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A Rare Cause of Respiratory Failure: Negative Pressure Pulmonary Edema

Solunum Yetmezliğinin Nadir Bir Nedeni: Negatif Basınçlı Pulmoner Ödem

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Abstract

We report here on the successful management of a patient who developed dyspnea after an elective arthroscopic procedure and who was diagnosed with Negative Pressure Pulmonary Edema (NPPE). A 29-year-old male patient with no known medical history was admitted to our hospital due to a rotator cuff tear sustained during a military operation. An arthroscopic procedure under general anesthesia was performed; however, the patient developed dyspnea 2 hours after extubation. He was desaturated, tachypneic and began to produce frothy pink sputum. Pulmonary computed-tomography angiography revealed widespread ground-glass opacities in both lungs suggesting non-cardiogenic pulmonary edema without acute pulmonary thromboembolism. The patient was thus diagnosed with NPPE and treated with non-invasive mechanical ventilation, intravenous furosemide and inhaled short-acting beta-agonists. Significant recovery was observed in a couple of days, and the patient discharged after his respiratory symptoms abated. NPPE should immediately be suspected in individuals who develop respiratory failure following extubation, as it can be life threatening.

Key words: *Sarcoidosis, Silicosis, differential diagnosis.*

Öz

Bu olgu sunumunda, elektif artroskopik işlem sonrası nefes darlığı gelişen, negatif basınçlı pulmoner ödem (NBPÖ) tanısı konan ve başarıyla tedavi edilen bir hasta sunulmuştur. Yirmi dokuz yaşında, herhangi bir tıbbi öyküsü olmayan erkek hasta, askeri operasyon sırasında gelişen rotator manşet yırtığı nedeniyle hastanemize başvurdu. Genel anestezi altında artroskopik işlem yapıldı. Ancak hastada ekstübasyondan 2 saat sonra dispne gelişti. Hastada satürasyon düşüklüğü ve takipne ile birlikte ve köpüklü pembe balgam görüldü. Bilgisayarlı tomografi pulmoner anjiyografide tromboemboli saptanmayıp, her iki akciğerde kardiyojenik olmayan pulmoner ödem düşündüren yaygın buzlu cam opasiteleri saptandı. Hasta NBPÖ tanısı konularak invazif olmayan mekanik ventilasyon, intravenöz furosemid ve inhale kısa etkili beta-agonistler ile tedavi edildi. Birkaç gün içinde belirgin bir iyileşme görülen hasta herhangi bir solunum semptomu olmadan taburcu edildi. Ekstübasyon sonrası solunum yetmezliği gelişen kişilerde hayatı tehdit edici bir tablo olan NBPÖ akla gelmelidir.

Anahtar Sözcükler: *Sarkoidoz, Silikoz, ayırıcı tanı.*

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Negative pressure pulmonary edema (NPPE) is a rare complication that can occur as a result of laryngospasm following extubation after an operation requiring general anesthesia (1). As a potentially life-threatening emergency usually seen in young and healthy individuals, and that can ultimately be recovered with a quick diagnosis, all physicians should keep NPPE in mind during the post-operative period as a clinically significant phenomenon.
Here we report on a patient who was managed successfully after developing dyspnea following an elective arthroscopic procedure and being diagnosed with NPPE.

CASE

A 29-year-old male soldier with no known medical history was admitted to our hospital with a rotator cuff tear sustained during a military operation. An arthroscopic procedure under general anesthesia was performed, and the tear was repaired. The patient developed dyspnea 2 hours after extubation, and was found to be desaturated (SpO_2 at room air 75%) and tachypneic (respiratory rate: 26-28/min), and began to produce a frothy pink sputum. The patient was immediately placed on 10 L/min oxygen support with a mask. Bilateral coarse crackles were heard during auscultation of the lungs, and a chest X-ray revealed bilateral recently-developed pulmonary infiltrations (Figure 1). Blood tests showed leukocytosis (predominantly neutrophils), mild hypoxemia, lactic acidosis and elevated D-dimer levels. The laboratory results are presented in Table 1. It was found out that earlier in the operation room the patient had developed laryngospasm following extubation. As a result of the sudden onset dyspnea and the elevated D-dimer levels, a pulmonary computed-tomography (CT) angiography for acute pulmonary thromboembolism (PTE) was performed. No sign suggesting acute PTE was observed, although widespread ground-glass opacities were seen in both lungs, suggesting non-cardiogenic pulmonary edema (Figure 2). The Electrocardiogram showed normal sinus rhythm and did not show any ischemic change. Transthoracic echocardiography revealed normal left ventricular systolic function.
The patient was diagnosed with NPPE, and was administered intravenous furosemide and inhaled short-acting beta-agonists. The patient was transferred to the intensive care unit where he received non-invasive mechanical ventilation, and significant recovery was observed in a couple of days. His dyspnea and sputum production regressed entirely. A control chest X-ray is shown in Figure 1. The patient was discharged after his need for oxygen support abated.

DISCUSSION

NPPE is a type of non-cardiogenic pulmonary edema, and is a post-operative complication that can be life-threatening unless diagnosed and treated early. It is usually seen in young and healthy individuals with a prevalence of 0.05–0.1% (1), and is prevalent in those who are prone to airway obstruction. Risk factors include obesity, short and thick neck, obstructive sleep apnea syndrome, oropharyngeal and head/neck surgeries (2). The duration between airway obstruction and the development of pulmonary edema is only a couple of minutes in many cases reported to date (1).
NPPE occurs due to an increase in negative intrathoracic pressure (greater than -100 cmH2O, which is approximately ten times the normal negative intrathoracic pressure), to upper airway obstructions and to the transfer of fluid to the pulmonary interstitium. High intrathoracic pressure results in increased venous return and elevated capillary hydrostatic pressure. In addition, adrenergic activation and increased pulmonary vascular resistance caused by hypoxia and acidosis result in right ventricular expansion, deviation of the interventricular septum towards left ventricle and left ventricular diastolic dysfunction. Increased venous return and elevated pulmonary wedge pressure cause fluid to pass into the pulmonary interstitium, and pulmonary edema develops (3).

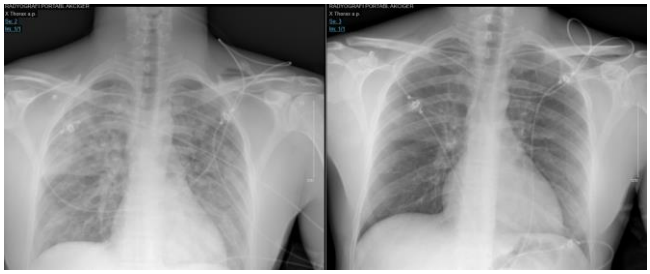

Figure 1: Bilateral pulmonary infiltrations on chest X-ray during respiratory distress (left), resolution of infiltrations after treatment (right)

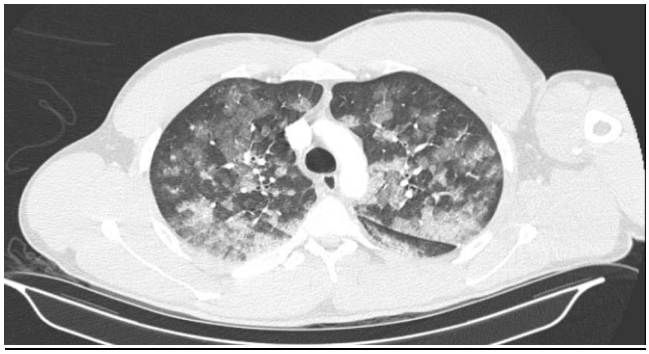

Figure 2: Bilateral widespread ground glass opacities that are more dominant posteriorly, and interstitial thickening (cobblestone pattern), which indicates non-cardiogenic pulmonary edema

Table 1: Laboratory results of the patient at the time of respiratory failure

Parameter	Value	Normal Range
Hemoglobin (g/dL)	16,9	13-17
Leukocyte count (x103/µL)	24,8	4,3-10,3
Neutrophil count (x103/µL)	23,2	2,1-6,1
Thrombocyte count (x103/µL)	286	156-373
Creatinine (mg/dL)	1,02	0,67-1,17
AST (U/L) [a]	30	<50
ALT (U/L) [b]	37	<50
Sedimentation rate (mm/h)	2	0-20
CRP (mg/dL) [c]	0,36	0-0,5
Myoglobin (µg/L)	67,4	17,4-105,7
Troponin I (ng/L)	3,4	14-42,9
CK-MB (µg/L) d	2	0,6-6,3
D-dimer (mg/L)	8,22	0-0,55
BNP (pg/mL) [e]	<10	0-100
pH	7,33	7,35-7,45
SO_2 (%) [f]	92,4	40-98
pO_2 (mmHg) [g]	71,5	80
pCO_2 (mmHg) [h]	42,6	35-48
$cHCO_3$ (Standard) (mmol/L) [i]	21,3	22,5-26,9

[a] Aspartate aminotransferase, [b] Alanine aminotransferase, [c] C-reactive protein, [d] Creatine kinase- MB, [e] Brain Natriuretic Peptide, [f] Oxygen saturation, [g] Partial oxygen pressure, [h] Partial carbondioxide pressure, [i] Bicarbonate concentration

Signs and symptoms often include dyspnea, tachypnea, bloody and frothy sputum, low oxygen saturation, pulmonary crackles and signs of pulmonary edema on chest X-ray. Differential diagnosis includes other causes of pulmonary edema, such as aspiration of gastric content, acute respiratory distress syndrome, congestive heart failure, hypervolemia and pulmonary thromboembolism (3). The absence of other etiologies listed above and the onset of symptoms shortly after laryngospasm led to our case being diagnosed with NPPE.

The treatment of NPPE comprises careful monitorization, treatment of airway obstructions, and oxygen and mechanical ventilatory support (1). There are different opinions on the optimum medical treatment. While some studies suggest that furosemide treatment is unnecessary, since NPPE results from leaky capillaries and there is not excessive fluid or hypervolemia (4). Others suggest furosemide treatment due to its support of symptomatic and radiological recovery (5). Even though there is no bronchospasm, studies have shown that beta-agonist treatment may heal pulmonary edema symptoms by making removal of the fluid in the alveoli easier (6). Cases of NPPE are usually recovered both clinical and radiological quickly in 12-48 hours (5). Our case recovered quickly with oxygen and non-invasive mechanical ventilator support, diuretic, and inhaler beta-agonist treatment, and no sequel was observed.

The prevention or early treatment of upper airway obstructions would decrease the incidence of NPPE. Intraoperative muscle relaxants, topical or spray lidocaine, or steroids might be used (3). Moreover, further precautions include careful aspiration of oropharyngeal secretions,

intubation of patients under very deep or very light anesthesia when the risk of laryngospasm development is the lowest and prophylactic continuous positive airway pressure (CPAP) administration (7).

CONCLUSION

NPPE should be recalled quickly for patients who develop respiratory failure following extubation, especially those with a history of upper airway obstruction, as it can be life threatening. Complete recovery is highly possible with early diagnosis and treatment, and so all physicians should be aware of this phenomenon.

CONFLICTS OF INTEREST

None declared.

AUTHOR CONTRIBUTIONS

Concept - B.S., O.A.U.; Planning and Design - B.S., O.A.U.; Supervision - B.S., O.A.U.; Funding - O.A.U.; Materials - B.S.; Data Collection and/or Processing - B.S.; Analysis and/or Interpretation - B.S., O.A.U.; Literature Review - B.S.; Writing - B.S., O.A.U.; Critical Review - O.A.U.

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