Acute arterial infarcts in patients with severe head injuries

Deepak Agrawal, Anil Garg
Department of Neurosurgery, JPN Apex Trauma Centre, All India Institute of Medical Sciences, New Delhi, India

ABSTRACT

Aims and Objectives: To study the incidence, demographic profile, and outcome of patients with severe closed head injuries who develop acute arterial infarcts. **Materials and Methods:** Patients with severe head injury (Glasgow coma score (GCS) ≤8) presenting within 8 h of injury in the Department of Neurosurgery over a period of 5 months were enrolled in the study. Patients with penetrating head injury, infarct due to herniation and iatrogenic arterial injuries were excluded from the study. Only arterial infarcts developing within 8 h of injury were included in the study. A computed tomography (CT) head was done on all patients within 8 h of injury and repeated if necessary. Arterial infarct was defined as well-demarcated wedge-shaped hypodensity corresponding to an arterial territory on plain CT of the head. Outcome was assessed using Glasgow outcome score (GOS) at 1 month post-injury or at death (whichever came earlier). **Results:** Forty-four patients of severe head injury were included in the study during the above period. Of these, four patients (9.1%) had arterial infarcts on the initial CT scan. The male:female ratio was 1:3. The mean age was 54 years (range 3–85 years). Two patients had infarcts in the middle cerebral artery distribution and two in the superior cerebellar artery distribution. Poor outcome (GOS 1–3) was seen in 100% of the patients with arterial infarct compared to 52.5% (*n*=21) in patients with severe head injury without arterial infarct. **Conclusions:** A significant percentage of patients with severe head injury have arterial infarcts on admission, which may imply arterial injury. Our study shows that these patients have a poorer prognosis *vis-à-vis* patient without these findings.

Key words: Arterial dissection, arterial infarct, computerized tomography, epidemiology, head injury, trauma

INTRODUCTION

Acute (within 8 h of injury) post-traumatic arterial infarcts of the brain have been described in post-mortem studies,[1-4] but ante-mortem data are lacking. This entity needs to be differentiated from post-traumatic cerebral infarction which includes a wide range of etiologies.[5,6] Although atherosclerosis with/without thrombosis is the most common cause of spontaneous cerebral infarction,[7] this mechanism is rarely responsible for post-traumatic infarction where arterial dissection remains the predominant cause.[8] A thorough review of literature revealed complete lack of ante-mortem data on the epidemiology and outcome of acute post-traumatic cerebral infarction in severe closed head injuries. Surprisingly, only isolated case reports of post-traumatic vascular injury leading to brain infarcts in patients with closed head injury are available.[8-10] This study is being done to study the incidence, demographic profile, prognosis, and distribution of arterial infarcts in severe head injury patients.

Access this article online	
Quick Response Code:	Website: www.ijns.in
	DOI: 10.4103/2277-9167.102276

MATERIALS AND METHODS

This prospective observational study was done over a 5-month period (November 2007–March 2008) at Jai Prakash Narayan Apex Trauma Center, which is a 277-bedded level 1 trauma center of the All India Institute of Medical Sciences, New Delhi. Consecutive patients with severe head injury (Glasgow coma score (GCS) ≤8) presenting within 8 h of injury in the Department of Neurosurgery were evaluated for this study. Approval from the institutional ethics committee was taken for the study. Patients with penetrating head injury, iatrogenic arterial injuries, and history of stroke in the past were excluded from the study. A computed tomography (CT) head was done on all patients within 8 h of injury and repeated if necessary. Arterial infarct was defined as well-demarcated wedge-shaped hypodensity corresponding to an arterial territory on plain CT of the head. Only arterial infarcts developing within 8 h of injury were included in the study and patients with diffuse

Address for correspondence: Dr. Deepak Agrawal,
JPN Apex Trauma Centre, All India Institute of Medical Sciences, Ansari Nagar, New Delhi – 110029, India.
E-mail: drdeepak@gmail.com

hypoxic injury, infarction in anterior cerebral artery and posterior cerebral artery territory due to subfalcine and transtentorial herniation were also excluded from the study. Mode of injury, clinical features, GCS (at admission), and CT findings were recorded for each patient and the outcome was recorded at discharge or death. Outcome was assessed using Glasgow outcome score (GOS) at 1 month post-injury or death, whichever came earlier. GOS 1–3 was considered as poor outcome and GOS 4–5 was considered as good outcome in this study. For the purpose of demographic and outcome comparisons, patients were divided into two groups: a control group consisting of the cohort of enrolled severe head injured patients without acute arterial infarcts and an acute arterial infarct group consisting of patients with arterial infarcts.

Statistical analysis was done using GraphPad statistical software (www.graphpad.com). Fisher's exact test was used to assess the *P* value between the two groups.

RESULTS

Forty-four patients of severe head injury were included in the study during the above period. Of these, four patients (9.1%) had arterial infarcts on initial CT scan of the head and comprised the acute arterial infarct group. The remaining 40 patients comprised the control group.

Control group (n=40)

The male:female ratio was 7:3. The mean age was 37 years (range 10–68 years). The mean admission GCS was 5.2 (range 3–8). Poor outcome (GOS 1–3) was seen in 21 (52.5%) patients and good outcome (GOS 4–5) was seen in 19 (47.5%) patients.

Acute arterial infarct group (n=4)

The male:female ratio was 1: 3. The mean age was 54 years (range 3–85 years). The mean admission GCS was 5.5 (range 4–8). The mechanism of injury was road traffic injuries in two patients, fall from height in one, and fall from bed in one patient.

The admission CT revealed infarcts in the internal carotid artery (ICA) territory in one case [Figure 1a and b], middle cerebral artery (MCA) territory in one case [Figure 2], superior cerebellar artery in one patient [Figure 3], and posterior inferior cerebellar artery in one patient [Figure 4]. One patient (with ICA territory infarct) underwent surgery for associated acute fronto-temporo-parietal subdural hematoma and three patients were managed conservatively. Of these four patients, two patients died and two remained in vegetative state 1 month post-injury. Mortality in acute arterial infarct group was 100% compared to 52.5% in the control group. Although not statistically significant [P=0.12 (Fisher's exact test, two tailed)], this is clinically important with a trend toward worse outcome. The female predominance of those with infarct, in comparison to those without infarct, although not being statistically significant, also shows a trend towards worse outcome (P=0.11).

DISCUSSION

Our study shows that a significant number (9.1%) of severe head injured patients may have acute arterial infarcts which may be responsible for the poor outcome in these patients. The etiology of these arterial infarcts remains poorly defined. Traumatic vascular occlusion following closed head injury is a well-recognized but uncommon cause of acute arterial cerebral infarction,[1-4] and both extracranial as well as intracranial vessels have been implicated in its pathogenesis.[8-10] Several multicenter reviews suggested that blunt carotid injury occurs in 0.08–0.17% of all blunt trauma victims admitted to the hospital.[11,12] Differences in the anatomy and structure of both extracranial and intracranial vessels make it likely that different pathogenetic mechanisms are operative in different circumstances.[4,13] However, subintimal dissection and thrombosis are thought to be the most common causes of vascular occlusion following closed head injury.[8] Injury to neck vessels or at the base of skull with intimal injury and subsequent embolization has also been postulated as a possible mechanism in some cases.[14,15] Reflex spasm or vasospasm following traumatic subarachnoid hemorrhage has been seen as a less likely cause of brain infarct following closed head injury.[16] As our study was epidemiological in nature, we are unable to comment on the causative factors responsible for cerebral infarction in our series.

Another surprising finding of this study was the overwhelming female predominance in our series, the reasons of which remain unclear. The outcome for these patients was also dismal in our series and could be due to the fact that all were managed conservatively. This outcome in itself shows that further studies are warranted using early identification and aggressive management, as the current management practices are failing in these patients.

As defined in the Materials and Methods section, the diagnosis of arterial infarct was made based on the pattern of hypodensity seen on CT scans and this remains a limitation of this study. Currently cerebral angiogram is not being done at our center in patients with severe closed head injury with suspected arterial

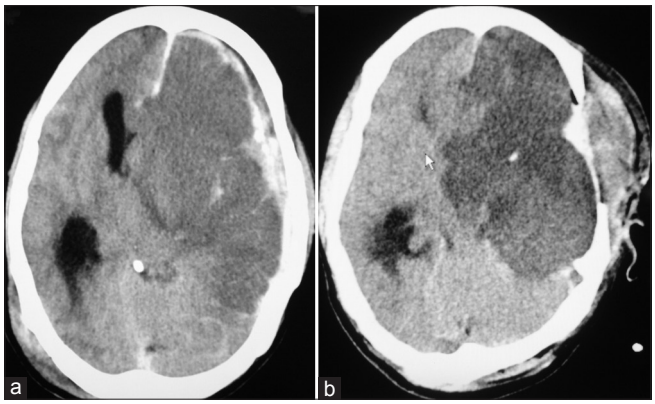

Figure 1: (a) Plain CT head of first patient showing left ICA territory infarct with mass effect and associated acute subdural hematoma. (b) Repeat CT after 24 h showing evidence of left fronto-temporo-parietal craniotomy and well-developed infarct in the same territory

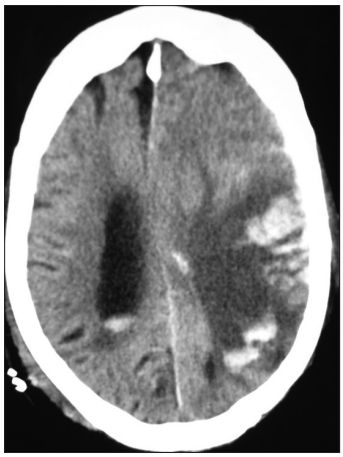

Figure 2: Plain CT head of second patient showing hemorrhagic transformation of left MCA territory infarct with effacement of ipsilateral lateral ventricle

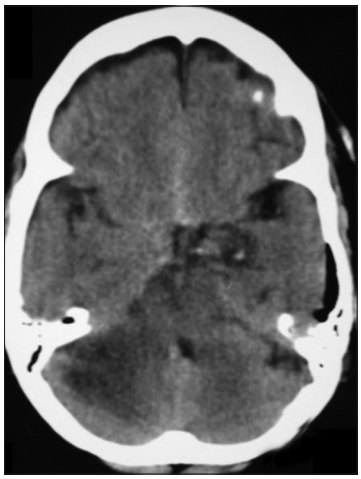

Figure 3: Plain CT of the third patient showing well-developed infarct in the right superior cerebellar artery distribution

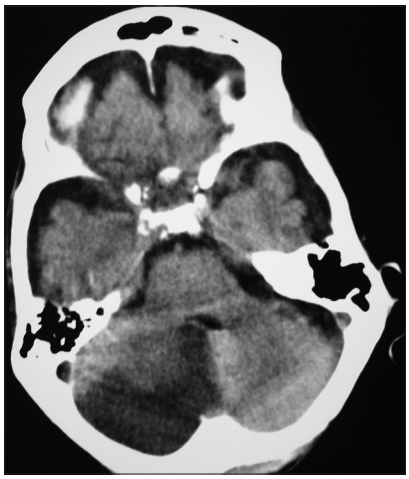

Figure 4: Plain CT of the fourth patient showing infarct in the right posterior cerebellar artery distribution

infarcts because of a variety of reasons. Firstly, due to lack of epidemiological and clinical data, the incidence, management, and outcome in these patients remain ill-defined. Secondly, these patients are usually critically ill and priority is given to evacuate mass lesions (if any) and control intracranial pressure. Thirdly, it can be argued that as the management does not change following detection of subintimal dissection in an established cerebral infarct on CT head, there is little justification in subjecting such critically ill patients to a cerebral angiogram.

Having a high index of suspicion always helps in such patients. Being an uncommon entity, cerebral vascular injury may be missed on initial examination. Also, there are few, if any, external signs which are also often masked by more obvious injuries. Clinical manifestations of arterial injuries may include alteration of consciousness, cerebellar signs, or signs of brainstem dysfunction, which can also be attributed to the head injury. The initial CT scan is often read as normal in a developing infarct. However, retrospective evaluation will always show the involved region. In our study, three patients who had well-developed infarcts on sequential CT studies had their initial CT head read as normal. Doppler ultrasound may be a useful screening modality in suspected blunt carotid trauma.[10] Angiography is the gold standard and should be done if the patient has external sign of neck injury associated with change in the level of consciousness or lateralizing neurological findings unexplained by CT scan findings. The role of MR-angiography is still not defined, but it may be a valuable diagnostic aid if the angiography is contraindicated.[9]

On confirmation of diagnosis, most of the patients are managed conservatively. Although the optimal management has yet to be found, treatment modalities available are anticoagulation, repair of the vessel, ligation or balloon occlusion of the injured artery, and

extracranial–intracranial (EC–IC) bypass grafting.[17-19] Anticoagulant therapy to arrest propagation of the thrombus and distal embolization has been advocated by some with success, but has not been widely accepted because of the risks of vessel rupture, extending pre-existing dissection, or transforming the infarction into hemorrhage.[17] Ligation or balloon occlusion with concomitant EC–IC bypass grafting have been tried but have usually been unsuccessful, except in patients with transient or mild neurological deficits.[18,20]

Strength of the study

This is the first study of its kind which has attempted to define the epidemiology of acute traumatic cerebral infarcts in the ante-mortem period.

Limitations of the study

The study number is small and larger studies will be required for confirming the findings of this study. Also, as the study design was retrospective, many variables such as Injury Severity Score (ISS) were not available and could not be analyzed. As previously mentioned, the diagnosis of arterial infarct was made based on the pattern of hypodensity seen on CT scans and this remains a limitation of this study.

Further directions of the study

This study raises several interesting questions regarding female preponderance, need for cerebral angiogram, high mortality, and lack of management protocols. Larger multicenter studies are urgently required to address these issues.

CONCLUSIONS

A significant percentage of patients with severe closed head injury have acute arterial infarcts on admission. Our study shows that females are more vulnerable to develop these infarcts for unknown reasons. These patients have a poorer prognosis *vis-à-vis* patient without these findings, with 100% of patients with infarcts in our series having a poor outcome. Further studies are warranted for identifying the pathogenesis and optimal treatment in such patients.

REFERENCES

1. Hart RG, Easton JD. Dissections of cervical and cerebral arteries. Neurol Clin 1983;1:155-82.
2. Sasaki O, Ogawa H, Koike T, Koizumi T, Tanaka R. A clinicopathological study of dissecting aneurysms of the intracranial vertebral artery. J Neurosurg 1991;75:874-82.
3. Biller J, Hingtgen WL, Adams HP. Cervicocephalic arterial dissections: A ten year experience. Arch Neurol 1986;43:1234-8.
4. Stehbens WE. Pathology of cerebral blood vessels. M Louis: Mosby; 1972. p. 1-97.
5. Petito CK. Cerebrovascular diseases. In: Nelson JS, Schochet SS, editors. Principles and practice of neuro-pathology. St. Louis: Mosby; 1993. p. 436-58.
6. Li MS, Smith BM, Espinosa J, Brown RA, Richardson P, Ford R. Nonpenetrating trauma to the carotid artery: Seven cases and a literature review. J Trauma 1994;36:265-72.
7. Mobbs RJ, Chandran KN. Traumatic middle cerebral artery occlusion: Case report and review of pathogenesis. Neurol India 2001;49:158-61.
8. Rommel O, Niedeggen A, Tegenthoff M, Kiwitt P, Botel U, Malin JP. Carotid and vertebral artery injury following severe head and cervical spine trauma. Cerebrovasc Dis 1999;9:202-9.
9. Cogbill TH, Moore EE, Meissner M. The spectrum of blunt injury to the carotid injury: A multicenter perspective. J Trauma 1994;37:473-9.
10. Davis JW, Holbrook TL, Hoyt DB. Blunt carotid artery dissection: Incidence, associated injuries, screening, and treatment. J Trauma 1990;30:1514-7.
11. Wilkinson IM. The vertebral artery. Extracranial and intracranial structure. Arch Neurol 1972;27:392-6.
12. Gunning AJ, Pickering GW, Robb-Smith AHT. Mural thrombosis of the internal carotid artery and subsequent embolism. Q J Med 1964;33:155-95.
13. Raney AA. Cerebral embolism following minor wounds of the carotid artery: Report of an autopsy. Arch Neurol Psychiatry 1948;60:425-39.
14. Wilikins RH, Odom GL. Intracranial arterial spasm associated with craniocerebral trauma. J Neurosurg 1970;32:626-33.
15. Dragon R, Saranchak H, Lakin P, Strauch G. Blunt injuries to the carotid and vertebral arteries. Am J Surg 1981;141:497-500.
16. Gianotta SL, Ahmadi J. Vascular lesions with head injury. In: Wilkins RH, Rengachary SS, editors. Neurosurgery. New York: McGraw-Hill; 1985. p. 1678-88.
17. Bishara SN, Dempster AG, Mee EW. Vertebral artery occlusion associated with closed head injury: Report of three cases. Br J Neurosurg 1989;3:495-501.
18. Fisher CM. Occlusion of the vertebral arteries causing transient basilar symptoms. Arch Neurol 1970;22:13-9.
19. Tian HL, Geng Z, Cui YH, Hu J, Xu T, Cao HL, *et al*. Risk factors for posttraumatic cerebral infarction in patients with moderate or severe head trauma. Neurosurg Rev 2008;31:431-6.
20. Tawil I, Stein DM, Mirvis SE, Scalea TM. Posttraumatic cerebral infarction: Incidence, outcome, and risk factors. J Trauma 2008;64:849-53.

How to cite this article: Agrawal D, Garg A. Acute arterial infarcts in patients with severe head injuries. Indian J Neurosurg 2012;1:126-9.

Source of Support: Nil, **Conflict of Interest:** None declared.