SPINAL CORD INFARCT AS A DELAYED COMPLICATION OF SUBARACHNOID HAEMORRHAGE

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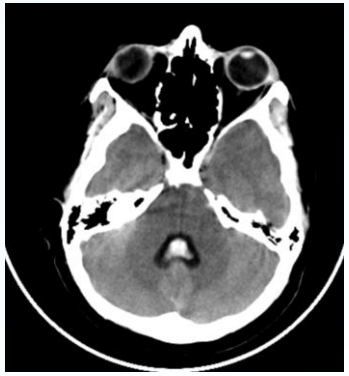
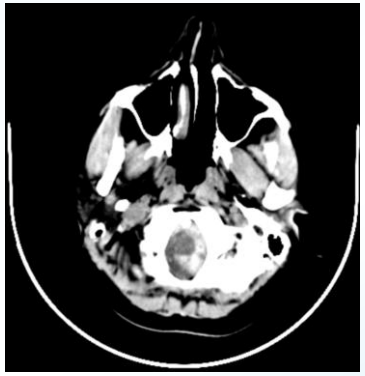
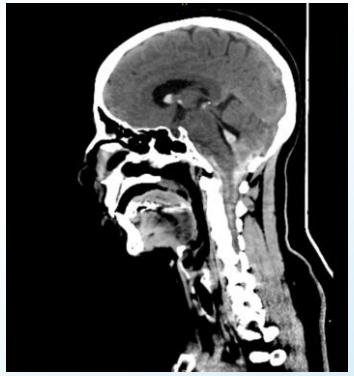
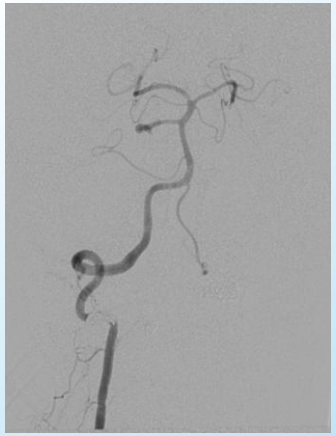
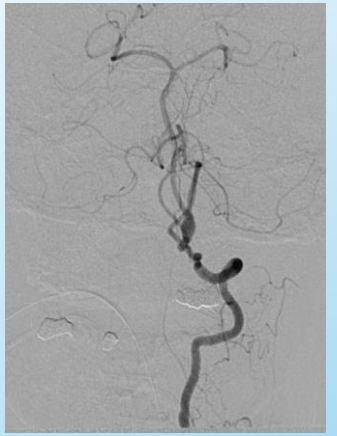
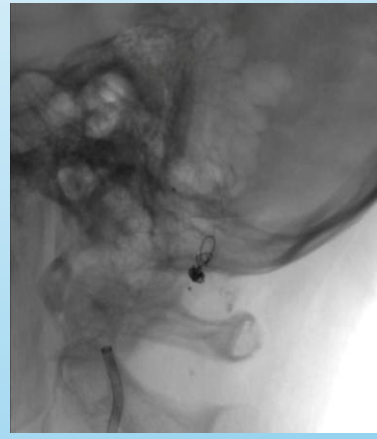
Introduction: Subarachnoid haemorrhage, often resulting from ruptured intracranial aneurysms, is a critical neurological event associated with significant morbidity and mortality. While cerebral vasospasm and delayed cerebral ischemia are well-documented complications of SAH, spinal cord infarction leading to acute quadriplegia remains an exceedingly rare and underrecognized sequela.

Aim:
To present a rare case of spinal cord infarction manifesting as acute quadriplegia following aneurysmal SAH, highlighting the diagnostic challenges and emphasising the importance of considering spinal vasospasm in the differential diagnosis of delayed neurological deficits post-SAH.

Case:

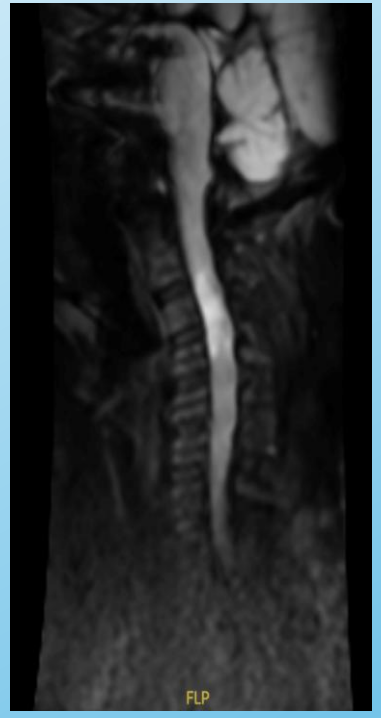
A 44-year-old woman presented with a sudden-onset severe headache. CT brain revealed SAH extending into the peri-medullary cisterns and upper cervical intraspinal spaces. CT angiography showed a fusiform aneurysm in the left PICA. She was managed conservatively and planned for coiling on a later date.

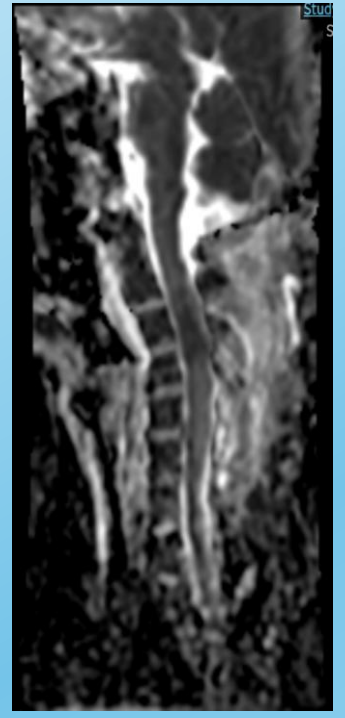
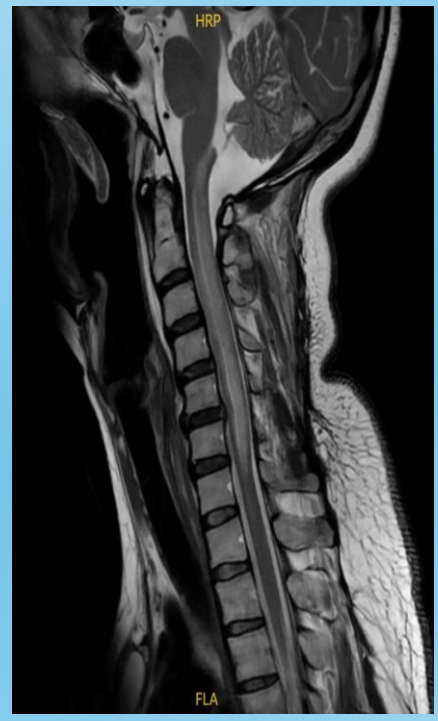







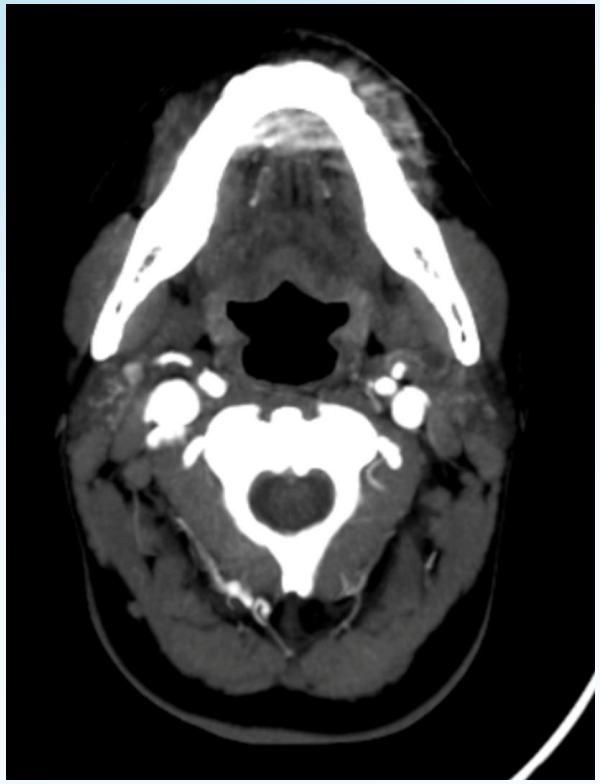
Endovascular flow diverter placement with coiling of the aneurysm was performed without any immediate complications. Twelve hours after the procedure, she developed sudden-onset quadriplegia.

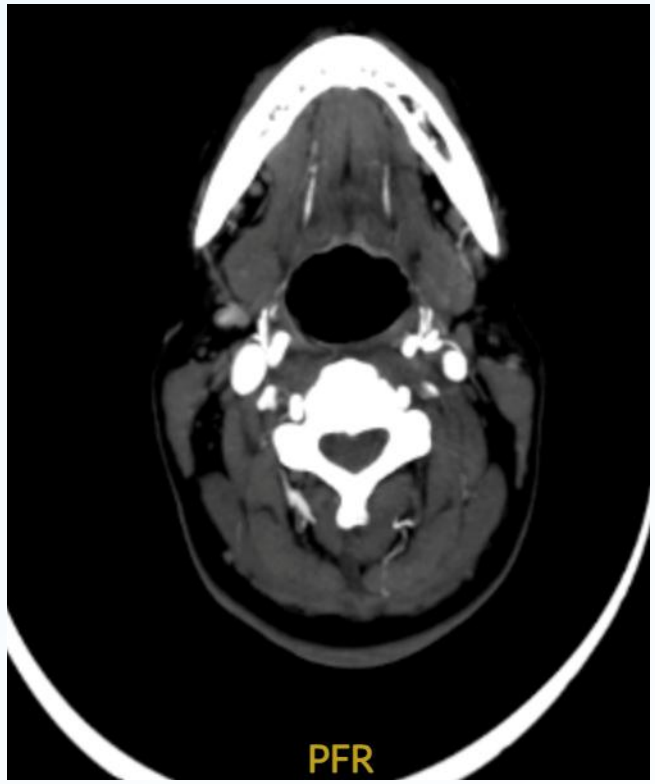
Imaging showed cervical spinal cord infarct with diffusion restriction at the C3 and C4 level and cord oedema extending up to the upper C7 vertebrae, indicating ischaemia. CT angio was showing decreased filling in the distal ASA .

The cause of the infarct was attributed to delayed vasospasm secondary to the initial SAH







Conclusion:
This case underscores the importance of recognizing spinal cord infarction as a potential delayed complication of SAH, even after seemingly successful aneurysm management. Clinicians should maintain a high index of suspicion for spinal vasospasm in patients presenting with new-onset motor deficits following SAH. Early identification and intervention are crucial to mitigate long-term neurological sequelae