

HEMIPLEGIA FOLLOWING MANDIBULAR FRACTURE

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Summary. The development of neurological symptoms incriminating one cerebral hemisphere following facial and/or mandibular injuries may be due to traumatic occlusion of the internal carotid artery, which may occur without apparent bruises in the neck.

Introduction

Traumatic occlusion of the internal carotid after a seemingly straightforward facial and/or mandibular fracture is rare. It is often misdiagnosed and has a high morbidity and mortality (Fleming & Petrie, 1968).

Case report

F. H., a 19-year-old girl, was a front-seat passenger in a car involved in a collision. She sustained several head and facial injuries including a bruise on the left side of her neck. She was rendered unconscious for approximately six hours. Radiological examination revealed a left middle third fracture of the facial bones and a fracture

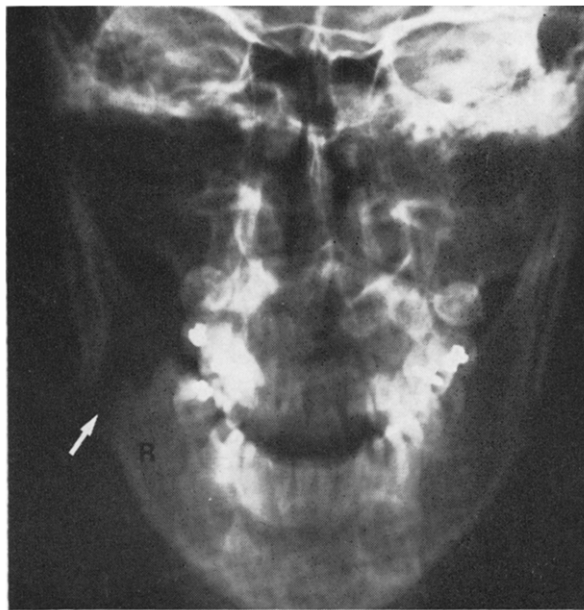


FIG. 1 Fracture right horizontal ramus of the mandible (arrow).

(Received 26 October 1978; accepted 14 December 1978)

of the right side of the mandible (Fig. 1). In hospital her level of consciousness steadily improved. Within 24 hours, she was able to get up to the washroom and her neurological examination was normal.

Approximately 36 hours later, she was found lying in bed with a total flaccid left hemiplegia and a left homonymous hemianopsia. She was drowsy but easily aroused. Her pupils were equal and reacted sluggishly to light, but her fundi were normal. Repeated recordings of her pulse, blood pressure and respiration showed no significant abnormality.

To exclude the possibility of a subdural haematoma, a computed tomography (CT) scan was performed. This showed an area of massive infarction in the right cerebral hemisphere (Fig. 2). Carotid angiography showed traumatic occlusion of the right internal carotid artery in the neck below the level of the mandibular fracture (Fig. 3). This was not treated surgically. The patient made a slow recovery and was discharged to a Rehabilitation Centre where she remained for six weeks.

Her latest follow-up was two and a half years after her accident. She was found to have a persistent hemianopsia and impairment of sensations to all modalities on the left side. Her motor weakness had improved and she could walk unaided.

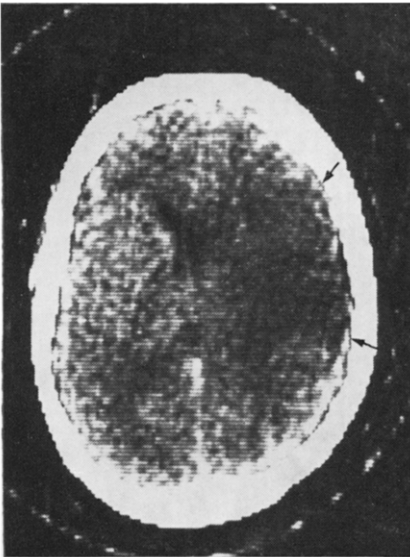


FIG. 2 (above). CT scan showing an area of diminished X-ray absorption in the right frontal and parietal lobes (arrows). There is slight displacement of the lateral ventricles to the left side, consistent with cerebral infarction.



FIG. 3 (right). Complete occlusion of the right internal carotid artery (arrow) opposite the upper surface of the fourth cervical vertebra. Note that the transverse shut-down of the vessel is unlike that seen in the majority of occlusions from other causes.

Discussion

In the present case, as in many of those published before (Hockaday, 1959; Miller & Ayers, 1967; Fleming & Petrie, 1968; Olafson & Christoferson, 1970; Jernigan & Gardner, 1971; Berman, 1975; Garrison & Ristuccia, 1975; Beatty, 1977), it appears that there is a uniform clinical pattern in traumatic occlusion of the internal carotid artery. The patient, who is often a young healthy adult, sustains a relatively minor trauma, usually in a car accident and may suffer from an apparently simple facial and/or mandibular fracture. After an initial period of concussion, the patient becomes alert and responsive. Neurological examination at that stage may reveal no focal cerebral abnormality. Few hours later, depending on the rate of thrombus formation or embolisation, a deteriorating level of consciousness is noted, motor and sensory weakness develop and the patient may become dysphasic. The duration of the lucid interval varies from one to 24 hours, but may be 48 hours or longer in few cases (Fleming & Petrie, 1968; Little *et al.*, 1969; Freidenberg *et al.*, 1973). Almost invariably, a diagnosis of epidural or subdural haematoma is made, but angiography reveals an occluded internal carotid artery. Few, yet important clinical observations have been made which help in the differential diagnosis. Olafson and Christoferson (1970) stated that the syndrome of carotid occlusion is characterised by a degree of responsiveness which is out of proportion to the patient's neurological deficits. Horner's syndrome due to damage of the sympathetic chain in the neck, may also be present in some cases (Fleming & Petrie, 1968).

Angiography is the examination of choice that establishes the diagnosis. Other non-invasive procedures that may be helpful and may reveal the extent of cerebral infarction are computed tomography (CT) and radionuclide brain scintigraphy (RNBS). On CT, cerebral infarction appears as a wedge-shaped, dark-grey low-density area involving the grey more than the white matter and may be associated with displacement of the ventricular system, as shown in the present case. RNBS reveals lack of cerebral perfusion on the dynamic study performed within few seconds from the intravenous injection of the radioactive isotope and its accumulation within the necrotic tissue at a later stage.

Regarding the mechanisms that lead to traumatic occlusion of the internal carotid artery, Fleming & Petrie (1968) have identified four types of injuries; direct blow to the neck, acute hyperextension of the neck, injury to the paratonsillar area in children who may fall on a foreign object carried in the mouth, and fractures of the skull base transecting the carotid canal. The first type is the most common and is the least difficult to diagnose if the possibility of carotid occlusion is remembered. The second type is much more difficult, because it is a 'whiplash' injury with no external evidence of trauma. How it leads to vascular damage is elegantly explained by Little *et al.* (1969). As a result of acute hyperextension or lateral neck flexion, the vessel is stretched, particularly opposite the lateral masses of the atlas and axis which project forward from the line of the transverse processes of the lower cervical vertebrae. The intima, being less elastic than the outer layer of the arterial wall, suffers most. An intimal tear is produced, usually about the level of the third cervical vertebra, leading to dissection or an intimal flap and thrombus formation. The adventia may remain intact without any visible breach of the artery (Murray, 1957). An observation which, was documented as early as 1872 by Verneuil, who made the first report of traumatic occlusion of the internal carotid artery (Little *et al.*, 1969). Other cases of extracranial internal carotid artery injury due to non-penetrating trauma have been recently reported (Chakera, 1979).

Acknowledgement

I am indebted to Dr Andrew Talalla for the clinical history of the case provided.

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