Recurrent Thrombosis and Periprocedural Collapse in a case of Hereditary Antithrombin Deficiency: Implications for Diagnosis and High-Risk Thrombectomy Management

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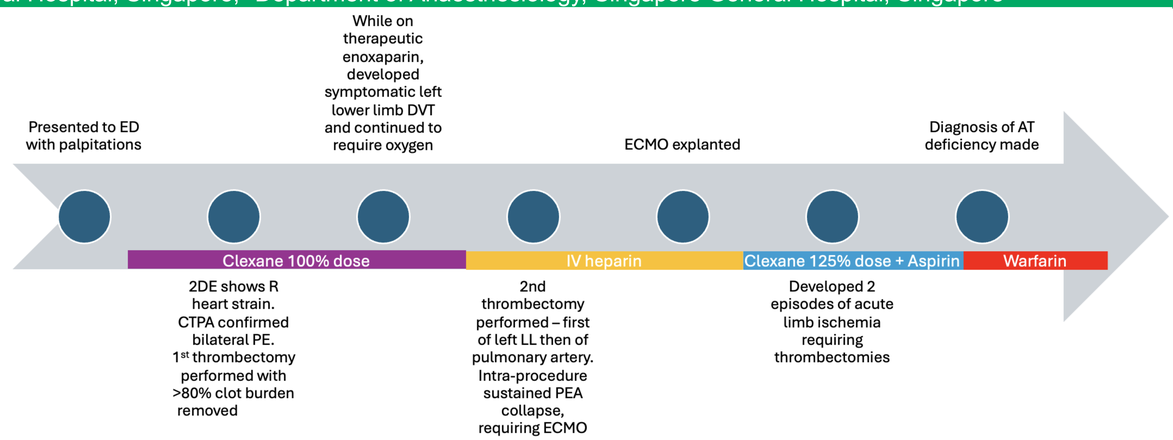
Background

Unprovoked venous thromboembolism (VTE) should prompt investigation for underlying pro-thrombotic disorders. Hereditary antithrombin (AT) deficiency is rare (0.02% prevalence[1]), and type I deficiency involves reduced AT levels (<80%), increasing the risk of VTE and heparin resistance.

Methods

Case Report: A previously healthy 17-year-old boy presented with sudden palpitations and tachycardia. Imaging revealed extensive bilateral pulmonary embolism (PE) and severe right heart strain.



Results

A 17-year-old boy presented with acute palpitations and tachycardia. CT Pulmonary Angiogram showed extensive bilateral main branch PE and severe right heart strain. Classified as an intermediate high risk PE, the pulmonary embolism response team recommended for a pulmonary thrombectomy. This removed >80% of the clot burden.

Despite therapeutic doses of enoxaparin, and initial clinical improvement, there was persistent hypoxia and an increasingly symptomatic proximal deep vein thrombosis. Decision was made for a lower limb venous thrombectomy and 2nd pulmonary thrombectomy. He had a cardiac arrest during the procedure and extracorporeal membrane oxygenation (ECMO) was started.

Whilst on ECMO, he was anticoagulated with IV heparin. Despite this, he developed an acute arterial thrombus in his left leg with worsening thrombocytopenia, which led to clinical concerns of heparin induced thrombocytopenia (HIT). Anticoagulation was switched to rivaroxaban. Another acute arterial thrombus in his right leg occurred, which required a second arterial thrombectomy.

By then, HIT was ruled out and patient was switched to enoxaparin dosed at 125% of body weight and aspirin.
In light of his recurrent venous and arterial thromboses, a thrombophilia screen was done and revealed type I AT deficiency (AT level: 42%). He was bridged to warfarin after these acute events, for which he remained thrombosis-free thereafter.

Conclusion

This complex case of progressive VTE despite anticoagulation invites discussion into the management options to be considered in these challenging scenarios, and the role of repeated thrombectomies and clinical issues in the periprocedural period. An important consideration will be to think of pro-thrombotic conditions which could render anticoagulation ineffective, including thrombophilia such as antithrombin deficiency that can cause heparin resistance.

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