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Pneumoscrotum in a Patient with *Pneumocystis jirovecii* Pneumonia: A Case Report

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Abstract

Pneumoscrotum is a rare condition described as the accumulation of air within the scrotum. We report the case of a patient with acquired immune deficiency syndrome (AIDS) with *Pneumocystis jirovecii* pneumonia who developed a secondary pneumoscrotum as a complication of volutrauma from mechanical ventilation.

Keywords

Mechanical Ventilation, Pneumomediastinum, Pneumoscrotum, Pneumothorax, Volutrauma

1. Introduction

Pneumoscrotum remains a clinical identity infrequently described in the urological literature. There have been 59 cases described in the literature from 1973-2013 and are organized into primary and secondary causes of air accumulation within the scrotal sac [1]. The primary mechanism involves the direct introduction of air or gas into the scrotum, often as a result of infectious etiologies or traumatic injury to the area. Secondary etiologies generally involve air traveling along the layers of Scarpa's and Camper's fascia or via diffusion of gas from the peritoneal cavity.

Pneumoscrotum is a potential complication of advanced *Pneumocystis jirovecii* pneumonia (PJP) in which spontaneous pneumothoraces can occur [2] [3]. With the advent of highly active antiretroviral therapy (HAART) for patients with acquired immune deficiency syndrome (AIDS), however, outcomes for PJP

have markedly improved and such complications are now rarely seen. Here we present the case of a patient who presented with advanced PJP, who, following mechanical ventilation, developed a large pneumomediastinum, diffuse subcutaneous emphysema, and subsequently a secondary pneumoscrotum.

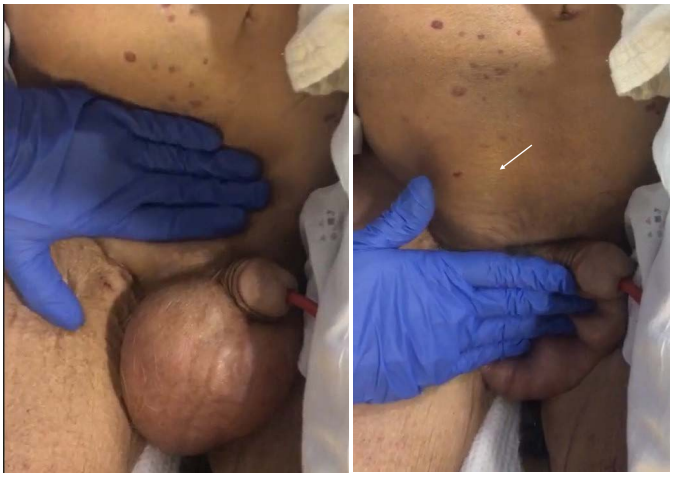
2. Case Presentation

A 50-year-old homeless male with a history of human immunodeficiency virus infection (HIV) and HAART for the past two years presented with progressive dyspnea for one month and several months of weight loss. He related a history of intravenous drug and active methamphetamine use. Admission vital signs were a temperature of 38.4˚C, heart rate of 103 beats/minute, blood pressure 126/88mmHg, respiratory rate of 28 breaths/minute, and blood oxygen saturation of 78% on room air. Physical examination showed cachexia, tachypnea and crackles over both lung fields.

A blood CD4 count was 21 cells/uL. Empiric treatment for PJP was started with glucocorticoids and trimethoprim-sulfamethoxazole (TMP-SMX). HAART was re-initiated on hospital day 9, and the patient was stable for transfer to a skilled nursing facility while on supplemental oxygen via nasal cannula at 2 L/min. On hospital day 25 oxygenation requirements increased and treatment for presumed pneumonia was started. On hospital day 27, he became tachypneic with a respiratory rate of 42 breaths/min and oxygen saturation decreased to 78% on nasal cannula oxygen, and required emergent intubation and initiation of low tidal volume (6 mL/kg) mechanical ventilation. On hospital day 29, the patient exhibited crepitus around the neck and chest x-ray demonstrated small bilateral apical pneumothoraces and subcutaneous emphysema. Bronchoalveolar lavage on hospital day 32 demonstrated PJP by direct fluorescent antibody (DFA) testing and a serum cytomegalovirus (CMV) titer of 94,643 IU/mL. Treatment was started with intravenous ganciclovir for presumed CMV pneumonitis, and glucocorticoids were continued.

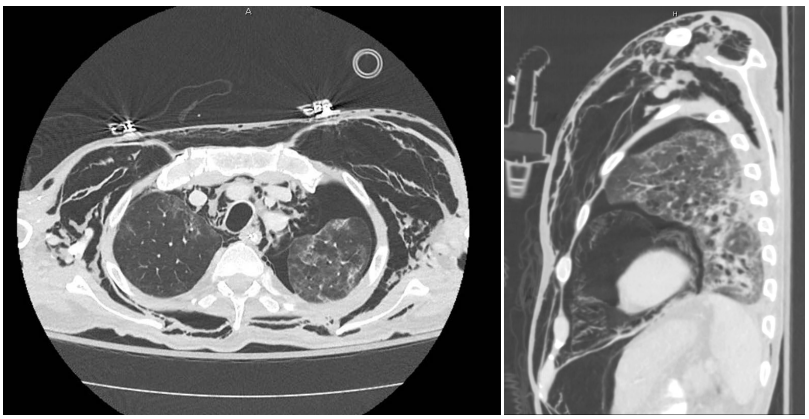
Mechanical ventilation was continued throughout the hospital course, leading to tracheostomy placement on hospital day 57. Following the procedure, the patient developed rapid expansion of the left apical pneumothorax, necessitating chest tube drainage for one day. Despite resolution of the pneumothorax patient continued to demonstrate progression of subcutaneous emphysema on hospital days 60 - 63, with radiographic imaging consistent with pneumomediastinum and pneumopericardium. On day 63, the patient developed hemodynamic instability caused by a tension pneumothorax involving the left lung, again managed with an emergent chest tube. Repeat bronchoscopic evaluation showed no defects within the tracheobronchial tree.

On the same day, genitourinary exam demonstrated palpable crepitus along the scrotum (**Figure 1(a)**, **Figure 1(b)**), worse on the right side and contiguous with subcutaneous emphysema over the lower abdominal wall. Computerized tomography (CT) imaging of the thorax and neck (**Figure 2(a)**, **Figure 2(b)**)



(a) (b)

Figure 1. Photographs showing pneumoscrotum with reciprocal shift of air along the subcutaneous tissue by direct pressure, illustrating communication between the (a) scrotum and (b) anterior abdominal wall (arrow).



(a) (b)

Figure 2. Computerized tomography (CT) imaging of the thorax and neck showing a large pneumomediastinum with concurrent pneumothorax (a) coronal view, (b) sagittal view.

demonstrated a large pneumomediastinum with concurrent pneumothorax, pneumopericardium, pneumoperitoneum and subcutaneous emphysema extending from the neck to the scrotum, with no evidence of apparent etiology for large volume airway leak. Despite conservative management, the left chest tube continued to manifest an airway leak, which was attributed to severe parenchymal disease. Given the patient's serious overall condition he was deemed not to be a suitable candidate for surgical management or single lung ventilation. With no surrogate decision-makers able to be located, the medical ethics committee and primary care team reached a decision to withdraw care.

3. Discussion

The incidence of pneumothorax in hospital admissions with PJP was found to

be 4.7% in an earlier prospective observational study [4]. The study also found that the risk of mortality with concurrent pneumothorax in an HIV patient, such as ours, was higher with in-hospital mortality reaching 30.8% compared to 5.8% in matched counterparts. In our case, the pneumothorax initially occurred in the setting of PJP, with positive pressure ventilation likely leading to worsening subcutaneous emphysema and eventual pneumoscrotum.

Although data on pneumoscrotum as a clinical entity are sparse, there have been case studies published suggesting that the most common causes are generally secondary trauma and iatrogenic injury [5] with surgical intervention [6] [7]. Because this is an underreported condition, there is little information on the best management of pneumoscrotum; treatment is generally supportive with reversal of the underlying etiology.

Unfortunately, in the case of our patient, end stage lung disease prevented reversal of the diffuse subcutaneous emphysema. In later stages of PJP, with severe inflammation of the lung tissue, the normal parenchyma can be replaced by pneumatoceles and cysts—which promote the occurrence of spontaneous pneumothorax [3]. Despite lengthy treatment for PJP, the patient's respiratory condition deteriorated following prolonged positive-pressure mechanical ventilation.

In addition to treatment of the underlying infectious etiology, methods of lung-protective mechanical ventilation are suggested to prevent volutrauma, including low tidal volumes (6 mL/kg) [8], targeting smaller driving pressures and monitoring respiratory compliance [9], all shown to be helpful in improving mortality. Despite applying low tidal volume, the air leak continued in this patient.

4. Conclusions

Patients with PJP pneumonia have a high risk for volutrauma, including pneumoscrotum, and mechanical ventilation should be used cautiously to prevent further complications in these patients.

All authors were equally involved in the drafting, editing, and review of the manuscript.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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Abbreviations

CT: computerized tomography, PJP: *Pneumocystis jirovecii* Pneumonia, HAART: highly active antiretroviral therapy, HIV: Human Immunodeficiency Virus, AIDS: Autoimmune deficiency Syndrome, CMV: cytomegalovirus, DFA: direct fluorescent antibody