner Emboli Vakası

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A previously healthy 10-year-old girl presented with pain and swelling of the right knee, fever, and progressive respiratory distress three days after falling from a tractor trailer onto her knee. On admission, she was confused and hemodynamically unstable, requiring pediatric intensive care. Vital signs were as follows: heart rate, 160/min; blood pressure, 90/50 mmHg; respiratory rate, 30/min; temperature, 38°C; and oxygen saturation, 91–92% on room air. Examination revealed warmth, erythema, and tenderness over the anterior knee and proximal cruris, without signs of septic arthritis.

Pathological laboratory findings showed leukopenia (WBC, $3.24 \times 10^3/\mu\text{L}$), elevated CRP (418.8 mg/L), procalcitonin (100 ng/mL), AST (200 U/L), and ALT (110 U/L), and hypoxemia (pO_2 , 59.2 mmHg). Coagulation parameters showed a PT of 15.2 seconds, INR of 1.36, aPTT of 33.7 seconds, and fibrinogen of 3.7 g/L. Ferritin levels progressively increased from 1,370 ng/mL to 22,938 ng/mL during hospitalization. With a diagnosis of septic shock, empirical vancomycin and meropenem were initiated, along with inotropic support and intravenous immunoglobulin as immunomodulatory therapy (1 g/kg).

Five days after IVIG therapy, ultrasonography revealed a long-segment thrombus involving the right great saphenous vein, with concomitant popliteal and distal deep vein thrombosis. Chest computed tomography demonstrated bilateral patchy pulmonary infiltrates with microcavitations and no evidence of central pulmonary embolism, findings suggestive of septic pulmonary embolism (SPE) (Figure 1). Methicillin-resistant *Staphylococcus aureus* (MRSA) was isolated from blood cultures, the central venous catheter tip culture, and wound cultures. Low-molecular-weight heparin (LMWH) was initiated at a dose of 100 IU/kg twice daily for anticoagulation.

Despite antimicrobial, anticoagulant, and supportive therapies, the patient developed massive pulmonary hemorrhage on the second day of anticoagulant therapy (day 9 of hospitalization) and subsequently died of multiorgan failure. On the day of death, the hemoglobin level was 6.5 g/dL, and the platelet count was $79 \times 10^3/\mu\text{L}$. Unfortunately, coagulation parameters could not be obtained at the time of clinical deterioration, which represents a limitation of this case report.

Septic pulmonary embolism (SPE) is a rare but potentially fatal condition in children that results from the hematogenous dissemination of infected thrombi from extrapulmonary infectious foci. Clinical manifestations are often nonspecific and may include fever and respiratory distress (1,2). Our case highlights a rare presentation of SPE complicated by deep vein thrombosis following minor trauma and subsequent soft tissue infection.

Although macrophage activation syndrome (MAS) and hemophagocytic lymphohistiocytosis (HLH) could not be formally diagnosed because the patient did not meet the established diagnostic criteria, the markedly elevated ferritin level suggests a profound hyperinflammatory state. This hyperinflammatory state may have contributed to endothelial injury, coagulation abnormalities, and subsequent multiorgan dysfunction.

The role of anticoagulation in SPE remains uncertain. While anticoagulation may prevent thrombus progression and further embolization, its use is challenging due to the potential risk of hemorrhage in infected and fragile pulmonary vasculature. Current evidence is limited, particularly in children, and management decisions should be individualized based on thrombotic burden, infection severity, pulmonary involvement, and bleeding risk (3–5).

A limitation of this report is the absence of coagulation parameters at the time of pulmonary hemorrhage. Although anticoagulation may have led to bleeding risk, the fatal hemorrhage was most likely multifactorial, resulting from the combined effects of severe MRSA infection, septic pulmonary embolism, pulmonary vascular destruction, hyperinflammation, sepsis-associated coagulation abnormalities, and anticoagulant therapy. This case emphasizes the need for further studies to define optimal anticoagulation strategies in pediatric SPE complicated by venous thrombosis.

Keywords: pediatric, septic emboli, pulmonary embolism

Anahtar Kelimeler: pediatri, septik emboli, pulmoner emboli

The patient's family provided informed consent.

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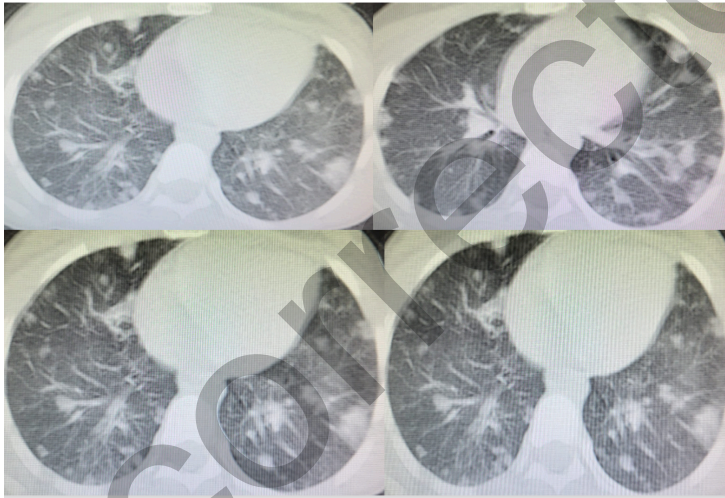


Figure I: CT images from different sections demonstrate wedge-shaped peripheral lesions, nodular lesions, and areas of microcavitation