Chronic kidney disease is one of risk factors for TACO

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Introduction: Transfusion-related acute lung injury(TRALI)and transfusion-associated circulatory overload (TACO) are the leading causes of transfusion-related morbidity and mortality.These adverse events are characterized by acute pulmonary edema within 6 hours of a blood transfusion.
Cardiovascular risk factors predominate in TACO; patients tend to be older and frequently have a history of congestive heart failure and/or coronary artery disease. Renal impairment as reflected by a history of chronic kidney disease is also common in TACO,whereas acute kidney disease and liver failure are prevalent in TRALI.(1,2,3,4,)
Echocardiographics evidence of elevated cardiac filling pressures or systolic and/or diastolic dysfunction suggest circulatory overload and a diagnosis of TACO in the setting of pulmonary edema. In contrast, the absence of echocardiographic abnormalities is central to the diagnosis of TRALI.

The pathophysiology of TACO resembles that of other forms of acute cardiogenic pulmonary edema. Blood transfusion can rapidly increase left atrial and pulmonary capillary pressures, resulting in transudation of fluid into the pulmonary interstitium and alveolar space (ie, TACO). Blood transfusion is thought to increase oncotic and pulmonary capillary pressures more significantly than an equivalent volume of intravenous crystalloid fluid, potentially accounting for its designation from other mechanisms of circulatory overload. However, similar to other mechanisms, TACO frequently occurs in patients with preexisting cardiac (left ventricular systolic or diastolic) dysfunction or renal impairment who are unable to compensate for an increase in circulating blood volumes.2 In these chronic conditions, pulmonary capillary wedge pressures may be persistently elevated with expansion of lymphatic capacity and result in pulmonary edema when further increased with transfusion(5)

References:

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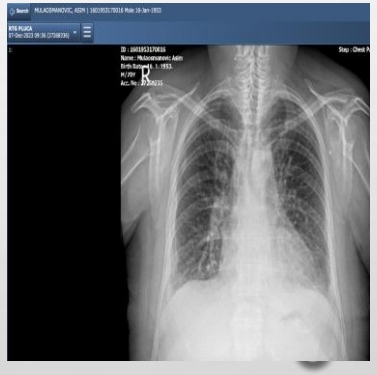
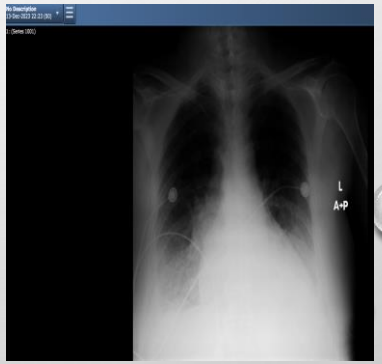
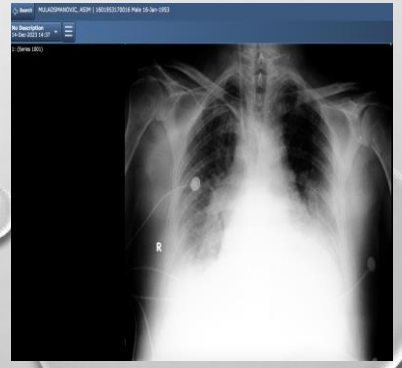
Case presentations: A 71-year old man was admitted for heart failure.He had a history of chronic kidney disease and ischaemic cardiomyopathy (ejection fraction 34%). He was treated with antibiotics and forced diuretic therapy, which was followed by an average diuretic response of about 2000 ml, but with a gradual progression of renal function parameters.
During hospitalization he was transfused for symptomatic anaemia, with haemoglobin (Hb) 69 g/L.and one dose of DE is prescribed,after the diuretic response is reduced by 50%.On the following day,a second dose of DE is prescribed,after the patient's clinical deterioration is monitored in terms of a decrease in hourly diuresis,now anuria and further progression of azotemia.
Echocardiographic,patient in the rhythm of atrial fibrillation,moderate MR and TR,with signs of severe pulmonary hypertension,and hypokinesis of the apex and medioapical segments of all walls(EFLV 34%)
Radiologically,inhomogeneous infiltative shadings of pl. parenchyma projections of the lower pl. fields on both sides and FC sinuses shaded on both sides,most likely by type pleyral effusions.
Considering the anuria that lasted for 24 hours and hypervolemia,CVVHDF is indicated and started, and a total of 2 CVVHDF treatments are performed.
Further,radiological and clinical regression of inflammatory and pathological changes in the lungs is monitored







1st day hospitalization; days after transfusion

Conclusion: Real-time identification of patients with increased risk of an adverse pulmonary transfusion event is essential. For example, clinical algorithm incorporating hemodynamic parameters or creatinine clearance could trigger recommendations for diuretic administration or alternatives to transfusion in patients at risk for TACO.