Acute Respiratory Distress Syndrome as a Complication of Generalized Pustular and Erythrodermic Psoriasis

Jeneralize Püstüler ve Eritrodermik Psoriasisin Bir Komplikasyonu Olarak Akut Respiratuar Distress Sendromu

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ABSTRACT

Psoriasis-associated acute respiratory distress syndrome (ARDS) (aseptic pneumonitis) is a rare complication that may occur in patients with erythrodermic psoriasis. Its pathogenesis includes inflammation, hyper proliferation and differentiation disorder. Herein this case report, a 48 years old male patient who was followed up with the diagnosis of psoriasis for 15 years, whose respiratory complaints started with exacerbation of skin lesions and who developed ARDS (aseptic pneumonitis) mainly due to severe psoriasis is presented.

Keywords: Psoriasis, erythrodermic aseptic pneumonitis, ARDS, respiratory failure

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ÖZET

Psoriasis ile ilişkili akut solunum sıkıntısı sendromu (ARDS) (aseptik pnömonit), eritrodermik psöriazis hastalarında görülebilen nadir bir komplikasyondur. Patogenezinde inflamasyon, hiper proliferasyon ve farklılaşma bozukluğu söz konusudur. Bu olgu sunumunda 15 yıldır psoriasis hastalığı tanısı ile izlenen, solunum şikayetleri deri lezyonlarının şiddetlenmesi ile başlayan ve ağırlıklı olarak şiddetli psoriazis nedeniyle ARDS (aseptik pnömonit) gelişen 48 yaşında bir erkek hasta sunulmaktadır.

Anahtar Sözcükler: Psoriazis, eritrodermik psoriazis, aseptik pnömonitis, ARDS

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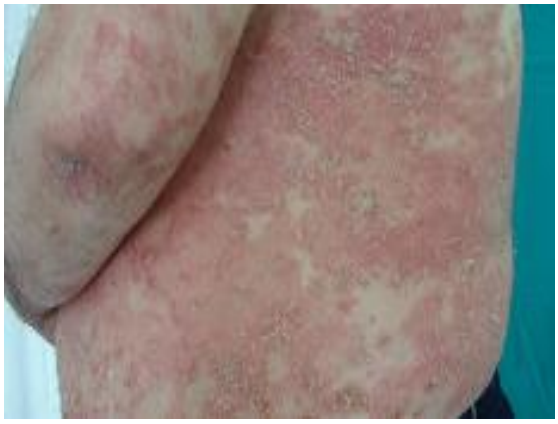
INTRODUCTION

Psoriasis is a recurrent chronic inflammatory skin disease, usually of benign character. Despite the high incidence of psoriasis in the general population, pulmonary complications are extremely rare. The main pulmonary complications developing in psoriasis are pulmonary infections due to immunosuppressive drugs used in the treatment of psoriasis and drug-induced hypersensitivity pneumonia. Psoriasis-associated acute respiratory distress syndrome (ARDS) (aseptic pneumonitis) is a rare complication that may occur in patients with erythrodermic psoriasis, which is a severe form of psoriasis characterized by widespread erythema and desquamation, accompanied by systemic symptoms such as fever, lymphadenopathy, and poor general condition. Its pathogenesis includes inflammation, hyper proliferation and differentiation disorder. Herein this case report, a patient who was followed up with the diagnosis of psoriasis, whose respiratory complaints started with exacerbation of skin lesions and who developed ARDS (aseptic pneumonitis) mainly due to severe psoriasis is presented.

CASE REPORT

A 48-year-old male patient who was followed up for 15 years with the diagnosis of psoriasis was admitted to the Emergency Department with the complaints of fever and dyspnea. He had a cough and sputum for about 1 month.

He was nonsmoker and had no additional disease. He used methotrexate and acitretin sporadically before, but he discontinued methotrexate 5 years ago due to allergies and acitretin 2 years ago due to nose bleeds. Two months ago he was hospitalized due to psoriasis exacerbation and cyclosporine was started. However, cyclosporine was discontinued due to the deterioration of liver function tests. Before admitting to our emergency department he was hospitalized with the diagnosis of pneumonia and used multiple antibiotics for two weeks in another center. s far as we are aware, during his prior hospitalization, there was no microbial development. He was discharged from there, stating that his lung findings did not regress with antibiotics, and he was recommended to apply to a university hospital for further examination. At admission, the patient's temperature, heart rate and respiratory rate, oxygen saturation with 4 lt/min nasal oxygen was respectively 37°C, 122/min, 33/min, and 77%, respectively. In his physical examination, widespread erythematous squamous plaques all over the body were remarkable (Figure-1). On respiratory examination, bilateral coarse crackles were heard. Arterial blood gas analysis in room air revealed pH:7.54, PCO_2:25.1 mmHg, PO_2:39.2 mmHg, and HCO_3^-:24 mmol/L., PaO_2/FiO_2 ratio:187. In the complete blood count test, white blood cell count: 11280 µl/ml, neutrophil count: 10320 µl/ml and hemoglobin: 10.1 g/dL. His biochemistry test was unremarkable except for sodium: 128 mmol/L (hyponatremia) and elevated liver enzymes (AST: 111 U/L, ALT: 76 U/L). There was diffuse bilateral patchy infiltration in the posterior-anterior chest X-ray (Figure 2). In the thorax computerized tomography (CT) taken with the angiography protocol, there were more prominent ground-glass infiltration areas in the bilateral lower zones (Figure 3a, b, c).



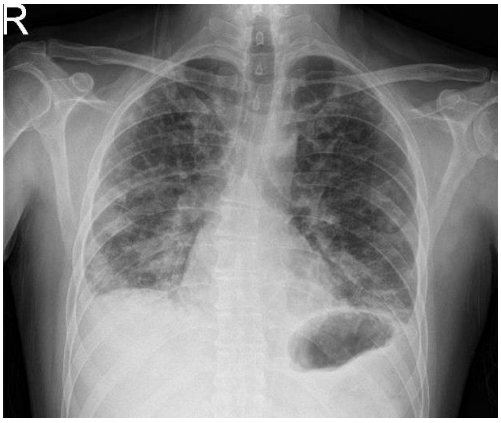


Figure-1: There were diffuse erythematous squamous plaques throughout the body
Figure-2: Posteroanterior chest X ray of the patient with diffuse bilateral patchy infiltrates

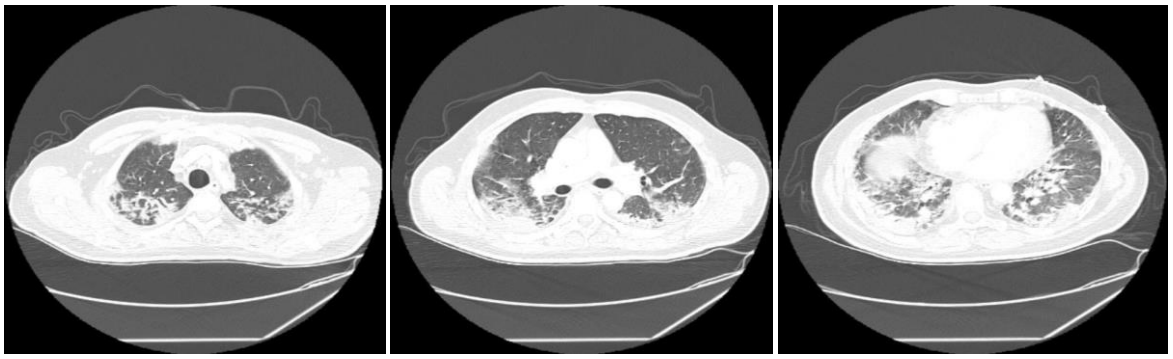


Figure-3a Figure-3b Figure-3c

Figure-3a, 3b, 3c: Thorax computerized tomography (CT) at admission to Emergency Department. Ground-glass infiltration areas especially in dependent regions; they were more prominent and together with consolidation areas bilaterally in lower zones

The patient, who was tachypneic and had worsening hypoxia, was intubated and followed up in the intensive care clinic with a preliminary diagnosis of ARDS. First, the patient was monitored in volume-assist controlled mode, with FiO2: 70%, tidal volume:480, respiratory rate: 20 and PEEP: 10. Under these settings PaO_2/FiO_2 was 104 mmHg.

In his laboratory, CRP (C-reactive protein) was 141 mg/L, procalcitonin was 0.48 ng/ml, white blood cell count was 14290 µl/ml, and neutrophil count was 12950 µl/ml. There was no growth in blood culture and urine culture. Bronchoscopy was performed and a bronchoalveolar lavage sample was obtained. Considering immunosuppressive pneumonia, sulbactam-cefaperazone, co-trimoxazole, valacyclovir and clarithromycin were given empirically.

The bronchoalveolar lavage sample analysis was negative for CMV (cytomegalo virus), candida PCR (polymerase chain reaction), Tbc (tuberculosis)-PCR, PCP (pneumocystis jirovecii pneumonia)-PCR, aspergillus PCR and ARB (acid resistant bacilli). The respiratory virus panel or the Legionella antigen/PCR were not examined. There was no growth in the bronchial lavage culture. Considering cardiac pathologies, cardiac enzymes and pro-BNP (pro-brain natriuretic peptide) were sent. Cardiac enzymes and pro-BNP were normal. Electrocardiography (ECG) was found in normal sinus rhythm. No cardiac pathology was detected in the echocardiography performed on the patient. He was evaluated by dermatology for widespread, uncontrolled lesions covering the whole body, a punch biopsy was performed and 80 mg methylprednisolone treatment was started. The patient, whose oxygen demand did not decrease, had been started neuromuscular blocker and followed up in the prone position at 12 to 16 hours intervals.

Ventilation was continued in the following days in pressure-controlled mode, with FiO_2:100%, PS:20 cmH_20, PEEP:12 cmH_20 and respiratory rate:20/min. Under these settings PaO2/FiO2 ratio of was 63.6 mmHg. Due to worsening clinic of the patient in addition to antibiotic therapies, treatment with intravenous vitamin C 1500 mg every 6 hours, intravenous hydrocortisone 50 mg every 6 hours and intravenous thiamine 200 mg every 12 hours was also initiated. Control thorax CT was performed; dense consolidations bilaterally with air bronchograms in dependent regions and wide spread ground glass attenuation among the other regions of the lung was observed that was typical for ARDS (Figure-4a-d). The chest X-ray taken at that time revealed bilateral white lungs (Figure 5). The patient is being followed with 100% FiO_2 and the PaO_2/FiO_2 ratio was 48.9 mmHg (Figure 6).

ECMO (Extra Corporeal Membrane Oxygenation) was planned for the patient who progressed to severe ARDS and whose PaO_2/FiO_2 ratio did not improve, but the patient died before ECMO could be performed.

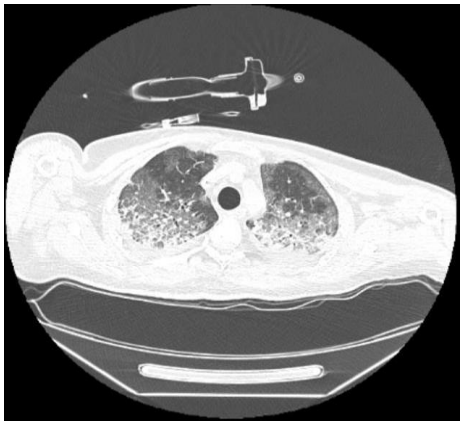


Figure 4a

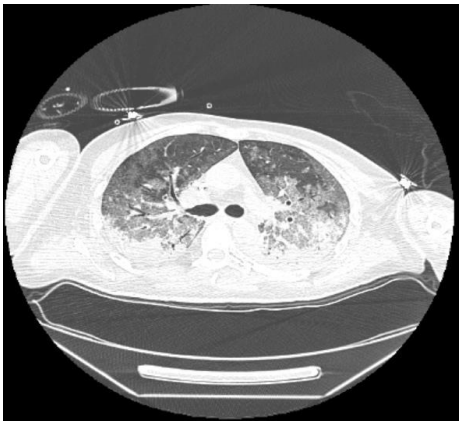


Figure 4b

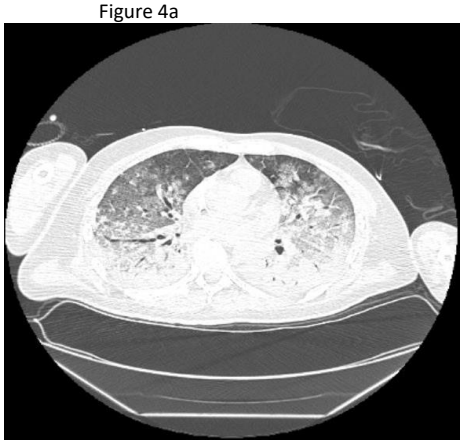


Figure 4c

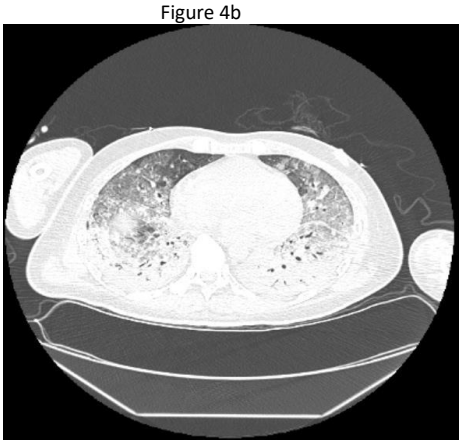


Figure 4d

Figure-4 a-d: Bilateral consolidation area with air bronchogram and ground glass appearance (typical for ARDS)

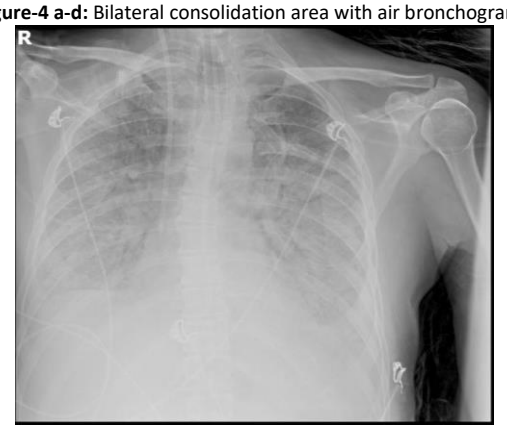


Figure 5- Chest X ray during followup in intensive care unit- Bilateral white lungs, typical for ARDS, indicating progression of respiratory failure

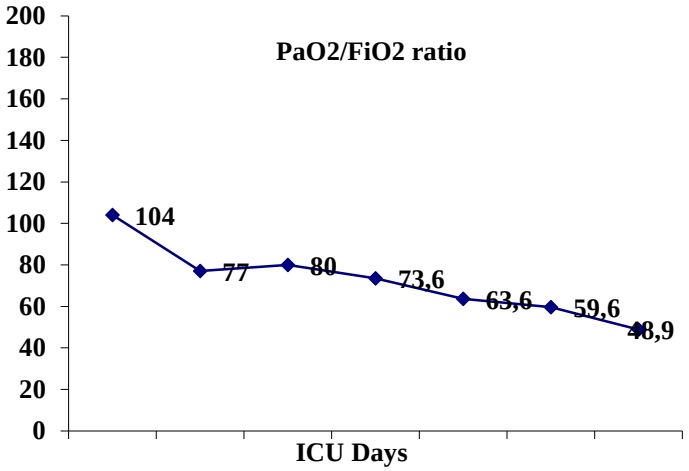


Figure 6- PaO2/FiO2 ratio course graphic during the ICU followup after intubation.

DISCUSSION

Here in this case report we want to present one of the rare pulmonary complications that may develop in psoriasis patients i.e., aseptic pneumonitis. This picture is seen in generalized pustular and/or erythrodermic psoriasis and can be serious and fatal. Congestive heart failure, acute lung infection-related or unrelated to immunosuppressive therapy, and drug hypersensitivity reaction should be considered in the differential diagnosis (1). In our case, drug-related causes were not considered, since the patient was not using methotrexate and acitretin for a long time, and the cyclosporine was used for a short time and discontinued before respiratory symptoms had appeared. Besides no infectious etiology was identified although frequent cultures were taken and PCR had been performed for opportunistic pathogens.

A case of sterile pneumonitis as a complication of generalized pustular psoriasis was first reported by Landry and Muller in 1972(2). Since then, a limited number of cases of ARDS (aseptic pneumonitis) due to generalized pustular or erythrodermic psoriasis have been reported (3). Interestingly, as in our case, fever, tachypnea, hypoxia, rapid deterioration in respiration and increased neutrophils were observed in the reported cases (Figure 7) (4-6). Bilateral infiltration areas were noted on chest X-ray or tomography. Its pathogenesis is unknown, but as seen in animal models, it is thought that active T-helper (Th) 1 lymphocytes in psoriasis trigger alveolitis, and lymphocytes that secrete cytokines that provide cell adhesion with the Th-1 major cytokine, tumor necrosis factor, cause lung infiltration (7). High-dose systemic corticosteroid therapy together with an early diagnosis will provide the management of this disease (6). In 2011, Klugger et al. reported a complication of ARDS in a 14-year-old girl diagnosed with generalized pustular arthritis (3). This patient responded successfully to CPAP therapy and pulse corticosteroid therapy. There are cases reported with the diagnosis of psoriasis-associated sterile pneumonitis in the literature, and some of these cases were treated with corticosteroids.

Paul Marik et al., on the other hand, applied low-dose systemic corticosteroid, intravenous vitamin C and thiamine treatment in the case they reported and received a successful response (6). With the use of this case report of Paul Marik et al. we also gave intravenous vitamin C and thiamine treatment to our patient, but no response had been achieved with this treatment.

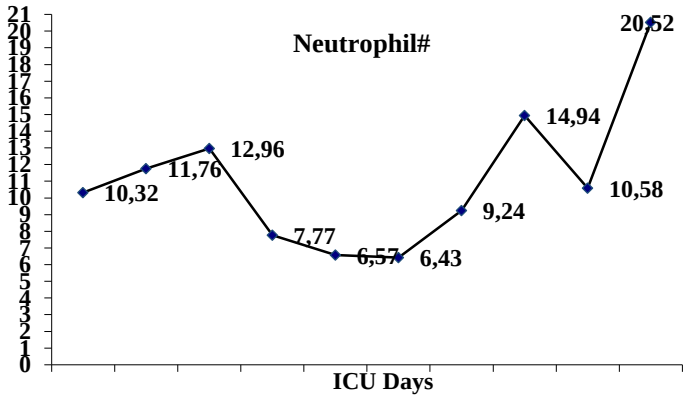


Figure 7- Neutrophil number course graphic during ICU follow-up

In conclusion, if respiratory symptoms occur in a patient with psoriasis, psoriasis-associated ARDS (aseptic pneumonitis) should also be considered in addition to pulmonary infections and drug pneumotoxic effects. Early diagnosis can improve the prognosis of these patients.

Conflict of interest

No conflict of interest was declared by the authors.

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