

AN UNUSUAL CASE OF SUPERIOR VENA CAVA SYNDROME

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Introduction:

Superior vena cava syndrome (SVCS) is a medical emergency, 80% of which is caused by malignant mediastinal tumours. However, non-malignant causes lead to 20% of cases of SVCS(1). We present an interesting case of SVCS after receiving ChAdOx1 CoV-19 vaccine (AstraZeneca).

Materials and Methods:

A 52-year-old man, presented with progressively worsening swelling of face, neck, chest and arms, pleuritic chest pain, abdominal pain, and breathlessness for 7 days. He had type 2 diabetes mellitus and was on Gliclazide, Metformin, and Sitagliptin. He received ChAdOx1 CoV-19 vaccine 5 weeks prior to his presentation. Physical examination showed a classical picture of superior vena cava occlusion with collateral vessels on the chest and reduced breath sound on the right base.

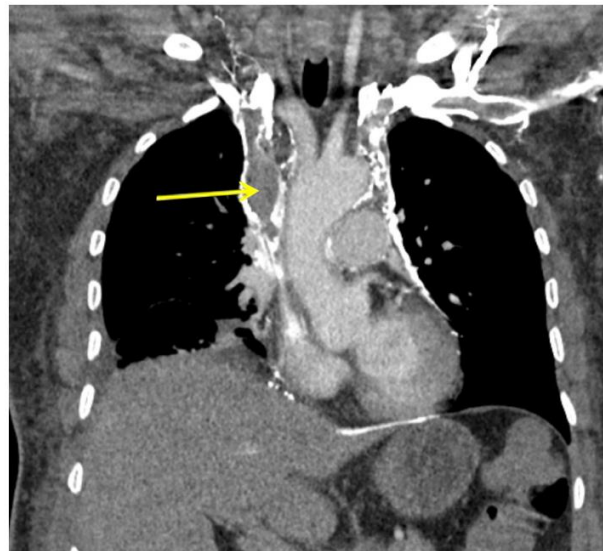


Figure: CT Thorax coronal view: yellow arrow showing the thrombus in the superior vena cava

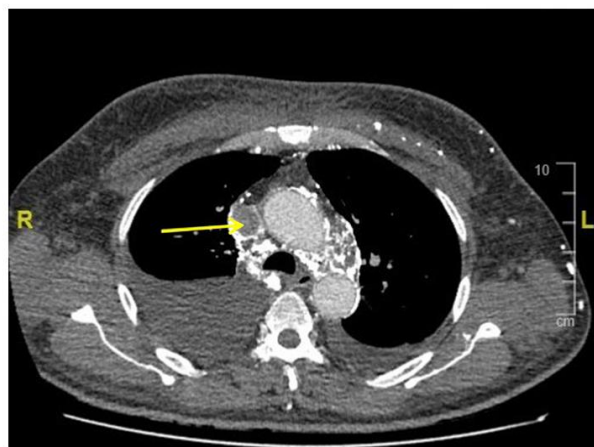


Figure: CT Thorax axial view: yellow arrow showing the thrombus in the superior vena cava

Investigations	Results
WCC	16.4x10 ⁹ /L
Neutrophil	13.53x10 ⁹ /L
Platelet	338x10 ⁹ /L
CRP	44mg/L
D-dimer	3.25ug/mlFEU
Fibrinogen	4.1 g/L
Prothrombin time, activated partial thromboplastin time, renal and liver function tests, C3, C4, IgG4 levels	unremarkable

Investigations	Results
Compression duplex ultrasound of upper limbs and neck	bilateral inferior jugular vein thrombosis, bilateral subclavian vein thrombosis extending into proximal axillary veins and superior vena cava thrombosis
CT neck, thorax, abdomen, and pelvis and CTPA	no evidence of malignancy, right sided pleural effusion, no pulmonary embolism.
Vasculitic screen, connective tissue disease screen, anti cardiolipin antibody, anti-beta2 glycoprotein antibody, lupus anticoagulant, PNH and JAK2 mutation	negative
Anti-platelet Factor4 ab (anti-PF4)	positive

He was managed with LMWH with good response and was switched to Apixaban with a follow up appointment in deep venous thrombosis clinic.

Results:

Patient's platelet count was persistently normal with a moderately raised D-dimer. Generally, patients with VITT presents within 5-30 days post vaccination and have thrombocytopenia (platelet count <150,000x10⁹ L), D-dimer >4 ug/mlFEU, positive anti-PF4 antibodies on ELISA and thrombosis (2). In VITT IgG antibodies that recognize PF4 bound to platelets leading to widespread platelet activation (3). Beside thrombosis and positive anti-PF4, our patient did not have any other features of VITT, although, in about 5% of patient with VITT can have normal platelet count (4). Moreover, in VITT cerebral vein, deep veins of the legs, pulmonary arteries and portal circulation are commonly affected by thrombosis (5) which did not occur in our patient, rather jugular, subclavian, axillary vein and superior vena cava were involved causing SVCS.

Conclusion:

Although exceedingly rare, VITT can be life threatening with a mortality rate of 22% (2) and thus highlighting the importance of not missing a diagnosis. This case report focuses on the fact that all cases of VITT might not have all the diagnostic features. Moreover, we think this is the first case report of VITT where patient presented with superior vena cava obstruction syndrome. Therefore, clinicians should be vigilant when patient presents with thrombosis in atypical site and has had a history of recent Covid-19 vaccination to avoid missing this life-threatening complication.

References:

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