

CEREBRAL INFARCTION
AS NEUROSURGICAL POST OPERATIVE COMPLICATION
: SERIAL CASES



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ABSTRACT

Many of the complications following surgery, previously critical, are now preventable or readily curable as a result of advances in anaesthesia, transfusion, fluid and electrolyte management, and control of infection. A cerebral infarction is the ischemic due to a disturbance in the blood vessels supplying blood to the brain. A cerebral infarction occurs when a blood vessel that supplies a part of the brain becomes blocked or leakage occurs outside the vessel walls. This loss of blood supply results in the death of that area of tissue. Cerebral infarctions vary in their severity with one third of the cases resulting in death.

Symptoms of cerebral infarction are determined by topographical localisation of cerebral lesion. If it is located in primary motor cortex- contralateral hemiparesis occurs, for brainstem localisation typical are brainstem syndromes: Wallenberg's syndrome, Weber's syndrome, Millard-Gubler syndrome, Benedikt syndrome or others. Infarctions will result in weakness and loss of sensation on the opposite side of the body. Physical examination of the head area will reveal abnormal pupil dilation, light reaction and lack of eye movement on opposite side. If the infarction occurs on the left side brain, speech will be slurred. Reflexes may be aggravated as well.

We have tried to investigate patients who have cerebral infarction as neurosurgical post operative complication. There were 23 patients in Dr. Soetomo hospital from April 2009 to March 2010. The present study was undertaken to determine the nature, timing, and pattern of the complications relative to the patient's preoperative status, the magnitude of operative injury, and the stage of his post-operative response in which the complication occurred. As a result, it might be possible to identify predisposing factors, or at least predict those patients in whom such accidents are likely to occur as a part of their response, or failure to response, to accidental or surgical trauma.

Key words : cerebral infarction

Introduction

Many of the complications following surgery, previously critical, are now preventable or readily curable as a result of advances in anaesthesia, transfusion, fluid and electrolyte management, and control of infection. A cerebral infarction is the ischemic due to a disturbance in the blood vessels supplying blood to the brain. A cerebral infarction occurs when a blood vessel that supplies a part of the brain becomes blocked or leakage occurs outside the vessel walls. This loss of blood supply results in the death of that area of tissue. Cerebral infarctions vary in their severity with one third of the cases resulting in death ⁽²⁾.

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CT and MRI scanning will show damaged area in the brain, showing that the symptoms were not caused by a tumor, subdural hematoma or other brain disorder. The blockage will also appear on the angiogram⁽²⁾.

Clinical Material and Methods

We reviewed medical records of 23 pediatric patients suffering cerebral infarction treated in Department of Neurosurgery, Soetomo General Hospital, Surabaya within period of April 2009 to March 2010. The review includes: the age and gender at onset of accident, the time of accident to hospital, the time of duration of surgery, type of lesion, type of brain injury, and location of infarction.

Results

Data of 23 patients consists of 17 male and 6 female were analyzed. The male to female ratio was 2.8:1 (Table 1). The mean age was 41 years old (range 17 to 65 years old). According to specific developmental characteristics, patients were divided into age groups as follows: 10-20 years, 21-30 years, 31-40 years, 41-50 years, 51-60 years, and 61-70 years, with the most prevalence in our series was between 41-50 years old (table 1). Careful history was taken, and detailed physical and neurologic examinations were performed.

Table 1. Patient Characteristics according to age and sex.

	<i>n (%)</i>
<i>Sex</i>	
Male	17 (71%)
Female	6 (29%)
<i>Age</i>	
10 – 20 years old	3
21 – 30 years old	4
31 – 40 years old	3
41 – 50 years old	8
51 – 60 years old	4
61 – 70 years old	1

The time of sending patients to the hospital was analyzed. The time was classified into 4 categories: 1-3 hours, 4-6 hours, 7-9 hours and more than 10 hours. Mostly patient was sent to hospital around 4 – 6 hours after the accident. There were 4 patients who sent to the hospital around 1 – 3 hours after the accident and there were 7 patients who sent to the hospital around 7 – 9 hours after the accident.

Table 2. The time of accident to hospital

<i>Time</i>	<i>N (%)</i>
1 – 3 hours	4
4 – 6 hours	12
7 – 9 hours	7
More than 10 hours	0

The time of duration of surgery means the length of operation has been taken. Mostly the surgery takes 4 – 6 hours , around 12 patients. Other patients have 1 – 3 hours. None have more than 7 hours.

Table 3. The time of duration of surgery

<i>Time</i>	<i>n (%)</i>
1 – 3 hours	11
4 – 6 hours	12
More than 7 hours	0

Type of lesions in these cases are epidural hematoma, subdural hematoma, intracerebral hematoma, depressed fracture, and double lesions. Double lesion means that there were more than one lesion when the accident occurred. There were 8 patients who have criteria of double lesion. There were 6 patients who suffered epidural hematoma and 3 patients who suffered intracerebral hematoma. There were 3 patients who suffered depressed fracture.

Table 4. Type of lesion

<i>Lesion</i>	<i>n (%)</i>
EDH	6
SDH	3
ICH	3
Depressed fracture	3
Double lesion	8

Brain injury is divided into 3 groups. Those are mild, moderate, and severe brain injury. We use Glasgow Coma Scale (GCS) as a standard. The criteria of mild brain injury is GCS 14-15. The criteria of moderate brain injury is GCS 9-13. The criteria of severe brain injury is GCS 3-8. There were only one patient with criteria of mild brain injury, 8 patients with criteria of moderate brain injury, and 14 patients with severe brain injury.

Table 5. Type of brain injury

<i>Brain injury</i>	<i>n (%)</i>
Mild brain injury	1
Moderate brain injury	8
Severe brain injury	14

Territorial cerebral infarction (complete or incomplete): well circumscribed hypodense lesions within a defined cerebral vascular territory, involving the entire territory (complete) or only part of it (incomplete). The following vascular territories were considered: anterior cerebral artery (ACA), middle cerebral artery (MCA), posterior cerebral artery, anterior choroidal arteries, lenticulostriate arteries (LSAs), thalamoperforating arteries (TPAs), basilar artery (BA), superior cerebellar artery (SCA), anterior–inferior cerebellar artery, and posterior–inferior cerebellar artery. The cerebral vascular territories of each artery were determined according to Tatu et al. There are 4 patients with infarction in location of ACA, 4 patients with infarction in location of MCA, 8 patients with infarction in location of PCA, 7 patients with infarction in half hemisfer.

Table 6. Location of infarction

<i>Location</i>	<i>n (%)</i>
Anterior cerebral artery (ACA)	4
Middle cerebral artery (MCA)	4
Posterior cerebral artery (PCA)	8
Half hemisfere	7

Discussion

Posttraumatic cerebral ischemia (PTCI) included functionally impaired yet still viable tissue, so called ischemic penumbra, and irreversible cerebral infarction. Cerebral infarction is frequent in patients who die after moderate or severe head trauma, with a reported incidence from 55 to 91%. Clinical investigations have focused on rapid detection and treatment of early PTCI to reduce the occurrence of cerebral infarction. However, because of its brief duration and heterogeneous nature, antemortem demonstration of PTCI has proved elusive ⁽¹⁾.

Diagnosis of cerebral infarction in head trauma patients might influence posttraumatic neurologic recovery. As selection of outcomes that are measurable, standardized, and relevant to lifestyle is critical to enhance planning and design of future trials, cerebral infarction could be used as an outcome measure in randomized controlled trials. Moreover, diagnosis of cerebral infarction could be used as a standard diagnostic reference with which early surrogate indicators of PTCI could be compared. In one retrospective cohort study, the prevalence of cerebral infarction was 1.9%; however, head trauma patients with all grades of severity were enrolled and potential causes were not investigated^(6,10).

Only cerebral infarction that developed after trauma was considered. Ischemic lesions identified on first CT scan whose density remained unchanged during the neuroradiologic follow-up were considered old infarcts and were not included in the analysis. Cerebral infarction was diagnosed according to the following criteria adapted from neuropathologic studies : 1) Territorial cerebral infarction (complete or incomplete): well circumscribed hypodense lesions within a defined cerebral vascular territory, involving the entire territory (complete) or only part of it (incomplete). The following vascular territories were considered: anterior cerebral artery (ACA), middle cerebral artery (MCA), posterior cerebral artery, anterior choroidal arteries, lenticulostriate arteries (LSAs), thalamoperforating arteries (TPAs), basilar artery (BA), superior cerebellar artery (SCA), anterior–inferior cerebellar artery, and posterior–inferior cerebellar artery. The cerebral vascular territories of each artery were determined according to Tatu et al. 2) Watershed cerebral infarction: well circumscribed hypodense lesions located in boundary zones between the territories of ACA, MCA, and PCA (superficial or leptomeningeal border zones) or situated in terminal zones of perforating arteries within the deep white matter (deep or medullary border zones). 3) Nonterritorial nonwatershed cerebral infarction: single or multiple hypodense lesions, unilateral, bilateral, or multifocal with marked borders without a precise localization in a vascular territory^(11,12).

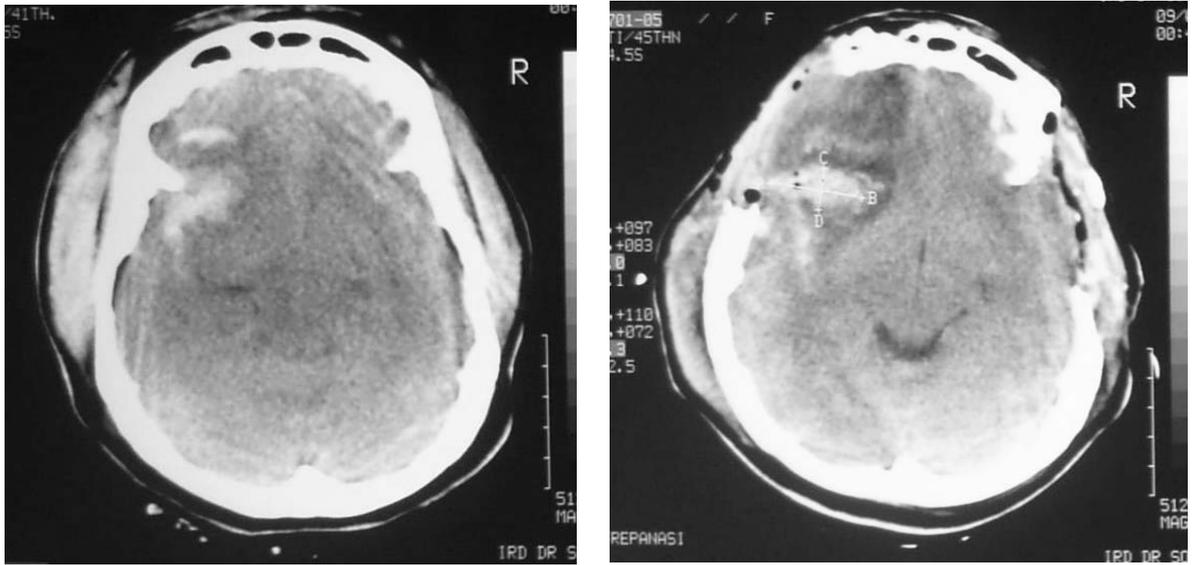


Figure 1. Territorial infarction. Patient 8. (A) Brain CT scan at admission: left frontotemporal intracerebral hemorrhagic lesion (B) Brain CT scan, day 1. Development of territorial infarctions in the left frontotemporal lobe (left ACA). Perifocal edema are also detectable in the left frontobasal cortex.

If a hypodense lesion was present on brain CT in the first 72 hours after trauma, in order to differentiate watershed and nonterritorial nonwatershed cerebral infarctions from primary posttraumatic cerebral contusion, we required that the lesion had to be still recognizable after 21 days to be classified as cerebral infarction⁽¹⁵⁾.

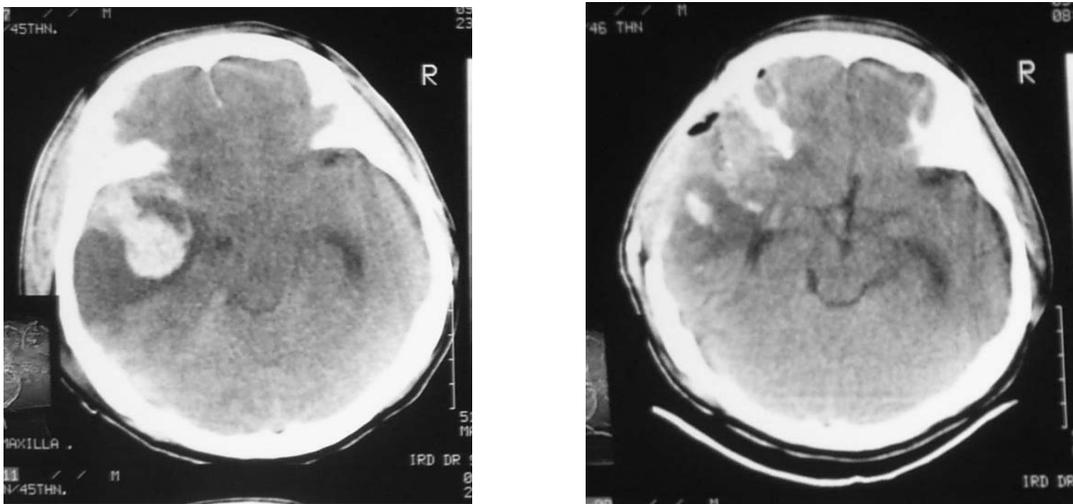


Figure 2. Territorial infarction. Patient 4. (A) Brain CT scan at admission: left temporal intracerebral hemorrhagic lesion. (B) Brain CT scan, day 1, 6 hours after decompressive craniectomy: brain herniation through the skull defect and unilateral hypodensity involving the temporal cortex and subcortical white matter in the vascular territory of distal middle cerebral artery branches.

Patients who sustain head trauma experience not only the effects of the primary traumatic injury but also secondary, mainly ischemic, brain damage. One neuropathologic series demonstrates cerebral infarction in up to 90% of fatal traumatic brain injury cases, particularly when intracranial hypertension complicates the clinical course. In this study, head trauma patients of all severities were included, and therefore patients with severe and moderate head trauma those mostly at risk of developing cerebral infarction were likely to represent a minority. In addition, neither intracranial hypertension nor other secondary cerebral insults were recorded, and hence potentially causative events and the mechanism of cerebral infarction remained largely speculative. We found that intracranial hypertension was strongly and independently associated with cerebral infarction. This finding has long been demonstrated in neuropathologic studies showing cerebral infarction to be secondary to compression, distortion, and internal brain herniation caused by increased ICP, but has never been demonstrated ante mortem in patients ^(9,16).

There are few proven therapies for traumatic brain injury. Owing to the complexity of factors that influence outcome, clinical trials have been difficult to design and conduct. Several trials that have been performed in recent years failed to demonstrate a significant improvement in outcome, despite promising preclinical data. There are several ways to improve planning of clinical trials, including selection of outcomes that are easy to measure and relevant to recovery. Early outcome availability would also be important. Our results demonstrate that Ct diagnosed cerebral infarction may be considered as an outcome measure in randomized controlled trials investigating the efficacy of drugs or complex monitoring-related strategies. Brain CT-diagnosed cerebral infarction can also be proposed as a reference diagnostic standard in observational cohort studies investigating surrogate outcome measures of PTCI. Validation of surrogate PTCI outcomes would help concentrate research on those measurements that effectively predict an increased risk of cerebral infarction, hence abandoning measures whose theoretical rationale is not demonstrated ^(5,7).

Cerebral infarctions in head trauma patients are related to brain herniation and distortion, compressive effects of intracranial hematomas or severe cerebral edema, vasospasm, direct vascular injury such as laceration, transection or dissection (with subsequent thrombosis and possible embolization), fat embolism, or injury to cortical cerebral vessels in association with skull fracture. Hippocampus, basal ganglia, cerebral cortex, and cerebellum are the most common sites of infarction upon neuropathologic investigations. Common patterns of brain damage in the cerebral cortex include the boundary zone, arterial territory, and multiple focal and diffuse cerebral infarctions, in this order of occurrence. In our series, direct vascular compression with MCA infarction, posttraumatic shearing injury with LSA infarction, transtentorial brain herniation with PCA infarction, subfalcial brain herniation with ACA infarction, and postdecompressive craniectomy MCA/LSA infarction were most common⁽⁸⁾.

Cerebral cortical infarctions were more common than subcortical infarctions, and territorial cerebral infarctions were more common than watershed infarctions, whereas nonterritorial nonwatershed cerebral infarctions were not detected. MRI, particularly diffusionweighted and perfusion-weighted techniques, is the reference standard to diagnose brain infarction, and future studies should clarify whether the use of more sensitive neuroradiologic investigations actually improves detection of posttraumatic cerebral infarction^(3,10).

Decompressive craniectomy, allowing herniation of brain tissue through the bone defect, exposes arteries and veins to compression by the dural margin of duraplasty with further congestion, edema, and ischemia in the herniated tissue, which was the possible mechanism causing MCA infarction. LSA can be stretched or lacerated by cerebral displacement after high-speed accidents with sudden deceleration, resulting in lacunar infarctions; we hypothesize that rapid reduction of ICP attained by surgical decompression caused a shearing injury of LSA with cerebral infarction. Whatever the pathophysiologic mechanism, physicians should be aware of possible ischemic complications of decompressive craniectomy, which is

increasingly used to treat refractory intracranial hypertension in head trauma patients (13,14)

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