Acute Pulmonary Vascular Talcosis: Mimicking Acute Pulmonary Embolism

Nandavaram S*, Chandrasekar VT and Savici D
State University of New York Upstate Medical University, Syracuse, USA

Abstract

Intravenous injection of powdered tablets intended to oral use is a common practice among drug abusers, given the greater effectiveness attributed to accelerate delivery of the drug through this route compared to oral intake. This can result in both acute and chronic changes in the pulmonary vessels and parenchyma. Various terms have been used to describe this entity and include self-induced pulmonary granulomatosis, pulmonary antithrombotic granulomatosis, pulmonary mainline granulomatosis, and angiocentric systemic granulomatosis. Acute right ventricular failure is a life threatening complication from intravenous injection of powdered medications and very few such cases have been reported in the literature.

Keywords: Talcosis; Pulmonary hypertension; Hypertension; Acute RV failure

Introduction

Sudden cardiac death secondary to intravenous drug abuse is an under diagnosed life threatening complication [1,2]. Here we present a case of acute pulmonary intravascular talcosis resulting in acute cardiogenic shock, right ventricular failure and death (Figure 1).

Case Presentation

30-year-old male with medical history significant for intravenous drug abuse presented to emergency room with complaints of fever chills and back pain. At the time of presentation he was afebrile, blood pressure was 132/76 mm Hg, pulse was 95, and respiratory rate 16 and oxygen saturation was 96% on room air. Physical exam was significant for tenderness to palpation in the paraspinal area. Diagnostic work up was significant for white blood cell count 15.5. Echocardiogram was within normal limits. Blood cultures were positive for MRSA. MRI thoracic spine revealed infiltrative process in the dorsal epidural space from T6-T7 down to T10 level with inhomogeneous enhancement and mass effect on the spinal cord at T8 and T9 levels representing a phlegmon. Patient was started on intravenous antibiotics. IR guided aspiration of the epidural lesion was unsuccessful and the decision was made to treat him with intravenous vancomycin for a total of 6 weeks and then to follow up with imaging. Given the history of drug abuse, the patient was not discharged home and was receiving intravenous antibiotics in the hospital.

Five weeks into the hospitalization, the patient was found injecting “drugs”. 48 hrs later he developed sudden onset chest pressure, had elevated troponin and was diagnosed with non-ST segment elevation myocardial infarction. Repeat echocardiogram showed severe RV systolic enlargement and dysfunction along with paradoxic septal motion of inter-ventricular septum. Estimated pulmonary artery systolic pressure was severely increased. Few hours later the patient’s oxygen requirements increased and his mental status worsened. Subsequently he was transferred to ICU.

Diagnostic work up at the time of transfer to ICU revealed WBC 27.2, Hb 10.1 g/dl, Platelet count of 33, potassium 5.6 mEq/L, Hco3 10 mEq/L, Creatinine 1.4 mg/dl, elevated liver enzymes: ALT 2890 IU/L, AST 3426 IU/L, Trop 0.89, Urine toxicology positive for opiates and tricyclics, INR 6.17, fibrinogen <60, D-dimer >20, procalcitonin 0.42, heparin induced anti platelet antibody negative, HIV negative, blood cultures were negative. Despite receiving maximal medical support with pressors, fluids, empiric antibiotics, the patient had PEA arrest. He was resuscitated with return of spontaneous circulation. However, he had multiple episodes of PEA requiring resuscitation and subsequently family has withdrawn the care.

The autopsy showed severe intravascular pulmonary talcosis with numerous perivascular deposits of refractile crystalline material with associated multinucleated giant cell reaction, eosinophilic infiltrates, fibrosis and vascular occlusion. There were also many intravascular crystalline particles entrapped within fresh thrombus material. Some of the larger vessels showed complete luminal occlusion with...
drug abuse can cause slow progressive symptoms, which may eventually lead to chronic respiratory failure, emphysema, pulmonary arterial hypertension, and cor pulmonale [20-22]. Intravenous injection of powdered medications can result in acute right ventricular failure secondary to obstructive shock similar to that seen in massive pulmonary embolism. Unfortunately there is no specific treatment in these acute cases, other than supportive care [5]. There are studies, which assessed the immediate pulmonary vascular hemodynamics following intravenous injection of crushed pills. These studies have shown an acute rise in pulmonary arterial pressure, pulmonary vascular resistance and persisted for the following 72 hours, 19 this can explain the acute right ventricular failure and cardiogenic shock observed in these patients.

However, only few such cases were reported in literature and were diagnosed only on autopsy. Autopsy studies in such patients revealed cardiac failure with pulmonary edema, cardiac arrhythmia secondary to pulmonary thromboembolus, multisystem organ failure secondary to sepsis, pulmonary fibrosis, and cardiopulmonary decompensation from pulmonary hypertension as the causes of death [2]. They were found to have increased lung weights and heart weight and dilatation of both right and left heart along with hepatomegaly and splenomegaly.

Though controversial, there have been reports in literature wherein, patients with intravascular talcosis and chronic pulmonary symptoms underwent lung transplantation however on long-term follow up, patients had recurrences, believed to be secondary to relapse of their drug abuse [15,23].

Conclusion

Though hospital is considered as a safe and secure place, possibility of intravenous drug abuse especially in patients with PICC lines while on ambulatory ward should be considered, especially in someone with unexplained hemodynamic collapse with history of intravenous drug abuse [4]. Deaths in such patients should be reported to coroner.

References