

Traumatic blunt carotid injury: clinical experience and review of the literature

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To evaluate the symptoms, the associated lesions, the treatment and the outcome of patients with blunt carotid injury (BCI), we reviewed the records of all patients admitted to our intensive care unit with head trauma between May 1991 and May 1995. A patient's assessment included the commonly used severity scores and cranial computed tomography (CT). Other diagnostic investigations were performed according to the clinical setting. Four patients (2 males, 2 females, age 29 ± 13 years) out of 145 were diagnosed to have BCI. At admission, the Glasgow Coma Scale (GCS) was ≥ 12 in all patients, and was associated with hemiparesis in three of them; the fourth became paretic 48 hours later. No pathological elements were demonstrated at the initial CT scan, whilst subsequent examinations showed signs of ischaemia after a variable interval from admission. In every patient the radiologic investigations demonstrated a thrombotic obstruction of the internal carotid artery (ICA), associated with an intimal dissection in two cases. Three patients were discharged with only minor neurologic symptoms. The fourth patient was referred to our ICU after the development of a massive hemispheric infarction, and died 3 days after admission.

Keywords: blunt head trauma; neck trauma; arterial thrombosis; arterial dissection; cerebral infarction; anticoagulation

INTRODUCTION

Whereas the carotid artery is commonly involved in stab or gunshot wounds in the head or neck, the occurrence of blunt carotid injury (BCI) is infrequent. Different kinds of injuries have been identified, including complete arterial disruption, thrombosis, carotid-cavernous fistulas (CCF), pseudoaneurysms and dissection of the vascular wall (Fabian *et al.*, 1990; Cogbill *et al.*, 1994). Mortality rate depends also on coexisting lesions, and ranges from 11 to 40% (Perry *et al.*, 1980; Cogbill *et al.*, 1994). For the same reason, long-term neurologic disability is variable, and depends on age, pretreatment neurologic conditions, and the results of treatment (Cogbill *et al.*, 1994, Li *et al.*, 1994).

After obtaining the approval of our Institutional Review Board and the permission of the patients or their next of kin, we reviewed the medical

records of all patients admitted to our intensive care unit (ICU) with the diagnosis of BCI during 4 years. In particular, we were interested in the patients' conditions at admission, estimated with the Glasgow Coma Scale (GCS) (Teasdale and Jennett, 1974), the Injury Severity Score (ISS) (Civil and Schwab, 1988) and the Simplified Acute Physiology Score II (SAPS II) (Le Gall *et al.*, 1989); the timespan elapsing between the trauma and the onset of neurological symptoms; the findings demonstrated by computed tomography (CT) and/or by other imaging techniques; the mechanism of trauma; the associated injuries; and the long-term neurologic outcome.

CASE DESCRIPTIONS

Case no. 1

A 17-year-old man was admitted to the orthopaedic ward for a right clavicular fracture received during a fall from a bike. At admission, the patient was fully alert; other neurological symptoms were absent (GCS 15), and the overall physical condition was good (SAPS = 2, ISS = 6). Four hours later, a right hemiparesis appeared, associated with a reduction in consciousness. The

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cranial CT was negative but a cerebral digital subtraction angiography (DSA) demonstrated an obstruction of the left internal carotid artery (ICA), localized 3 cm distally from the bifurcation. Blood was effectively supplied to the affected hemisphere through the anterior communicating artery. The patient was then operated on and, after the removal of a clot, an intimal tear appeared. The dissected area was replaced with an autologous saphenous bypass and, after the intervention, the patient was moved to the ICU. Intravenous heparin was started, but, despite the anticoagulative treatment, a DSA performed 3 days after the intervention demonstrated thrombosis of the bypass. A CT obtained on the same day showed no changes, apart from a small right parietal hypodensity. The patient was then transferred to the rehabilitation unit with a left hemiparesis. Two years after discharge, the patient had a left-sided epileptic fit, and since then he has been treated with anti-epileptic drugs; 4 years after the trauma, the patient still suffers from a slight left upper limb paresis.

Case no. 2

An 18-year-old woman reported a left bleeding tonsillar injury after a motorcycle accident. At admission, the patient was fully alert and in good condition (GCS = 15, SAPS = 2, ISS = 3). A tonsillectomy was then performed. During the intervention, the pharyngeal wall appeared normal. Seven hours after admission, the patient was confused, and a right hemiparesis and aphasia were present. The patient was then transferred to our ICU, and a contrast-enhanced CT of the neck revealed a thrombosis of the left ICA which extended from its origin up to the middle cerebral artery (MCA) (Fig. 1). Gas bubbles were present in the homolateral pharyngeal space. Aspirin and intravenous heparin associated with mannitol were then started. Despite this treatment, the CT scan performed on the following day demonstrated a hypodensity involving the whole left hemisphere; on DSA the left ICA was totally obstructed and possibly dissected (Fig. 2). The clinical course was unremarkable and the patient was transferred to a rehabilitation unit with a residual right hemiparesis. Three weeks after admission, a further DSA demonstrated the patency of the MCA, even if the ICA remained partially thrombosed. Two years after the event, the residual complaints were reduced strength in the left arm and a mild dysphasia.

Case no. 3

A 42-year-old woman was transferred to our intensive care unit from another hospital following an accidental fall in which she suffered the



Fig. 1. Cranial CT scan showing the lack of contrast in the left internal carotid artery (indicated by the arrow). The internal jugular vein is patent. There are small air bubbles in the left parapharyngeal space.

fractures of the right orbit and maxillary bones and multiple right rib fractures (SAPS = 2, ISS = 26). The patient was alert (GCS = 15), right hemiparetic and complained of diplopia. The CT scan demonstrated a diffuse brain oedema, and mannitol was then started. Two days later, after the transfer to the neurological ward, a colour-Doppler scan and a magnetic resonance angiogram (MR-ANG) revealed a reduced flow in the left ICA. The finding was considered compatible with an arterial dissection, and oral ticlopidine and subcutaneous heparin were given. The MR scan also demonstrated two cortical contusions, localized in the right frontal lobe and in the left occipital lobe, respectively. Both contusions were surrounded by perifocal oedema. Ten days after the trauma the patient was moved to another hospital. However, 1 month later she had a Jacksonian fit in the right arm, which subsided with the administration of carbamazepine. One year after discharge, the patient is free of symptoms, but still on anti-epileptic treatment.

Case no. 4

A 39-year-old soccer player was admitted to the neurological department with aphasia, right hemiparesis and a right > left anisocoria, which

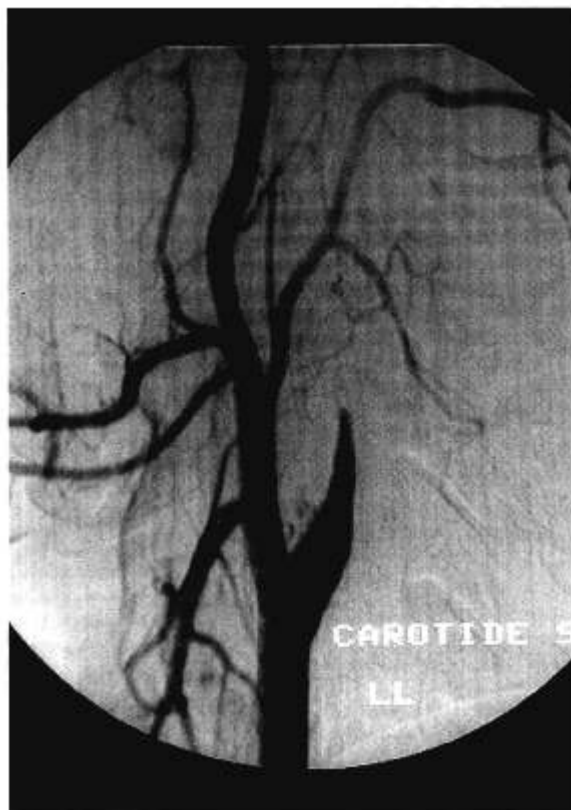


Fig. 2. Digital subtraction angiography of the left internal carotid artery, showing the progressive narrowing of the lumen, up to its complete obstruction.



Fig. 3. Digital subtraction angiography of the left internal carotid artery, showing the progressive, subtotal narrowing of its lumen.

arose during a football match. Initial evaluation included a GCS of 12 (SAPS = 3, ISS = 18). His friends reported that a trivial head and neck trauma occurred 3 weeks before admission. The CT scan demonstrated a slight asymmetry of the left ventricle, without any shift of the midline, and the patient was given intravenous mannitol associated with subcutaneous heparin and oral ticlopidine as a standard anti-thrombotic regimen. The next day, the hemiparesis worsened even though the patient's GCS remained 12. In the following 3 days, repeated CT scans demonstrated an ever-increasing diffuse left hemispheric oedema. At the same time, the hemiparesis further worsened, in association with a rapidly progressing reduction of consciousness and a left sluggishly light-reacting mydriasis. A transcranial Doppler scan demonstrated the absence of blood flow in the left MCA and in the carotid siphon, and the DSA demonstrated narrowing and possible dissection of the left ICA lumen (Fig. 3). The following day, the patient became comatose (GCS 3–4, SAPS 19) and was transferred to our ICU, where he was intubated and mechanically ventilated, under heavy barbiturate sedation. The CT scan showed a massive left hemispheric infarction

with a 1.5 cm-shift of the midline and the disappearance of the basal cisterns (Fig. 4). A fibre optic indwelling catheter was advanced into the left jugular bulb in order to monitor the saturation of the jugular blood (SjO_2), and intravenous heparin was started. At the same time, fluids were administered through another catheter inserted in the contralateral internal jugular vein. Despite this aggressive treatment, the $SjVO_2$ rose from 70 to 98% in the next few hours, and the patient died 3 days after admission to the ICU. At autopsy, a massive left hemispheric infarction was found, in the absence of major intimal lesions of the left ICA and abnormalities of the Willis circle. Instead, a complete thrombosis of the basal venous sinuses which extended bilaterally to the jugular veins was found.

DISCUSSION

These cases indicate that the diagnosis of BCI is challenging, and diagnostic suspicion is often raised by the lack of other lesions compatible with the presented symptoms. Even if it is supposed to be relatively uncommon, the true incidence of BCI is not precisely defined: in a recent multicentre

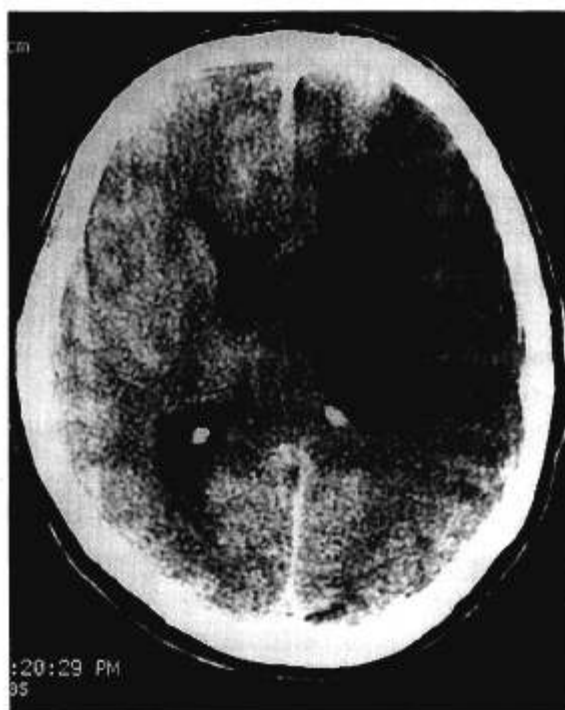


Fig. 4. Cranial CT demonstrating a wide left temporo-parietal hypodensity, corresponding to the area supplied by the middle cerebral artery. The left lateral ventricle is compressed, and there is a 1.5 cm rightward shift of the midline.

survey performed at 11 institutions in the USA during a 6-year period, 60 BCIs were identified in 49 patients (Cogbill *et al.*, 1994); unfortunately, the total number of patients at risk admitted to these institutions in that period is not reported. In a previous study, Davis *et al.* (1990) estimated that carotid artery dissection occurs in less than 1 of 1000 victims of blunt cervical injury. Using a more aggressive diagnostic approach, which included the carotid angiography when the symptoms were not compatible with the other radiologic findings, Watridge *et al.* (1989) could identify a BCI in as many as 21% of patients investigated. During the period of this study, 145 patients were admitted to our ICU with acute neurological symptoms following blunt head injury: thus, apparently, the rate of BCI diagnosed in patients admitted to our ICU is less than 3%. However, our standard diagnostic protocol of patients with head trauma does not include the imaging of the CCA and ICA in the absence of a clinical suspicion or radiological findings (see Patient no. 2): therefore, we cannot exclude that more patients could have been diagnosed to have BCI if they had been more comprehensively studied (Berlot *et al.*, 1992).

The precise time allocation of the trauma is exceedingly important in order to provide medi-

cal or surgical treatments aimed at re-establishing the blood flow through the injured vessel: not dissimilarly from patients with stroke, the window of therapeutic opportunity is supposed to be relatively narrow (Barnett *et al.*, 1995). Unfortunately, the exact timing of the initiating event is often difficult to establish due either to the possible delay of appearance of focal signs suggesting the BCI and to the initial symptoms. Moreover, in patients suffering from BCI, the free interval is extremely variable, ranging from a few hours up to as long as 10 years (Fabian *et al.*, 1990; Pretre *et al.*, 1994). Other investigators, reviewing the data from isolated case reports or small series, reported that 45% of patients were asymptomatic 24 hours after admission (Li *et al.*, 1994). Acute symptoms are often unspecific, and can include headache, agitation and dizziness (Pretre *et al.*, 1994). In a previous study, the initial neurological evaluation was negative in 53% of patients, and in another 27% the onset of symptoms was delayed 24 hours or more following the initiating event (Perry *et al.*, 1980). In a recent multicentre survey, the initial evaluation included a GCS ≤ 7 in 37% of patients and the absence of neurological symptoms in 49%; 58% of patients in this latter group became symptomatic within 12 hours after admission (Cogbill *et al.*, 1994). The interaction of different factors accounts for this wide array of initial symptoms, including the presence of other brain injuries; the absolute reduction of the blood flow through the damaged vessel; the possible distal embolization from the BCI-associated thrombosis; and the efficiency of the collateral blood flow supplied through the circle of Willis, which is extremely variable in 80% of subjects (Liekweg and Greenfield, 1978). Thus, as stated by Cogbill *et al.* (1994), the two groups of patients in whom a BCI is more likely to go undetected are constituted by neurologically asymptomatic patients without other relevant lesions, who are often discharged from the emergency ward after a short-term observation and, at the opposite extreme, comatose patients with other severe head injuries; moreover, especially in the first group, the symptoms can be ascribed to the effects of drugs or alcohol. According to Davis *et al.* (1990), the suspicion of BCI can also be raised by the presence of shock and of injuries involving the face, the cervical column and the upper thorax.

We observed a delayed onset of symptoms in two of our patients, and could identify the trauma in three of them. In the patient who died we could not identify the exact time of the injury, because, even if he was admitted immediately after the onset of the hemiparesis, the history revealed an apparently mild neck trauma occurring 3 weeks before admission, which was not followed by any

disturbance. Should this apparently trivial trauma have caused the BCI, one could argue that a minor intimal injury could have progressed to the dissection and to the consequent thrombosis of the ICA during the physical exercise immediately preceding the appearance of the hemiparesis. The absence of autoptically demonstrated abnormalities of the arterial wall is striking, even if a similar case has also been reported by Li *et al.* (1994). We failed to establish the cause of the venous thrombosis: it could be related to an hypercoagulable state associated with the dehydration (the patient received mannitol for several days before the transfer to our ICU and his serum Na⁺ was 160 mEq/l), possibly associated with the bilateral cannulation of the jugular veins; anyway, whatever its cause, it is certain that the venous thrombosis further worsened the arterial thrombosis by reducing the blood outflow (Stephens *et al.*, 1991; Segal, 1993).

Besides neurological symptoms, our patients did not present any physical signs (i.e. bruising, localized cervical swelling and/or haematoma, etc.) which could point towards the diagnosis of BCI (Cogbill *et al.*, 1994; Li *et al.*, 1994), and in three of them other commonly used indices of severity were not particularly elevated.

The diagnostic investigation of head trauma patients include the cranial CT scan in order to select the more appropriate treatment and to provide prognostic information. In patients with BCI, CT scans of the brain performed at admission can be negative or provide misleading information. Only in Patient no. 2, could the contrast-enhanced CT scan, performed 7 hours after the admission, demonstrate the obstruction of the ICA. It is thus conceivable that the use of a contrast could have increased the rate of positive scan also in the other patients. Also according to other authors (Cogbill *et al.*, 1994), initial CT scans can be normal in as many as 39% of patients; in another study, CT scans performed at admission were normal in 20 patients and demonstrated an infarction in 18 further patients; concomitant findings included subarachnoid haemorrhage, contusions and subdural haematomas (Li *et al.*, 1994). Actually, in patients with BCI, imaging techniques able to demonstrate the site and the extension of the obstruction are more valuable than non-enhanced CT scans. Even if the DSA remains the gold standard, ultrasound identified BCI in the same proportion (86%) of patients studied; the false negative patients had a BCI in the immediate proximity of the base of the skull (Cogbill *et al.*, 1994). Thus, it is desirable that a more comprehensive diagnostic investigation, including the imaging of the CCA and the ICA, should be

implemented in the presence either of an inconsistency between clinical and radiological findings or of otherwise inexplicable areas of cerebral oedema or infarction. The use of MR can provide, non-invasively, information either on the brain and on its vascular supply: recently, it has been demonstrated to be as sensible and specific as DSA in detecting surgically amenable carotid obstructions (Anson *et al.*, 1993). Unfortunately, MR cannot always be performed on a 24-hour basis and/or in emergency and in patients on life-support equipment.

Even if this information is not always available, the diagnostic suspicion of BCI can be raised by the type of the initial insult. Either direct or indirect trauma involving the head and the neck can be associated with BCI. While in the first occurrence the mechanism of injury is the application of a direct force on the vascular wall, different factors underlie the pathogenesis of BCI following indirect trauma. The extracranial carotid artery can be stretched against the first and the second cervical vertebrae after an extreme rotation or hyperextension of the head. Alternatively, the same forces can squeeze it on the transverse process of the third cervical vertebra. A hyperflexion of the head can cause the compression of the ICA against the mandible and the spine. A carotid dissection can also be related to the traction exerted by the hypoglossal nerve on the arterial wall (Li *et al.*, 1994). The intrapetrous part of the artery can be damaged following fractures of the skull base or intraoral injuries (Perry *et al.*, 1980). It is clear, however, that all these mechanisms can coexist in the same patient, making it difficult or impossible to identify a precise cause in most cases. The more frequent concomitant BCI-associated injuries include the head, the cervical spine and the upper thorax (Davis *et al.*, 1990; Goodwin and Johnson, 1994). The circumstances of trauma may not provide much information, because the occurrence of BCI has been reported following virtually every kind of event, even if motor-vehicular accidents (MVA) accounts for most of them (Mokkri *et al.*, 1988; Cogbill *et al.*, 1994; Davis *et al.*, 1990; Pretre *et al.*, 1994). Seatbelts have been implicated in the pathogenesis of BCI in MVA victims, probably due to a CCA or ICA overstretching mechanism (Benito *et al.*, 1990; Reddy *et al.*, 1990). The occurrence of BCI has been described also after penetrating neck trauma (Lebos and Saadia, 1994). We argue that, in our patients, a direct trauma can be specifically identified only in Patient no. 2, who suffered a homolateral tonsillar trauma. In Patients nos 1 and 3 the hyperextension of the neck is the most likely mechanism, whereas in Patient no. 4 we failed to identify a precise initiating event. Actually, either a remote direct trauma

and/or a cervical hyperextension in the time immediately preceding the onset of symptoms could be related to the BCI.

The treatment of BCI is controversial, and basically depends on the presence of concomitant cerebral or extracerebral injuries, on the type and on the site of the lesion, on the severity of symptoms and on the time lag elapsing between the initiating event and the onset of symptoms. As in every case of vascular injury, an anticoagulant treatment is indicated, in the absence of contraindications (Rothrock and Hart, 1991; Barnett *et al.*, 1995); however, despite an intensive protocol which included subcutaneous or intravenous heparin, aspirin and ticlopidine, as indicated by the current literature (Becker, 1995), in our patients this approach was not always able to prevent the progression of symptoms. Although over the past few years an early surgical treatment of BCIs was recommended (Perry *et al.*, 1980), more recently this aggressive approach underwent a reappraisal: at the present time, it appears that surgery should be limited to selected cases, namely patients with surgically repaired CCF and carotid pseudoaneurysms, which can successfully be managed with balloon embolization and extracranial-intracranial bypass procedures, respectively (Cogbill *et al.*, 1994; Li *et al.*, 1994). The complete thrombosis, even if associated with an intimal dissection, constitutes a contraindication to the operation, because the surgical trauma can further deteriorate the cerebrovascular instability related to the initiating event. In patients with BCI associated with a complete thrombosis of the damaged vessel, surgery is associated either with a high rate of reocclusion and a high mortality rate (Cogbill *et al.*, 1994; Li *et al.*, 1994). Only the first patient in our series was operated on, with disappointing results: the saphenous bypass rapidly occluded, and an ischaemic area appeared in the contralateral cerebral hemisphere, probably due to a streak of blood or to a distal embolization from the thrombosed bypass. Independently from the chosen treatment, patients with BCI and symptoms indicating an increase of the intracranial pressure should be admitted to the ICU and aggressively managed (White and Likavec, 1992): as illustrated by the case of Patient no. 4, a delayed aggressive treatment can be associated with a poor prognosis.

The long-term outcome of patients with BCI largely depends on the extension of the ischaemic cerebral area, on the presence of concomitant injuries and on other factors, including the neurologic condition at admission, the systemic haemodynamic derangements and the appropriateness of the treatment. As stated by Li *et al.* (1994), a

good neurologic outcome is determined either by a valid collateral circulation and by an early aggressive approach; according to the same authors, a good neurologic outcome is expected in 39% of patients whose initial CT scan is normal, but only in 5% patients in whom an infarction is demonstrated. The prognosis has also been associated with the type of BCI, being good in patients with CCF, whereas complete thrombosis has a mortality rate of 40% (Cogbill *et al.*, 1994). In our experience, we observed both the extremes of this spectrum: in Patient no. 3, the valid collateral blood supply to the jeopardized areas impeded the formation of large ischaemic areas, and the small cortical contusions were likely to be responsible for the residual disturbances. Conversely, in Patient no. 4 several factors, including the massive cerebral oedema, severe dehydration and reduced blood outflow, contributed to diminishing the collateral blood flow. The survivors recovered after a prolonged time in the rehabilitation unit, and, at the present time, complain only minor impairments which did not prevent them from attending to their usual pre-trauma duties.

CONCLUSIONS

Despite a more in-depth knowledge of its causes and pathophysiology, the reported incidence of BCI could be largely underestimated, due principally to the common presence of concomitant neurological disorders. Not dissimilarly from missed cervical injuries, unrecognized BCI carries a severe prognosis, in terms either of outcome or residual neurological disabilities. Unfortunately, there are no specific signs of BCI and the history and the physical examination may provide scant, if not misleading, information. Due to this relative lack of early warning signs, a prudent attitude should be to consider BCI in every patient with post-traumatic neurologic disturbances, and to adopt widely available diagnostic tools, such as ultrasound, as screening procedures, independently from the initial CT findings. Patients diagnosed as having BCI should be referred to experienced centres, which should be familiar with all the required diagnostic and therapeutic procedures.

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