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An Early CT Sign of Ischemic Infarction: Increased Density in a Cerebral Artery

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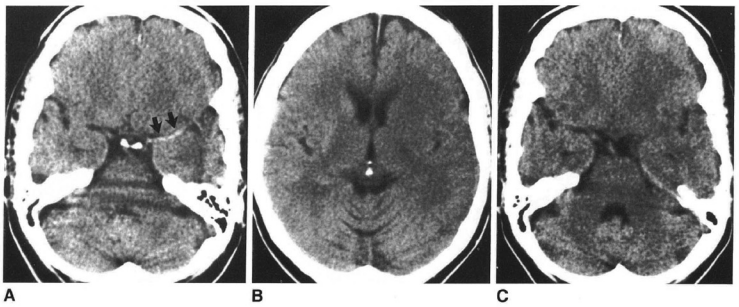
The diagnosis of ischemic cerebral infarction by CT usually is not poss hr after the event. A sign that allows earlier diagnosis is increased densit vessel secondary to a thrombus or embolus. We report 19 cases of incr in a middle cerebral artery or one of its major branches visible on the init patients subsequently clinically proven to have suffered a cerebrovascul three cases angiography confirmed the presence of embolus or thromb the CT scans. In several cases subsequent CT scans showed the densit thereby confirming its nature as thrombus or embolus. Recognition of t allow earlier diagnosis of ischemic infarction, which may be important in appropriate therapeutic regimen.

The early diagnosis of ischemic infarction by CT depends on the r often subtle changes of reduced attenuation and slight mass effect of cerebral infarction that may be present shortly after the ictus is incr in a major cerebral artery. Gacs et al. [3] reported this finding in a study, and they considered it most likely secondary to intraluminal cl

We have found this sign to be useful in the early recognition of no cerebral infarction. It has been seen in a number of patients in the fir the ictus, and frequently it is the only CT finding or is an important finding in association with other subtle changes of infarction.



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Carotid Cavernous Fistulae: Indications for Urgent Treatment

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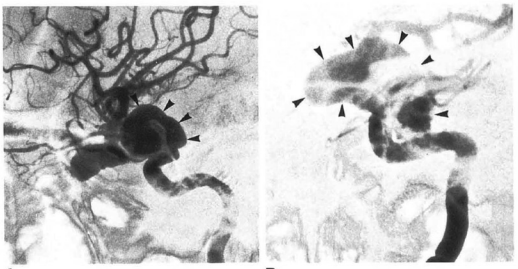
Angiographic and clinical data from 155 patients with carotid caverno retrospectively reviewed to determine angiographic features associate risk of morbidity and mortality. These features included presence of a ps large varix of the cavernous sinus, venous drainage to cortical veins, an venous outflow pathways distant from the fistula. Clinical signs and characterized a hazardous carotid cavernous fistula included increa pressure, rapidly progressive proptosis, diminished visual acuity, he transient ischemic attacks. Cortical venous drainage from the carotid c is secondary to occlusion or absence of the normal venous outflow p associated with signs and symptoms of increased intracranial pressure a risk of intraparenchymal hemorrhage. Angiographic demonstration of a c varix, with extension of the sinus into the subarachnoid space, is ass increased risk of fatal subarachnoid hemorrhage. Identification of thes tures provides a basis for making decisions about treatment.

Carotid cavernous fistulae (CCFs) are spontaneous or acquire between the carotid artery and the cavernous sinus, and can be clas or indirect. Direct connections between the internal carotid artery sinus may occur as a consequence of trauma, ruptured intracav aneurysms, collagen deficiency syndromes, arterial dissection, fibromuscular dysplasia, and direct surgical trauma [1–10]. Indirect fistulae are usually supplied from dural branches of the external carotid artery but can be supplied from dural branches of the internal carotid artery. Although the cause is often unknown, factors associated with the development of indirect fistulae include pregnancy, sinusitis, trauma, surgical procedures, and cavernous sinus thrombosis. Symptoms caused by CCFs are related to their size, duration, location, adequacy and route of venous drainage, and presence of arterial and venous collaterals [11].

Surgical and angiographic techniques that have been described for the closure of CCFs include carotid occlusion; trapping procedures; direct surgical exposure and closure; and embolization with muscle, glue, thrombus, wires, and, more recently, detachable balloons [12–19].

Unfortunately, the natural history of CCFs is incompletely understood. Spontaneous closure, which is more common in indirect than direct CCFs, as well as closure following diagnostic angiography, has been documented by Seeger et al. [20]. Carotid compression therapy has been successful in closure of 17% of direct and 30% of indirect CCFs [21]. While aggressive forms of therapy may be successful in closure of CCFs, no technique is without risk. Ideally, the decision to institute a potentially hazardous treatment should be based on full understanding of the disease's natural history.

To identify those patients whose poor natural history mandated the need for emergent or aggressive therapy, we evaluated the angiographic and clinical data from 155 patients with CCFs. The delineation of high-risk features enables rational choices to be made regarding the timing and method of treatment.



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