Carotid artery pseudoaneurysm with Horner's syndrome: delayed complication of internal jugular venous cannulation

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INTRODUCTION

Internal jugular venous catheterization is associated with various complications. The complications can occur either during cannulation (Mastan et al, 2001), or while the catheter is *in situ* (Mastan et al, 2003). The author reports an unusual delayed complication which was noted a few days after removal of the central venous catheter.

DISCUSSION

Horner's syndrome consists of mild to moderate ptosis of the upper eyelid as a result of a paresis of Muller's muscle. Other findings of Horner's syndrome include miosis of the affected pupil and anhydrosis of the affected face. The sympathetic fibres from the posterolateral hypothalamus descend uncrossed through the brainstem and synapse with second-order neurons in the intermediolateral column of the spinal cord between the eighth cervical and the second thoracic vertebrae. These preganglionic fibres exit in the anterior roots, pass over the pulmonary apex and ascend in the neck in the cervical sympathetic chain, behind the common carotid artery and outside the carotid sheath, and end by synapsing in the superior cervical ganglion, located at the angle of the jaw.

The postganglionic fibres run in a plexus along the internal carotid artery to the cranium, entering the orbit to supply the smooth muscle of the levator palpebre superioris (Muller's muscle) and the dilator pupillae muscle (Reddy et al, 1998). It was found that 10% of the causes for Horner's syndrome were iatrogenic, mainly resulting from neck surgery and carotid angiography (Maloney et al, 1980).

Although catheterization of the carotid artery is a well recognized cause of Horner's syndrome, internal jugular vein cannulation has been less frequently reported as a cause. Horner's syndrome was noticed on the side where the patient had internal jugular venous cannulation. There was a swelling to the neck at the site of needle puncture for internal jugular cannulation. It was not tender nor had he complained of ipsilateral headache. Contrast computed tomography scan and ultrasonograghy of the neck revealed it as carotid artery pseudoaneurysm. Horner's syndrome developed a few days after the internal jugular venous cannulation. Retrospectively, it can be hypothesized that during internal jugular venous cannulation, the carotid artery might have been damaged. This might be a result of the cannula perforating the artery, or damaging the wall of the artery during repeated attempts. Consequently, this resulted in a pseudoaneurysm. The size of the aneurysm was significant enough that it had disrupted the oculosympathetic pathway and resulted in Horner's syndrome.

The incidence of carotid artery puncture depends on various factors: the clinical skill of the performer, position of the head and the technique used. The increased overlap of carotid artery and internal jugular vein with head rotation of more than 40º increases the risk of inadvertent puncture of carotid artery (Sulek et al, 1996). Internal jugular vein frequently collapses with needle insertion. This may result in puncture of the posterior wall of the vessel and then the carotid artery when two vessels overlap (Sulek et al, 1996). There is evidence to support that the central venous catheterization under two-dimentional ultrasound guidance is quicker and safer than the landmark method (Hind et al, 2003).

CONCLUSIONS

Carotid artery pseudoaneurysm and Horner's syndrome appeared a few days after removal of the catheter.

Consideration should be given to the reduction of the arterial injury by using

CASE REPORT

A 58-year-old man presented to hospital with a 1-week history of left arm swelling. Two days after admission he became pyrexial. Chest X-ray revealed a right-sided effusion, which was an empyema. He further deteriorated and developed respiratory failure. He was intubated and ventilated. Right internal jugular venous cannulation was conducted to allow central venous pressure monitoring. He was transferred to intensive care, where he gradually improved and was weaned off ventilation.

The right internal jugular venous cannula was removed without difficulty 9 days later. Four days after removal of the internal jugular venous catheter, miosis and moderate ptosis on the right side (Horner's syndrome) was noticed. The remainder of the cranial nerves were intact. A computed tomography scan (CT) of the head and neck was done to investigate the cause of the right Horner's syndrome and the left arm swelling. The CT scan (with contrast) showed an enlarged carotid artery (*Figure 1*). Ultrasonography showed this to be a 4 cm x 2.5 cm false aneurysm. The carotid artery pseudo aneurysm corresponded to the site of internal jugular vein cannulation. The scan did not reveal a cause for the swollen left arm, which was resolved gradually. Later he was referred to the vascular unit where he underwent angiography and successful stenting of the aneurysm.

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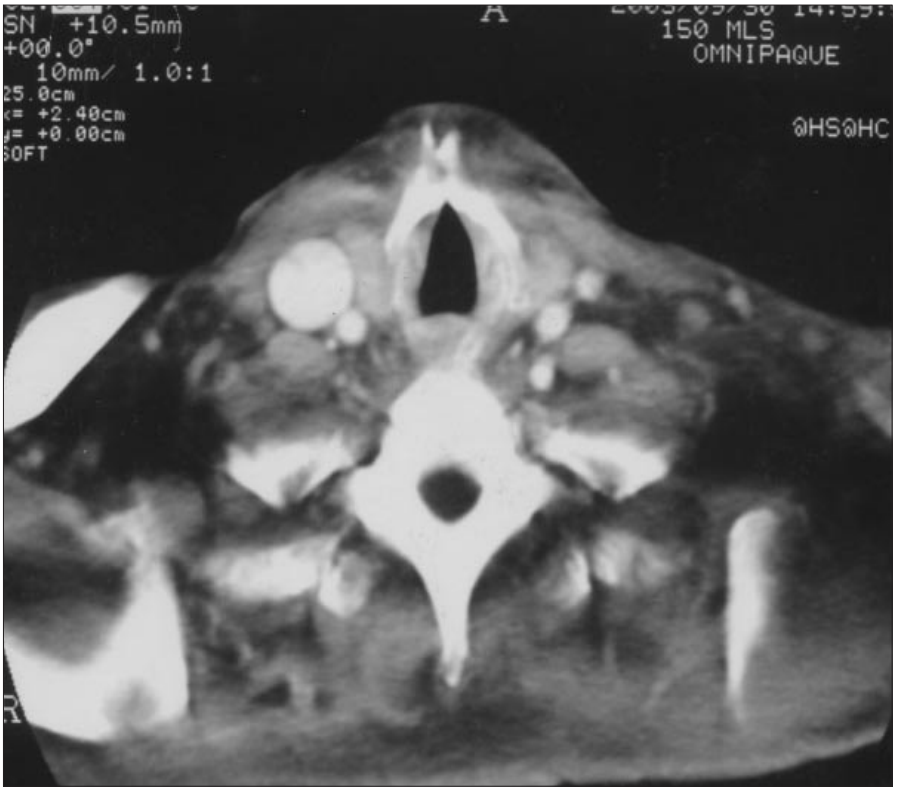

Figure 1. Computed tomography scan showing right carotid artery pseudoaneurysm.

ultrasound guidance during insertion, and the head should be in as neutral a position as possible, with less than 40 degrees head rotation.

Arterial injury and Horner's syndrome should be mentioned when consent is obtained. It is important to follow up the patients for any evidence of delayed complications even after removal of the central venous catheter. **HM**

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Correspondence

Polymyalgia rheumatica: beware of systemic presentation

Sir,

A 76-year-old woman was referred to medical outpatient by her general practitioner (GP), with a 3-month history of weight loss, loss of appetite, dry cough and night sweats. She had a mild anaemia (haemoglobin 10.2 g/dl, mean corpuscular volume 84), raised erythrocyte sedimentation rate (ESR) (107 mm/hour), normal liver and thyroid function test, normal chest X-ray and a negative serum electrophoresis.

She admitted to experiencing a deep aching pain in her shoulders, but denied any morning stiffness, jaw claudication, visual disturbance, joint swelling and/or pain, or a change in bowel habit. Past medical history included stable coronary artery disease. Physical examination was unremarkable. Further investigations included a negative autoantibody screen, negative antineutrophil cytoplasmic antibody, and a negative rheumatoid factor. Serum ferritin was high, but serum B12 and red cell folate assays were normal. ESR remained elevated at 107 mm/hour and creatinine kinase was normal.

Clinical diagnosis of polymyalgia rheumatica was made and prednisolone, 15 mg once daily, was started. Her GP was advised to monitor ESR and her clinical condition closely, and to continue prednisolone in case of positive therapeutic response. At her 2-month hospital follow-up, she described herself as being 'on top of the world'. ESR was now 17 mm/hour and haemoglobin 12.7 g/dl. Her appetite had normalized and weight had increased.

Clinicians need to maintain a high index of suspicion to diagnose cases of polymyalgia rheumatica presenting predominantly with systemic features, known to occur in one third of all cases. Patients may present with anorexia, weight loss, malaise, fatigue, depression, low-grade fever and mild anaemia. Exclusion of underlying malignancy and infection is mandatory prior to treatment with steroid.

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