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CASE REPORT

ARTERIOVENOUS MALFORMATION OR CONTUSION : A DIAGNOSTIC DILEMMA

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A young man was involved in a motor vehicle accident and sustained cerebral contusion in the right frontal and occipital lobes. Computed tomography (CT) scan done 2 weeks after the injury revealed multiple serpenginous structures which enhanced following intravenous contrast media administration. The possibility of arteriovenous malformation (AVM) was raised and angiography was suggested. However, he was managed conservatively and a repeat CT scan 4 months post trauma revealed encephalomalacia in the right frontal and occipital lobes with no more surrounding enhancement. The cause for the multiple serpenginous enhancement demonstrated previously confirmed those are areas of hyperperfusion in healing surface brain contusion. The aim of this case report is to discuss on the possible causes of focal enhancement following head injury versus the features of AVM on plain and post contrast CT scan with the effort to clear the doubt and to avoid future confusion.

Key words : **head injury, contusion, hyperperfusion.**

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Introduction

Head injury is one of the common causes for requesting cranial CT. In the acute stage, a plain CT scan of the brain is sufficient to achieve the diagnosis of cerebral contusion, intracranial hemorrhage or skull fracture. Only in the subacute or chronic head injury, intravenous contrast is occasionally used to facilitate detection of isodense hemorrhage or complications such as subdural empyema.

In cerebral contusion, it is noted that an area of hyperemia may occur surrounding the contused brain. This has been studied and proven in both human and experimental animals.

The natural history of brain contusion includes the acute damage phase, liquefaction with edema, a repair stage and a final cystic degenerative stage. During healing, there will be neovascularization but the new vessels formed have defective endothelial junctures causing disturbance in blood brain barrier. Thus, at this stage, if intravenous contrast is given for cross sectional imaging, enhancement will be demonstrated.

Case Report

MAO, a 17 year-old young man, was involved in a motor vehicle accident and sustained head injury with a Glasgow Coma Scale (GCS) of 7/15 at presentation. CT scan brain showed a right frontal and occipital lobe contusion. A right frontal craniectomy and evacuation of clot was done immediately. He was transferred to another hospital on the request of his family members. A follow-up cranial CT scan performed two weeks later revealed focal gliosis of the right frontal and occipital lobes secondary to previous contusion (Figure 1). Post contrast scan showed serpenginous gyriform enhancement around the contusion in the right frontal and occipital lobes (Figure 2). The possibility

Figure 1 : Plain CT brain showing focal gliosis of the right frontal and right occipital lobes.

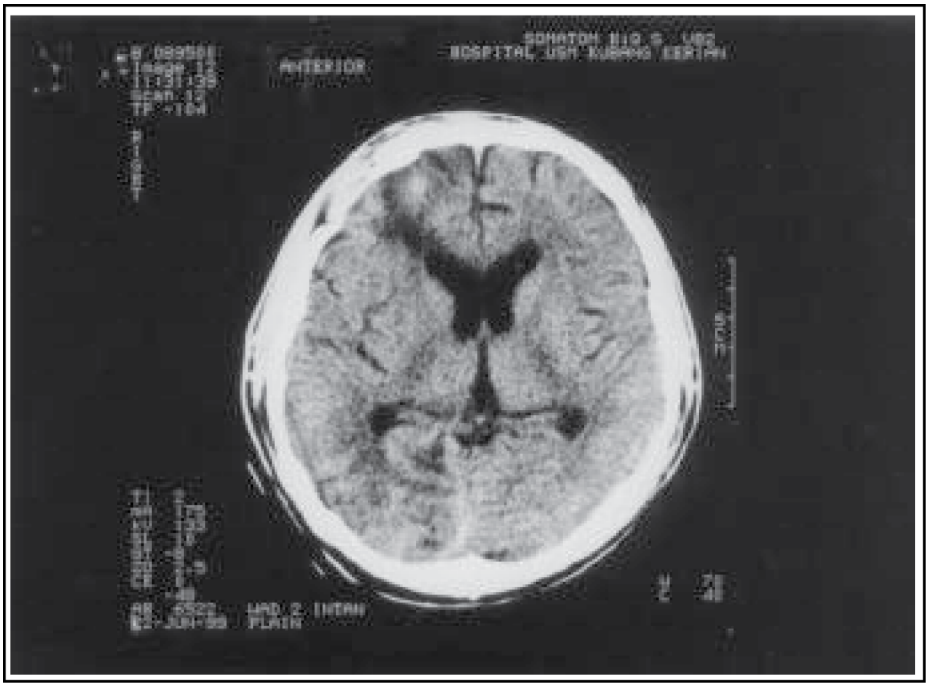
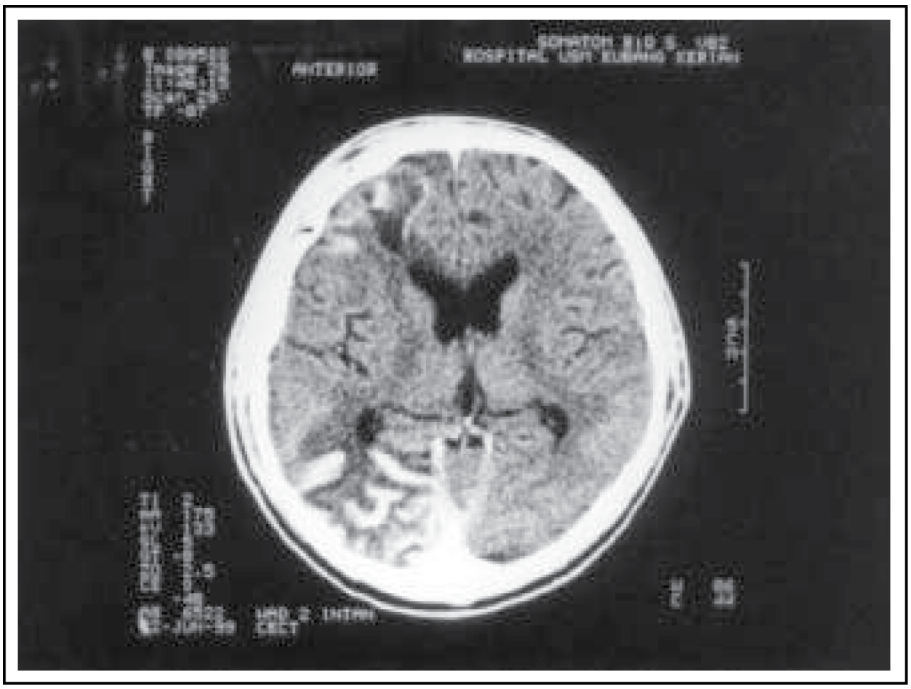


Figure 2 : Post contrast scan showed serpenginous gyriform enhancement around the contusion in the right frontal and occipital lobes.



of arteriovenous malformation was raised and cerebral angiography was suggested. However, he was managed conservatively and was discharged with GCS of 9/15. He was followed up in the clinic and noted to be improving slowly.

Four months after the accident, a repeat cranial CT scan was done. His GCS was full. He was well except for poor vision (only perception of light bilaterally), probably secondary to corneal ulceration. A contrast enhanced CT scan of the brain showed areas of encephalomalacia in the right frontal and occipital lobes following previous injury. The multiple serpenginous enhancement demonstrated in the previous CT scan was not visualized any more. There was also focal dilatation of the right frontal, occipital and temporal horns due to brain volume loss (Figure 3). From this CT scan, there was no feature to suggest arteriovenous malformation.

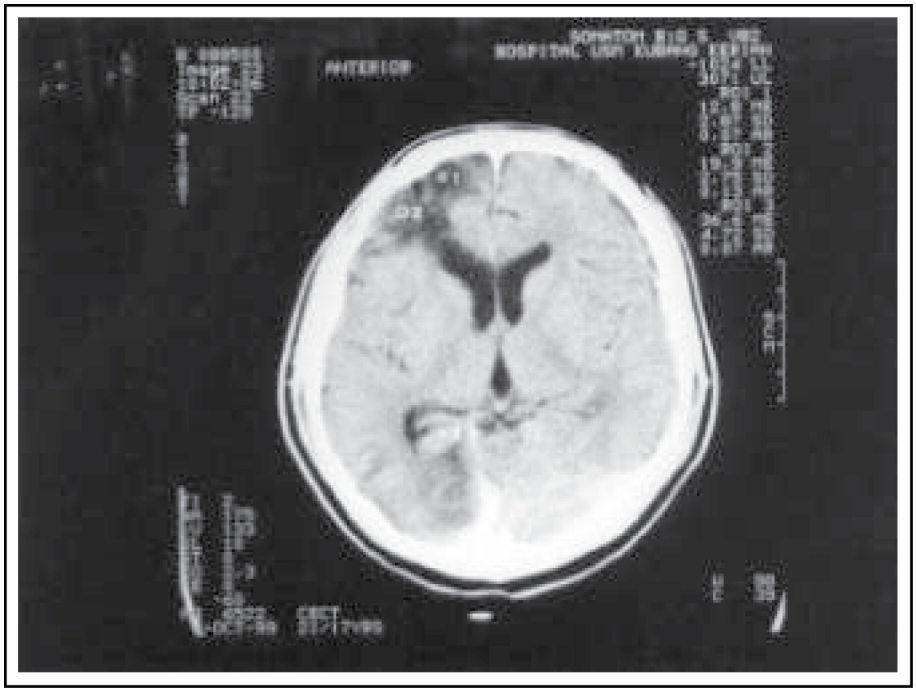
Discussion

Since the introduction of CT scan as a non-invasive technique for evaluating central nervous system pathology by Hounsfield in 1972, CT has revolutionized the management of head injury patients. It has replaced cerebral angiography for identifying brain herniation and intracranial mass lesions. In the acute setting, CT is also generally preferred over Magnetic Resonance Imaging (MRI) (1).

In a case of head injury, a complete CT evaluation includes 3 main window width and levels which are (i) brain window, optimized for brain; (ii) intermediate windows, to assess subdural or epidural hematoma; and (iii) bone window, used to determined bony fracture (2). Intravenous contrast is not normally administrated in an acute case and it may mask the presence of subarachnoid hemorrhage. However, in subacute or chronic head injury, contrast enhancement may be necessary to demonstrate an isodense hematoma, post-traumatic infarction, resolving hemorrhagic contusion and other complications such as empyema or intracerebral abscess.

In this patient, the initial CT scan done in this hospital was at 2 weeks post trauma. The findings of focal gliosis in the right frontal and occipital lobes reflect coup and contrecoup contusions, in which the frontal lobe lesion was most likely the coup injury. Intravenous contrast was administered then and demonstrated multiple serpenginous enhancement adjacent to and surrounding the region of contusion. It was thought that these enhancement were probably large draining veins and thus, arteriovenous malformation was suspected.

Figure 3 : A repeat contrast enhanced CT scan of the brain done 4 months post trauma. There were areas of encephalomalacia in the right frontal and occipital lobes. The multiple serpenginous enhancement demonstrated in the previous CT scan was not visualized any more.



However, upon reviewing the images again and after literature review, it was thought that the contrast enhancement could be just part of the course or pathophysiology of brain contusion.

Clinical studies suggest that focal hyperemia can occur following traumatic brain injury. Alexander M.J. et al (3) had performed a study on regional cerebral blood flow using ^{133}Xenon in head injury patients with focal contusion and cerebral edema. They had demonstrated that the initial oligemia in the contused area was associated with a subsequent hyperemic rim about the contusion. In another study by Sakas D.E. et al (4), using ^{99m}Technetium-hexamethylpropyleneamineoxime, hyperemic areas were demonstrated in 38% of patients with head injury. The hyperemia were commonest in patients with focal contusions and intracerebral hematomas. It affected both gray and white matter. From their study, most hyperemia was found to have disappeared by the second week after injury. However, in one of their patients, hyperemia was still present 3 weeks post injury. Recent tomography studies have demonstrated that hyperemia occurs most frequently between the second and fourth day after trauma.

Sakas D.E. et al (4) reported that hyperemia occurs only in normal tissue directly adjacent to focal mass lesions. However, Fumeya H. et al (5) had found hyperemia within the edematous tissue revealed by MRI. They had observed that these hyperemic lesions are likely to be related to post traumatic seizures and tend to dissipate on follow up MRI.

The incidence, pathophysiology significance and clinical relevance of hyperemia following head injury are not yet understood. Sakas D.E. et al (4) postulated that the swollen astrocytes found within the edematous tissue adjacent to contusions may cause a hyperemic response by compressing the microvasculature. They found that this focal and persistent hyperemia is "benign" and has minimal effect on intracranial pressure and level of consciousness. This "benign" form of hyperemia was associated with better outcome of the patient.

Besides the hyperemic phenomenon, there is another possibility for the enhancement demonstrated in this patient. It could be part of the neuropathological changes that occur as a cerebral contusion undergoes resolution. Four distinct phases have been described for brain contusion (1, 2). The first phase is the acute damage. The second stage is liquefaction of the contusion with development of edema. This occurs between the third and seventh day following injury. In the third phase, repair mechanism sets in. Macrophages remove the blood elements and the damaged tissue. Neo-vascularisation occurs around the area of healing. The new blood vessels lack tight endothelial junctures, causing blood brain barrier disturbance. At this point, within one week of injury, if a contrast agent is administered during CT scanning or MRI, enhancement will be seen at the margin of the contusion. The final stage of evolution occurs when the necrotic tissue is sloughed and cystic cavities are formed.

Arteriovenous malformation (AVM) is an admixture of arteries and veins. There are three morphologic components : the nidus; the feeding arteries and the draining veins. On non-contrast enhanced CT scan, intra-parenchymal AVM most commonly presents as an area of heterogeneous hyperdensity relative to brain parenchyma (2, 6, 7). The lesion is poorly defined and irregular in outline. Calcification within the vascular channels and adjacent brain parenchyma are often present. Following administration of intravenous contrast agent, there is ill-defined heterogeneous enhancement of the lesion. Feeding arteries or draining veins may be demonstrated as dilated vessels on contrast enhanced CT. Occasionally, AVMs are predominantly hyperdense and heavily calcified. The adjacent brain parenchyma shows destructive and atrophic changes.

Lobato R.D. et al (9) had found that 96% of AVMs were hyperdense (either homogeneously or mottled) on pre-contrast CT study and only 19% were hypodense with the remaining being isodense to the brain. From their study, 76% of AVM showed enhancement post intravenous contrast.

Correlating the CT findings with the clinical history of this patient, the serpenginous gyriform enhancement in the right frontal and occipital lobes are most appropriately explained by the focal hyperemic phenomenon following brain injury or as part of the evolution of cerebral contusion. AVM is very much unlikely and this is supported by the repeat CT scan which failed to demonstrate the similar enhancement.

Conclusion

Focal enhancement surrounding an area of brain injury or contusion could be the "benign" hyperemic phenomenon or as part of the evolution of cerebral contusion. This should not be confused with an AVM in which the patient might then be

subjected to further invasive investigation such as cerebral angiography.

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