DOI:10.24938/kutfd.792374 Kırıkkale Üniversitesi Tıp Fakültesi Dergisi 2020;22(3):478-482
Case Report *Olgu Sunumu*

PULMONARY THROMBOEMBOLISM IN A CHILD AFTER BURN INJURY

Yanık Sonrası Pulmoner Tromboemboli Gelişen Bir Çocuk Olgu

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ABSTRACT

Pulmonary embolism is rarely diagnosed in pediatric burn patients and is often only discovered at autopsy. Herein we would like to present the case of a pediatric patient with pulmonary thromboembolism who was burnt by hot water at home. A 4-year-old girl who was treated at an external center for 9 days due to second degree burn injuries in both legs. After the discharge she had suddenly become restless and than gone unconscious. Since pulmonary thromboembolism was suspected as the cause of cardiopulmonary arrest, a pulmonary computed tomography angiogram was taken, which showed a thrombus in the lobar and segmental branches of the right pulmonary artery supplying the right lower lobe. Enoxaparin sodium treatment (2×1mg/kg) was initiated. At day 16, a control computed tomography angiogram showed that the thrombus in the lobar and segmental branches of the pulmonary artery had disappeared. This case report illustrates the importance of chemoprophylaxis against pulmonary embolism in both pediatric and adult burn patients. In such cases, especially the severe ones, a sudden deterioration of patient's clinical status in the form of respiratory distress or failure should raise suspicion about the possibility of pulmonary embolism.

***Keywords**: Burn, pulmonary thromboembolism, pediatric, heparin*

ÖZ

Pulmoner tromboemboli, pediatrik yaş grubunda nadiren tanısı konulan bir hastalıktır ve tanı genellikle otopsi ile konulur. Bu çalışmada evde, sıcak su teması sebebiyle oluşan yanıktan sonra pulmoner tromboemboli gelişen bir çocuk olgusu sunulmuştur. İki bacakta ikinci derece yanık yaralanması nedeniyle 9 gün boyunca dış merkezde tedavi görmekte iken taburculuk günü, aniden solunum sıkıntısı ve bilinç kaybı ile kardiyopulmoner arrest gelişen 4 yaşındaki kız çocuğu yoğun bakım ünitemize kabul edildi. Kardiyopulmoner arrest nedeni olarak pulmoner emboli düşünülmesi üzerine hastaya akciğer bilgisayarlı tomografi ile anjiografi yapıldı. Sağ pulmoner arterin lober ve segmental dallarında trombüs saptandı. İntravenöz anfraksiyone düşük molekül ağırlıklı heparin (Enoksaparin sodyum 2×1mg / kg) tedavisine başlandı. On altıncı günde yapılan kontrol bilgisayarlı tomografi anjiyografisinde, pulmoner arterin lober ve segmental dallarındaki trombüsün kaybolduğu saptandı. Yanık hastalarında solunum sıkıntısı ile kardiyopulmoner arrest gelişmesi halinde ayırıcı tanıda pulmoner emboli mutlaka düşünülmelidir. Bu olgu sunumu hem çocuk hem de yetişkin yanık hastalarında pulmoner emboli olasılığına karşı kemoprofilaksinin önemini göstermektedir.

***Anahtar Kelimeler**: Yanık, pulmoner tromboemboli, çocuk, heparin*

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Received / Geliş Tarihi: 09.09.2020 **Accepted** / Kabul Tarihi: 21.12.2020
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KÜ Tıp Fak Derg 2020;22(3):478-482
Doi: 10.24938/kutfd.792374

INTRODUCTION

Pulmonary thromboembolism (PTE) is rarely diagnosed in pediatric burn patients and is often only discovered during autopsy. Multiple risk factors can be found other than burn, which include hemoptysis, chest pain, dehydration, septicemia, central venous lines, trauma, surgery, ongoing hemolysis, vascular lesions, malignancy, renal disease, foreign bodies, and nonbacterial thrombotic endocarditis (1). Many retrospective studies have reported incidences as low as 0 to 0.4% for thromboembolic complications including symptomatic PTE among burn patients (2). Burn patients exhibit an acquired hypercoagulable state with increased risk of venous thromboembolism (2-4). Herein we would like to present the case of a pediatric patient with PTE who was injured by hot water at home and was transported to our special burn unit.

CASE REPORT

A 4-years-old girl was brought with cardiopulmonary arrest. She had been hospitalized at another hospital for 9 days for secondary degree burn injuries in both legs (Figure 1). The total body surface area burnt was 15%. At the day of discharge, she had suddenly become restless and then gone unconscious. Her past medical history was unremarkable. There was no parental consanguinity, nor there was any family member with a history of coagulopathy or stroke. The patient was in a comatose state upon presentation, with a Glasgow coma scale score of E1M1V1 (4). Her blood pressure was 60/30 mmHg, heart rate 100/min, body temperature <36 °C, and SpO2 98% on room air. Her BMI weight was 25 kg/m2 (overweight). Her pupils were fixed and dilated. She was intubated and ventilated with a mechanical ventilator.

Laboratory results were as follows: hemoglobin 13.5 g/dl, white blood cell count 19.700/mm3, platelet count 553.000/mm3; blood urea nitrogen 16 mg/dl, creatinine 0.4 mg/dl, AST 49 U/L, ALT 56 U/L, total bilirubin 0.38 mg/dl, creatinine phosphokinase (U/L) 212 (39-308), albumin 4.7 mg/dl, APTT 25.2 seconds, INR 1.02, cholesterol 214 mg/dl (<170), HDL cholesterol 17 mg/dl (45-65), triglycerides 274 mg/dl (30-100), free T4 0.735 ng/dl (0.8-2.2), TSH 1.32 mIU/ml (0.7-6.4), sedimentation rate 60 mmh/hr, and D-dimer 3429 µg/L (80-500). The echocardiographic examination was normal. Pulmonary computed tomography angiogram showed thrombus in the lobar and segmental branches of the right pulmonary artery supplying the right lower lobe. The left main bronchus was obstructed. The left lung was almost totally collapsed (Figure 2a,b). The laboratory results of thrombosis markers were as follows: homocysteine 5,56 µmol/L(3.3-8.3), protein C level 4.03 mg/L (N), protein C activity 138.5% (70-140), protein S activity 67.9% (60-130), antiphospholipid antibody Ig M 1.68 U/ml (<12), Ig G 0.23 U/ml (<12), negative ANA, and negative Anti DNA. We did not perform embolectomy because of the hemodynamic stability and good oxygenation of the patient. Enoxaparin sodium treatment (2×1mg/kg) was initiated. Since there was pneumonic infiltration on the right lung, wide spectrum antibiotics were administered. At day 16, a control pulmonary CT angiogram showed that the thrombus in the lobar and segmental branches of the pulmonary artery had disappeared and that branch was recanalized (Figure 2c,d). During follow-up, she suffered persistent multiorgan failure secondary to respiratory arrest with no apparent improvement. At day 20, she sustained a cardiopulmonary arrest and died.

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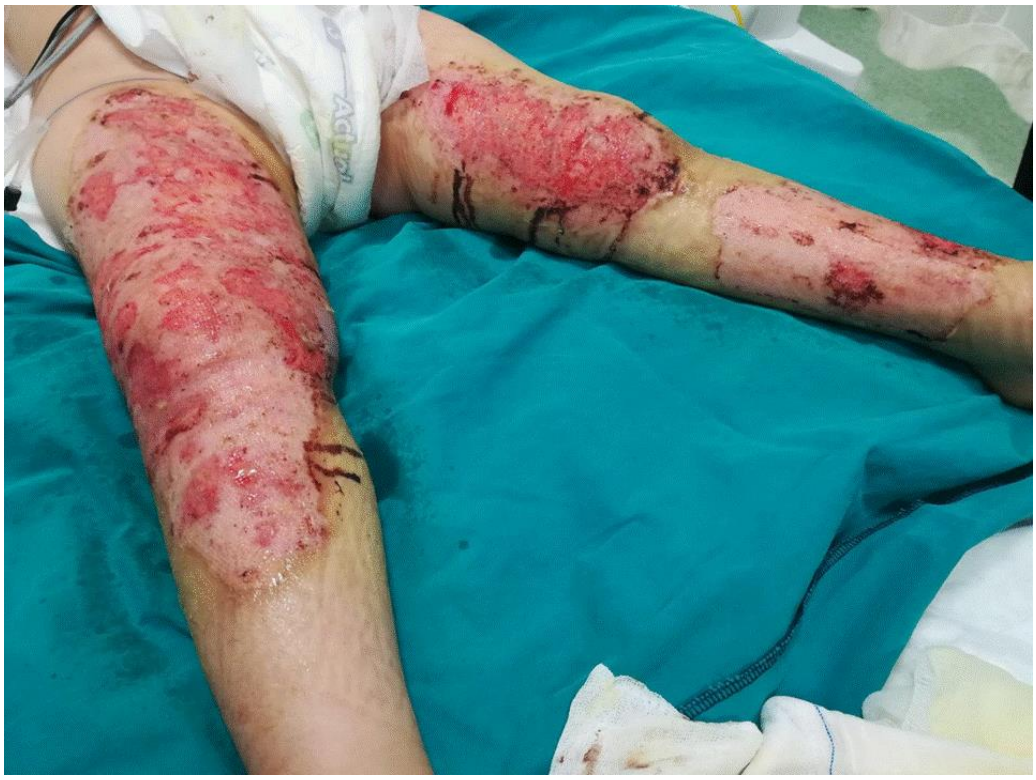

Figure 1: Second degree burn injuries in both legs

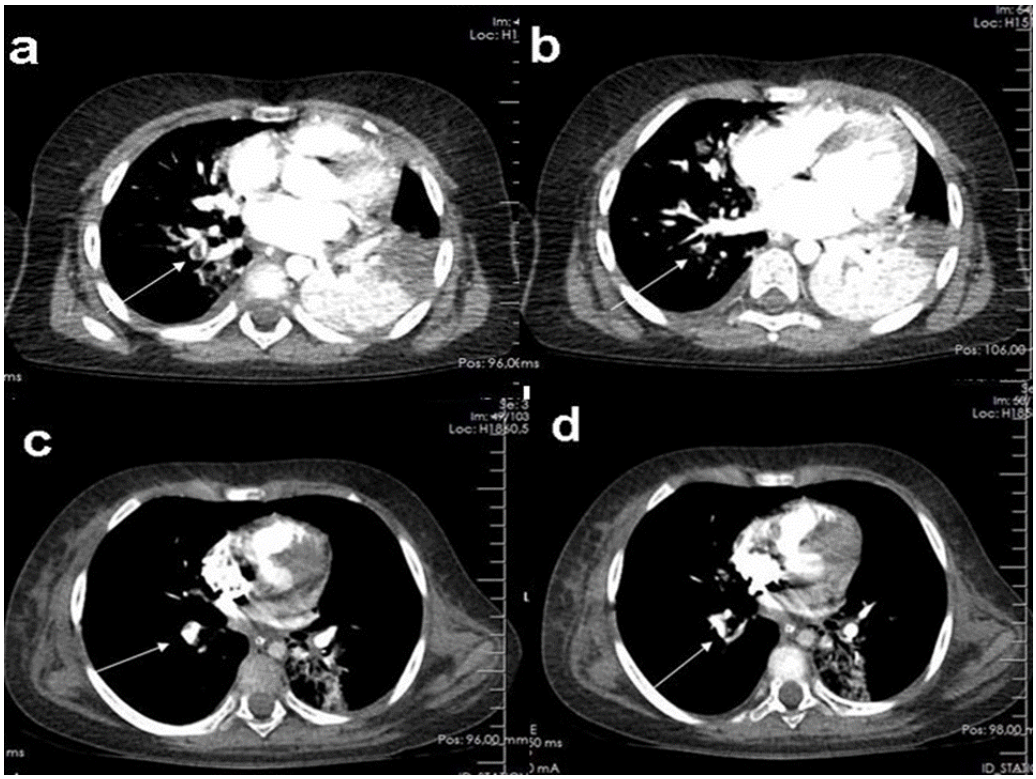

Figure 2: Axial images (a,b) of pulmoner CT angiogram of the case show, a thrombus in the lobar and segmental branches of the right pulmonary artery supplying the right lower lobe (arrow); and images (c,d) show that the thrombus in the lobar and segmental branches of the pulmonary artery disappeared and that branch was recanalized (arrow).

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DISCUSSION

Burn patients exhibit an acquired hypercoagulable state, which puts them at risk for thromboembolic complications. Barret et al. reviewed a series of 3331 burn patients in an attempt to determine the incidence of arterial thrombosis, deep venous thrombosis, and PTE (5). Ten patients presented with thrombotic complications, corresponding to a raw incidence of 0.3%. One complication occurred in the pediatric population (incidence 0.1%). This 8-years-old boy mentioned, had thrombosis of the common femoral artery. Three of the adults presented with PTE. Thrombotic complications accounted for 3.38% of all deaths in burn population (5). Our case is unique compared with those described in the literature, given that, to the best of our knowledge, no pediatric burn patient with thromboembolism in this age group has been described. Mullins et al. described multiple coagulation derangements in burn patients, including elevated platelets, fibrinogen, factor V, and factor VIII, which could contribute to a pro-coagulable state (6). Since burn patients often fulfill all components of the Virchow's triad, namely stasis, local injury, and hypercoagulability, it does not seem unreasonable that burn patients may be at increased risk for PTE (7). Among the risk factors, long-term immobilization was associated with increased risk of PTE. Regarding the risk factors for PTE, Gülcü et al. reported immobilization in 41.9% of their patients and recent surgery in 32.3% (8). Hence, our patient had some risk factors such as immobilization, burn, and being overweight.

Pulmonary thromboembolism is often caused by partial or complete obstruction of blood flow in the pulmonary arterial bed by a thrombus formed in the systemic veins (9,10). Computed tomographic pulmonary angiography has been increasingly used as the imaging method of choice for suspected PTE. The extent of a PTE is commonly expressed by the anatomical level of the most proximal vessel affected by a thrombus (11). For a definitive diagnosis of suspected PTE, D-dimer is a useful marker. Plasma D-dimer measurement provides information about fibrin degradation fragments following fibrinolysis. D-dimer assays have a low specificity albeit a high sensitivity and negative predictive value in most patients with suspected thromboembolism (12).

PTE occurs in a broad spectrum, from non-massive asymptomatic thrombi to massive thrombi causing shock or cardiopulmonary arrest, and the incidence of PTE is less than 2% of post-burn deaths in the pediatric population (13-15). Likewise, the localization of the thrombus in our case was in a relatively large vessel, namely lobar branch of the pulmonary artery, which caused such a severe clinical presentation.

Anticoagulant prophylaxis, which includes low molecular weight heparin, low dose unfractionated heparin, heparin, warfarin, acetylsalicylic acid and factor Xa inhibitors, has been shown to be effective in preventing deep vein thrombosis in burn patients (16,17). Unfortunately, it was not possible to evaluate Factor V Leiden and prothrombin gene mutation G20210A for this patient, which may increase the patient's risk of thromboembolism.

Heparin was successfully used in our clinic during the follow-up period to remove thrombus in our patient who had not received any anticoagulant prophylaxis before admission to our clinic.

This case report illustrates the importance of chemoprophylaxis against pulmonary embolism in both pediatric and adult burn patients. In such cases, especially the severe ones, a sudden deterioration of patient's clinical status in the form of respiratory distress or failure should raise suspicion about the possibility of pulmonary embolism.

Conflict of Interest: All authors declare that they have no conflict of interest.

The study does not have any financial support

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