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A Case of Catastrophic Antiphospholipid syndrome triggered by traumatic spinal cord injury

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Introduction

Antiphospholipid syndrome (APS) is a multisystem autoimmune condition characterized by vascular thromboses associated with persistently positive antiphospholipid antibodies (aPL). Catastrophic APS (CAPS) is the most severe form of APS with multiple organ involvement developing over a short period of time, usually associated with microthrombosis. We report a case of patient with traumatic spinal cord injury, who developed rapid course of complication due to underlying CAPS.

Case

A 41 years old tetraplegia man with diagnosed traumatic incomplete SCI (American Spinal Injury Association impairment scale C, neurologic level of injury at C3 cervical vertebra) was transferred to our department after receiving laminoplasty at C4-5-6 level with partial laminectomy (on 19th post operation day(POD) and 20th hospital day(HD). He had been receiving early rehabilitative intervention from POD 5, including ROM exercise of both upper and lower extremities twice a day, 5 days a week. At the time of transfer, there were 5 noticeable complications. 1) Fever of Unknown Origin with no definite routine laboratory evidence of inflammation, 2) Rapid development of large sized deep vein thrombosis (DVT) (Fig. 1.) with pulmonary thromboembolism (PTE) (Fig. 2.) that developed within 10 days and refractory to prophylactic administration of dabigatran, 3) Multiple atypical ulcerations in ascending colon and rectum (Fig. 3.), 4) Grade 3 pressure sore in coccyx, and 5) uncontrollable orthostatic hypotension that did not response to midodrine. At first, these problems were regarded as independently related post spinal cord injury complications that occurred at similar time coincidently. However, time course of the complications was abrupt and when considering patient's neurologic functional ability, the degree of these complications was vicious.

Results

To exclude possibility of combined systemic disorders, additional laboratory examination was performed and elevated anti B2-GPI IgG (34.5 U/mL) was detected. Based on diagnostic criteria, he was diagnosis as APS, warfarin was started at a dose of 2 mg per day, targeting international normalized ratio of 2-3. Moreover, fludrocortisone was administered for orthostatic hypotension. Then, the patient's fever was subsided and after 10 days warfarin therapy started, follow up CT showed resolving state of PTE and a recurrence of thrombotic events was not observed later. Symptom of autonomic dysfunction was improving that he was able to endure tilt table training up to 70 degrees.

Conclusion

The clinical manifestations of CAPS may develop rapidly leading to fatal systemic complications and may be challenging to make diagnosis without high index of clinical suspicion when accompanied with post spinal cord injury complications. To provide urgent treatment, possibility of underlying systemic diseases should not be missed when unexplainable rapid onset of vascular complications in SCI patients is encountered.

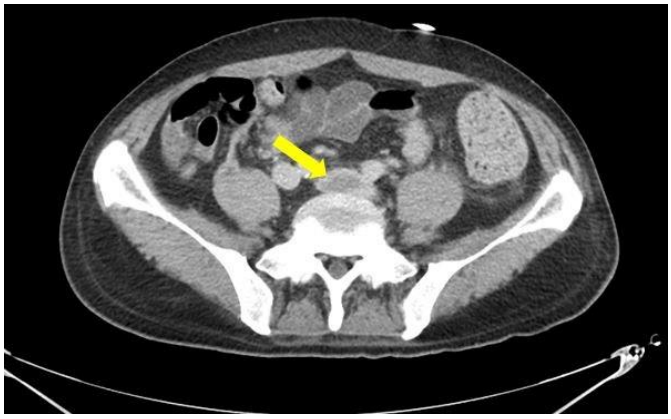

fig1. DVT CT with angiography showed a filling defect in left common iliac vein (arrow)

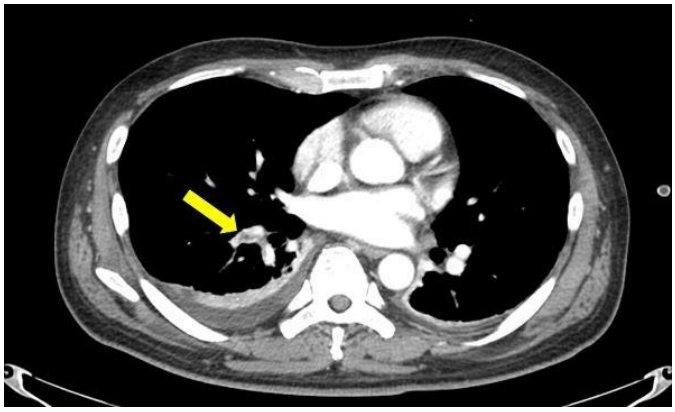

fig2. DVT CT with angiography showed a filling defect in right pulmonary artery (arrow)

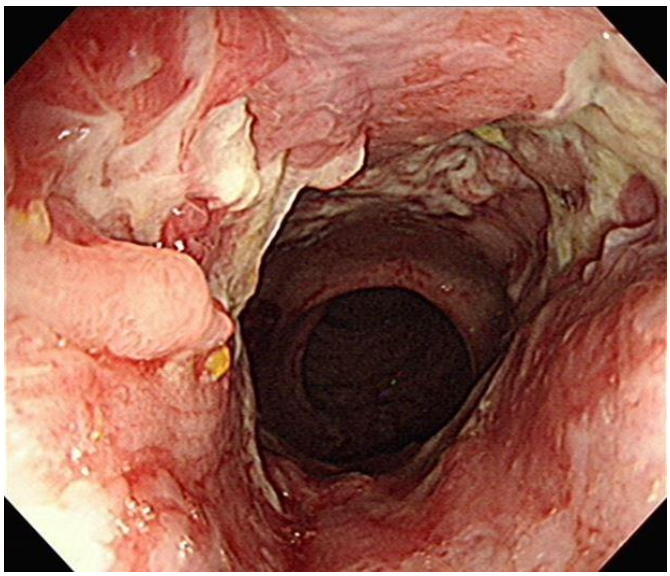

fig3. Colonoscopy showed multiple linear, circular, transverse and geographic patterns of ulceration