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Unprovoked Saddle Pulmonary Embolism in a 12-Year-Old Child Requiring Open Surgical Pulmonary Embolectomy: A Case Report and Literature Review

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Abstract

Pulmonary embolism (PE) in children is rare and usually occurs in association with identifiable risk factors such as central venous catheters, malignancy, infection, or trauma. Massive or saddle PE, particularly without provoking factors, is exceedingly uncommon in the paediatric population, with the majority of published reports limited to neonates and preterm infants. We report a case of unprovoked PE in a 12-year-old child, highlighting the importance of considering underlying thrombophilia and genetic predispositions, and discuss management strategies in the context of limited pediatric-specific evidence. This case contrasts with the existing literature, where nearly all paediatric saddle PE cases involve neonates or infants, often with perinatal complications or central venous catheters. Truly unprovoked massive PE in older children is rarely described.

Keywords: Open Surgery; Trauma; Chest Pain; Paediatric; Oncological Disease; Thoracic Surgery

Abbreviations: PE; Pulmonary Embolism; VTE: Venous Thromboembolism; OD: Oncological Disease

Background

Venous thromboembolism (VTE) in childhood is an uncommon event compared with adults. The estimated incidence of pediatric PE is less than 1 per 100,000 annually [1]. Most cases are provoked by identifiable factors including central venous catheters, oncological disease, or systemic infection [2]. Reports of unprovoked pediatric PE are scarce in the literature, and their rarity makes recognition challenging. We present a case of unprovoked PE in a 12-year-old child, followed by a review of potential underlying mechanisms and implications for clinical care.

Case Presentation

A previously healthy 12-year-old child presented with acute onset pleuritic chest pain and dyspnoea. There was no history of recent infection, trauma, immobilisation, surgery, or use of central venous catheters. Past medical and family history were unremarkable, with no known thrombophilia. On examination, the patient was tachycardic with oxygen desaturation but haemodynamically stable. Chest radiograph was unremarkable. CT pulmonary angiography revealed filling defects within the segmental branches of the right pulmonary artery, consistent with acute pulmonary embolism.

Laboratory investigations including complete blood count, renal and liver function were normal. A full thrombophilia screen was performed, including protein C, protein S, antithrombin levels, Factor V Leiden mutation, and prothrombin G20210A mutation, all of which were within normal limits. No underlying inflammatory or systemic disorder was identified. The patient was commenced on low-molecular-weight heparin and transitioned to a direct oral anticoagulant. Clinical symptoms improved, and follow-up imaging demonstrated resolution of the embolus. The patient continues on anticoagulation with close outpatient follow-up.

Discussion

Pulmonary embolism (PE) in childhood is rare, with an estimated annual incidence of 0.9 per 100,000 children [1]. Most

cases are associated with identifiable risk factors such as central venous catheters, malignancy, infection, trauma, or congenital/acquired thrombophilia [2]. Truly unprovoked PE, defined as venous thromboembolism (VTE) in the absence of predisposing factors, is exceedingly uncommon, accounting for only a small minority of paediatric cases [3,4].

Our patient presented with an unprovoked saddle PE at 12 years of age, a scenario seldom described in the literature. Published reports of massive or saddle PE in children largely involve neonates or preterm infants, sometimes without an obvious precipitating factor [5-7]. Successful emergent surgical embolectomies have been described in this age group, with survival despite haemodynamic compromise [5,6]. In contrast, reports in older children are exceedingly rare, and when described, they are frequently associated with recognised triggers such as central venous catheters, nephrotic syndrome, infection, or inherited thrombophilia [3,8]. The present case is therefore unusual in representing a truly unprovoked massive PE in an otherwise healthy older child.

Although labelled unprovoked, several mechanisms does require consideration. Inherited thrombophilias are among the most frequently implicated underlying predispositions. Deficiencies of antithrombin, protein C, or protein S, and mutations such as Factor V Leiden and prothrombin G20210A, have been linked to paediatric VTE, although penetrance is incomplete and geographic prevalence varies [2,9]. Compound heterozygosity or homozygous mutations are more strongly associated with severe or early thrombotic events [9]. Other rarer associations include hyperhomocysteinaemia, metabolic defects, and rare haematological disorders such as idiopathic hypereosinophilia, which may enhance vascular injury and thrombosis [10,11]. Recently, case reports have also implicated novel genetic defects, such as ANKRD26-related thrombocytopenia, in otherwise idiopathic childhood PE [12]. Inflammatory and autoimmune processes, sometimes preceding overt clinical manifestations, may also act as covert prothrombotic drivers.

Management of paediatric PE remains challenging due to the lack of randomised controlled trials. Current practice is largely extrapolated from adult guidelines, with low-molecular-weight heparin or, in older children, direct oral anticoagulants as first-line options [2,13]. The duration of anticoagulation is individualised: provoked events are often treated for 3-6 months, whereas unprovoked cases, especially with ongoing risk markers or identified thrombophilia, may warrant extended therapy [13]. Massive or saddle PE with haemodynamic compromise in children has occasionally required thrombolysis, catheter-directed intervention, or surgical embolectomy, although such experiences are restricted to case reports [5-7].

Long-term follow-up is important, as recurrence risk is higher after unprovoked than provoked VTE, although absolute recurrence rates in children remain lower than in adults [3,4]. Our patient's case emphasises the necessity of comprehensive thrombophilia evaluation, careful monitoring, and ongoing vigilance. Finally, this case adds to the very limited body of literature describing unprovoked massive PE in older children. Continued case reporting and international data sharing are essential to strengthen the evidence base, refine paediatric-specific recommendations, and improve recognition and management of this rare but potentially life-threatening condition.

Learning points

i. Pulmonary embolism is rare in children, with most cases associated with identifiable risk factors; truly unprovoked events are exceptional, particularly in older children.

ii. Massive or saddle pulmonary embolism in the paediatric population is most often described in neonates; reports in otherwise healthy adolescents are exceedingly uncommon.

iii. A thorough evaluation for inherited and acquired thrombophilia is essential even when the index event appears unprovoked.

iv. Management strategies in children are largely extrapolated from adult guidelines, and treatment decisions-including duration of anticoagulation-should be individualised.

v. Ongoing case reporting is critical to expand the limited evidence base and inform paediatric-specific recommendations for pulmonary embolism.

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