



Love bites—an unusual cause of blunt internal carotid artery injury

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We describe an unusual case of blunt neck trauma leading to internal carotid artery (ICA) thrombosis and subsequent cerebral ischaemic infarct in a 44-year-old Māori woman.

Case report

A 44-year-old Māori woman presented to the Emergency Department 24 hours after developing sudden onset of left upper limb monoparesis. The weakness was initially mild and occurred while the patient was watching television. It had progressed to moderate weakness with functional impairment precipitating the admission.

On presentation normal vital signs and a Glasgow Coma Scale (GCS) of 15/15 were recorded. Neurological examination revealed moderate upper motor neurone weakness in left upper limb. The remaining examination was normal. She was in sinus rhythm with clinically normal heart sounds.

A small vertical bruise was noted in the right anterior neck, superficial to the upper third of the sternocleidomastoid muscle. This was attributed to a love bite with the minor trauma occurring several days prior to the onset of neurological symptoms.

The patient has a history of systemic lupus erythematosus (SLE) controlled with combination of steroids and methotrexate. She smokes tobacco with occasional use of marijuana.

She was admitted to hospital and treated for acute ischaemic stroke. A non-contrast computer tomography (CT) of her brain was normal. Carotid duplex Doppler revealed an echogenic material thought to be thrombus in the right ICA causing a 65 to 70% luminal stenosis (Figures 1a and 1b). CT angiography of her cervical vessels confirmed a partially occlusive luminal thrombus in the right proximal ICA without arterial dissection (Figure 2a).

She was commenced on intravenous heparin and a follow up repeat CT angiography seven days later revealed significant reduction of the thrombus size (Figure 2b). The patient's neurological symptoms resolved during her admission.

Warfarin was commenced with a target international normalised ratio (INR) of 2 to 3 before a thrombophilia screen was performed. At 1 month follow-up she has remained free of neurological sequelae. She is to continue warfarin for a total of 3 months.

Figure 1a. Longitudinal view of right proximal ICA (the luminal thrombus indicated by white arrow)

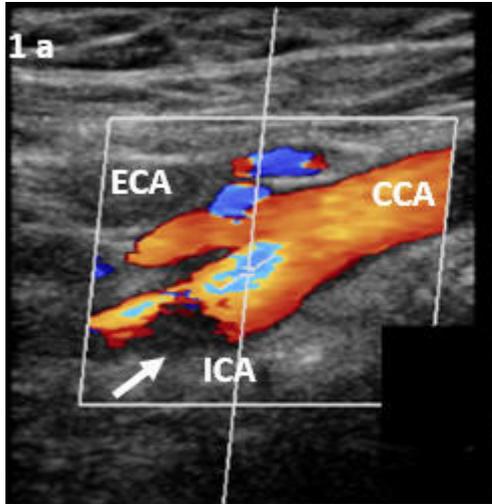
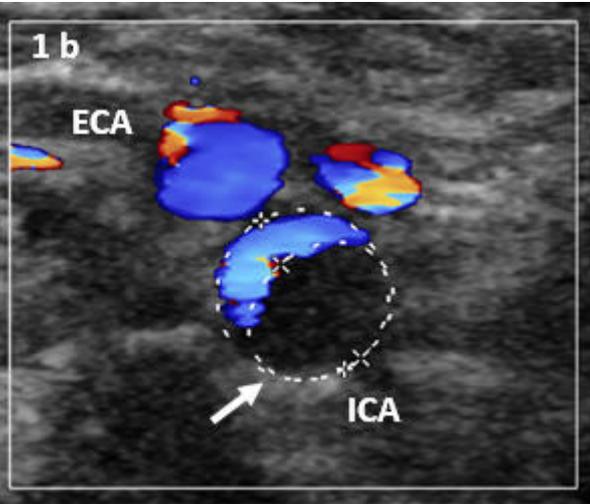


Figure 1b. Transverse view of the carotids (the right ICA and thrombus are marked by the dotted circular lines; thrombus is indicated by the white arrow)



Abbreviations: ECA: External carotid artery; ICA: Internal carotid artery; CCA: Common carotid artery.

Figure 2a. Initial axial CT angiography with filling defect in the right ICA (shown by black arrow)



Figure 2b. Follow-up CT angiography showing reduction in thrombus size



Discussion

Blunt carotid artery injury with subsequent thrombosis and stroke is considered a rare phenomenon. It was first described by Verneuil in 1872 but remains an uncommon diagnosis even in the setting of trauma.¹

Recent case series suggested an overall incidence of up to 1% in blunt trauma victims.¹ Majority of patients in case series have carotid injuries resulting from rapid deceleration with neck hyper-extension/flexion, predominately caused by motor vehicle accidents. This causes bony compression of carotid artery;² other mechanisms of injury are intra-oral trauma, direct blows and basal skull fracture.³ The injury results in traction on carotid artery leading to tearing of intima or media with platelet aggregation, thrombosis and cerebral embolisation.

Diagnosis requires a high index of suspicion as there is usually a latency from the initial trauma to onset of neurological deficit. Over 90% of patients present with symptoms at least greater than 1 hour from trauma and over a third with a delay of 24 hours or more.⁴ Diagnosis is often delayed due to absence of initial neurology or lack of superficial signs of injury. The delay in onset of neurological symptoms is thought to be due to progressive thrombosis causing hypoperfusion or subsequent embolisation.^{3,4}

Blunt carotid artery injury should be suspected in the following clinical circumstances: neurological examination incompatible with CT findings; physical signs of anterior neck injury; development of neurological deficit after hospital admission; presence of Horner's Syndrome.⁵

CT angiography or Magnetic Resonance angiography are diagnostic tools of choice, as they are non-invasive and allow assessment of the wall and lumen of the vessel.

Treatment options include surgical revascularisation, anticoagulation or supportive medical management.⁶ Earlier reports advocated surgical revascularisation⁶ but recent research have lent towards conservative management with anticoagulation, particularly when mild to moderate neurological deficits are present.⁸ Patients treated with anticoagulation seem to also achieve good clinical outcome.

Earlier reports indicate a mortality rate of up to 30%⁴ but more recently rate as low as 5% has been observed⁷. This is likely due to earlier diagnosis with advent of non-invasive imaging such as MRI or CT angiography leading to earlier intervention.

Our patient suffered an uncommon mode of blunt trauma to the neck and internal carotid artery following a love bite. There was no evidence of arterial dissection but most likely there was intimal injury from the compression of the internal carotid artery with subsequent thrombosis.

Conclusion

We report on an interesting case of blunt carotid artery trauma caused by a love bite. Blunt carotid artery trauma can lead to arterial injury, thrombosis and cerebral embolisation. It is a rare phenomenon and diagnosis requires a high index of suspicion. Current literature suggests a conservative approach to treatment with anticoagulation. Most patients achieve a favourable outcome especially when initial neurology was non-disabling.

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