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THE DEADLY CLOTS

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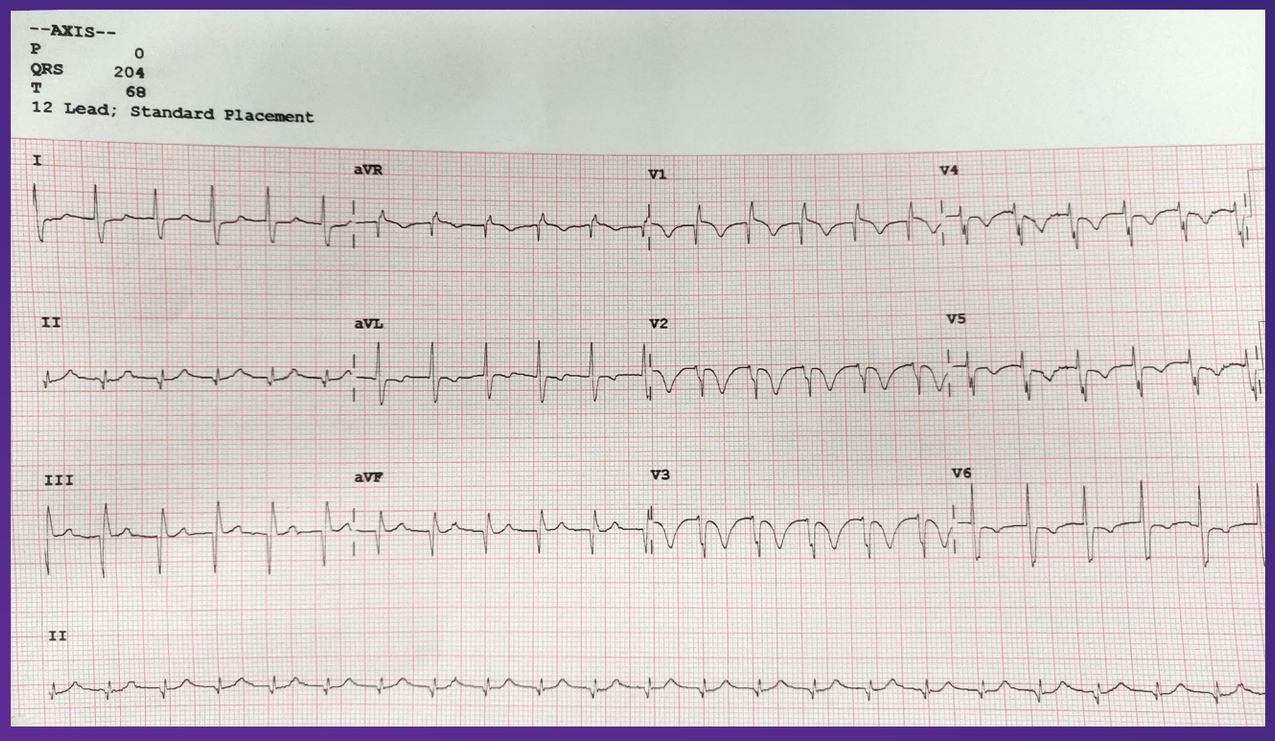
Introduction

Massive pulmonary embolism (PE) is a condition of acute PE with profound hemodynamic instability and evidence of right ventricular dysfunction. It is important to be well prepared, considering the increased risk of thromboembolism in COVID19 patients.

Case and Result

A 48-year-old man presented with 3-days history of chest pain and shortness of breath. He had no underlying illness and identifiable risk factors for PE. On examination, he was alert but tachypneic, tachycardic and hypotensive (Picture 1). Bedside echocardiogram showed a grossly dilated right ventricle (RV) with flattening of the septal wall and evidence of McConnell's sign. He was arranged for urgent computer tomography pulmonary angiography (CTPA). However, he had cardio-pulmonary arrest before any treatment was initiated. He was resuscitated and intra-arrest thrombolysis was administered. Despite our best effort, patient eventually succumbed to death.



Picture 1: Patient's ECG

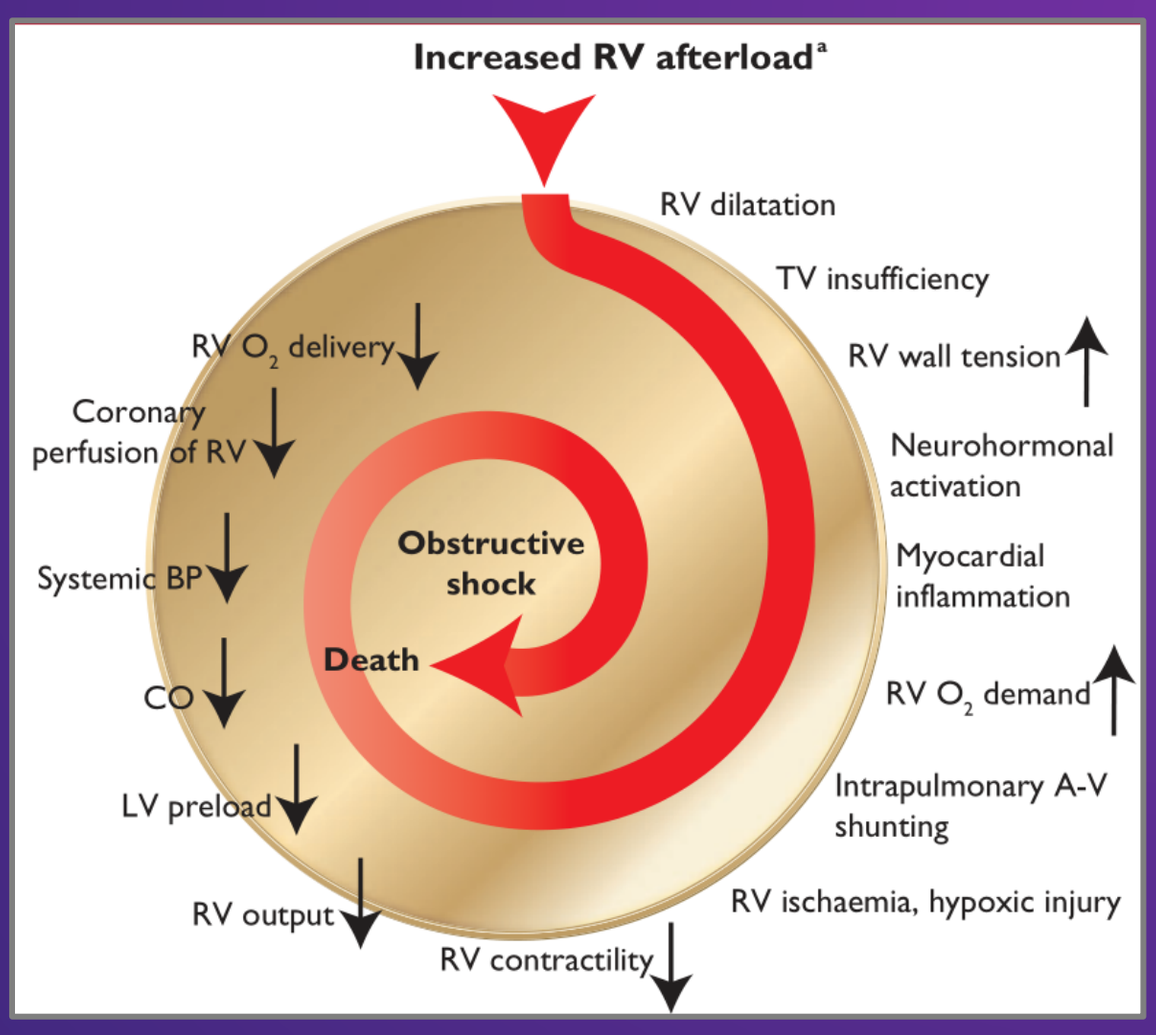


Figure 1: Vicious cycle in patient with massive PE

Discussion

Massive PE accounts less than 5% of acute PE cases. However, it can be fatal as early hospital mortality varies between 25-65%.1 The management of massive PE is incredibly complex. Early vasopressor support is recommended in hypotensive patients. Fluid challenge has the potential to over-distend the RV and ultimately causing the vicious cycle (Figure 1). Every attempt should be made to avoid intubation. If intubation is necessary, awake intubation is probably the preferred approach. CTPA is the imaging modality of choice for PE patient. However, for unstable patient, echocardiography provides vital information on the visualization of RV pressure overload and dysfunction.2 This can be enough to justify an emergency thrombolytic therapy. Streptokinase is readily available in most center and can be given loading dose intravenous 250,000 IU over 30 minutes, followed by 100,000 IU/h over 24 hours.[3] Other treatment considerations include administration of recombinant tissue plasminogen activator (rtPA), surgical embolectomy and percutaneous catheter-directed thrombolysis by interventional radiologist.[4]

Conclusion

The ultimate aim is to dissolve the embolic burden and improve RV function. Early involvement of multidisciplinary teams enables rapid identification and augmentation fine-balance of resuscitation in crashing pulmonary hypertension.

Reference

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