

eral times per day and she was then weaned off ketamine. At this time the patient was on phenytoin and phenobarbital and remained in a coma. There were no adverse effects that could be ascribed to the use of ketamine. During the following 3 months the patient developed intermittent seizures and has profound short-term memory and global cognitive deficits. A cranial MRI study at this time showed a moderate degree of diffuse cerebral atrophy.

**Discussion.** This patient had SE that was refractory to treatment with phenobarbital, phenytoin, continuous midazolam infusion, propofol, IV valproate, or IV lidocaine infusion. Pentobarbital coma induced for 4 weeks was not sufficient to control her SE.

SE refractory to all currently recommended medications is a relatively rare but challenging neurologic problem. A recent review of the management of SE did not list ketamine as a therapeutic agent.<sup>4</sup> In our patient, IV ketamine immediately controlled both clinical and electrographic seizures (see figure), although gradually higher doses of ketamine were required to maintain the response. Animal data suggest that ketamine may possess anticonvulsant properties, possibly by binding to the phencyclidine recognition site of the NMDA receptor.<sup>5</sup> NMDA receptor blockade may be a protective response to the deleterious effects of excessive excitatory amino acid.<sup>6</sup> Accordingly, ketamine may have both an anticonvulsant and a neuroprotective effect in SE.

Ketamine has a half-life of 2 to 3 hours and is extensively and hepatically metabolized via cytochrome P450-mediated oxidative pathways to the active metabolite norketamine, which is pharmacologically active. Pharmacokinetic interactions with concomitant antiepileptic medication is not well defined, although there does appear to be significant interaction. The patient's age and the effect of a co-administered enzyme-inducing anticonvulsant medication may underlie her requirement for an increasing dosage of ketamine to maintain the anticonvulsant effect.

Ketamine may prove to be a useful adjuvant in the treatment of refractory SE. The pharmacologic characteristics and potential neuroprotective properties of ketamine may make it particularly attractive in the therapy of refractory SE, although confirmation of our observation of efficacy is required.

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## Multifocal brain MRI hypointensities

### secondary to embolic metal fragments from a mechanical heart valve prosthesis: A possible source of epileptic seizures

Markus Naumann, MD; Erich Hofmann, MD; and Klaus V. Toyka, MD

Recently, Wingerchuk et al.<sup>1</sup> described a patient with brain MRI lesions secondary to embolic metal fragments from a Bjork-Shiley heart valve prosthesis. The incidence and possible clinical symptoms of such lesions are unknown. We present another patient showing similar lesions on MRI who, in addition, had epileptic seizures. This raises the question of whether metal deposits may be associated with focal neurologic symptoms.

**Case report.** A 66-year-old man had an aortic valve prosthesis (Starr-Edwards) in 1969 to treat aortic valve stenosis. Twenty years later, this valve was replaced by a St. Jude prosthesis be-

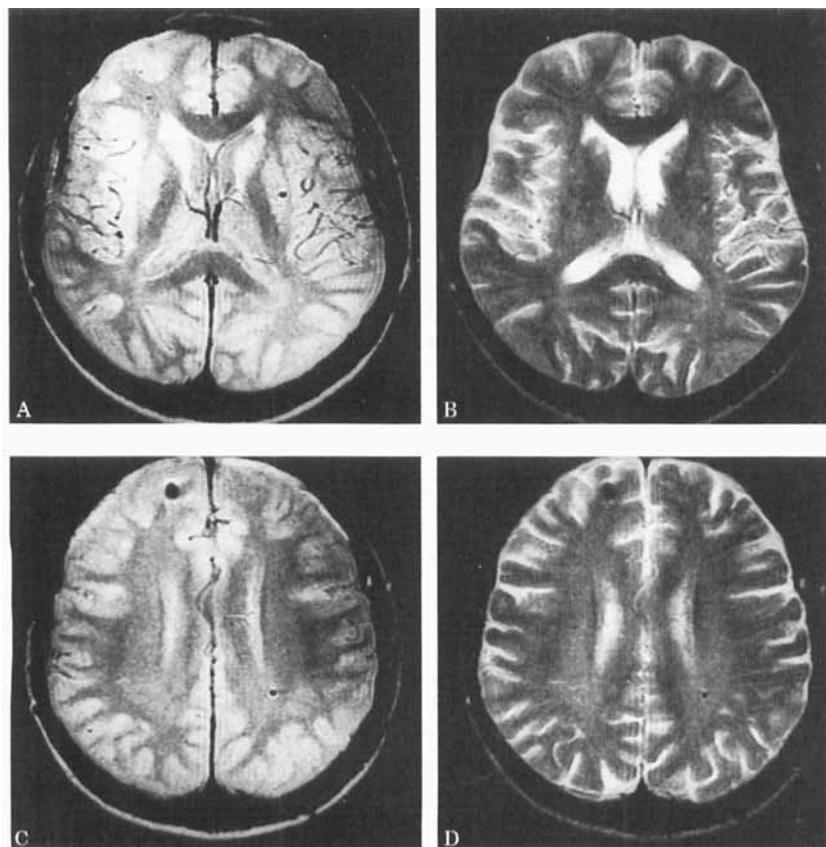


Figure. Proton-density (A, C) and T2-weighted (B, D) MR images (spin echo 2,420/30,100). Bilateral lacunar infarcts in the caudate heads (B). Punctate susceptibility artifacts in both hemispheres (basal ganglia, medullary layer, subcortex) typical for metallic emboli.

cause of continuously deteriorating pulmonary artery hypertension. Macroscopically, no fracture of the removed Starr-Edwards valve and no thrombotic material were seen.

In 1989, a first generalized tonic-clonic seizure occurred during sleep, and anticonvulsant therapy with phenobarbital was initiated at an outside hospital. In addition, complex-partial seizures occurred that were described by the patient as isolated, recurrent short episodes of a dreamy state with ascending nausea, occasionally associated with visual disturbances. Approximately 10 to 20 complex-focal seizures occurred per month, with a duration of about 10 seconds. In 1997, the patient was admitted to our department for the first time for better seizure control. On admission, neurologic examination, laboratory tests, and CSF were normal, and no cardiac output failure was present. Repeated EEG studies showed intermittent right-sided frontotemporal slow wave bursts.

On cranial MRI, several punctate hypointensities were seen randomly distributed over both hemispheres (figure, A through D). No other pathology could be found in the temporal lobes even on specifically angulated high-resolution coronal series. The lesions were especially prominent in the proton-density images of the T2-weighted spin echo series. They showed a peripheral hyperintensity mainly at the rostral and dorsal margin, i.e., perpendicular to the direction of the phase encoding gradient, thus fulfilling the criteria for susceptibility artifacts from metal emboli.<sup>2</sup>

**Discussion.** The susceptibility artifacts in the brain of our patient resemble the lesions reported by Wingerchuk et al.,<sup>1</sup> and most likely reflect shedded metal material from the original Starr-Edwards valve prosthesis. Although in the report by Wingerchuk et al. a fracture-prone, but not obviously broken, Bjork-Shiley valve was associated with brain metal emboli, brain metal artifacts were observed in our patient after implantation of a Starr-Edwards prosthesis that looked macroscopically normal when removed. Thus, metal emboli from unbroken mechanical heart valve prostheses are possible.

It remains unclear whether some of these metal fragments are the cause of the epilepsy in our patient. Because we did not find any other structural abnormalities, this appears likely. In our patient, punctate susceptibility artifacts were seen in both hemispheres, especially in the basal ganglia, the medullary layer, and the right frontal subcortex. The lesions in the basal ganglia and the medullary layer most likely do not contribute to epileptic seizures, and a causal association between the right frontal subcortical lesion and complex-focal seizures is conceivable. This localization would also be in accordance with the intermittent right-sided frontotemporal slow wave bursts seen on EEG. Further support could be adduced by depth electrode recordings; these were not obtained because the patient became seizure free with carbamazepine. Metal emboli may be more common after cardiac valve implantation than previously estimated but may not be detected because patients cannot be studied with MRI with these valves in place.

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## Giant cell arteritis presenting as cluster headache

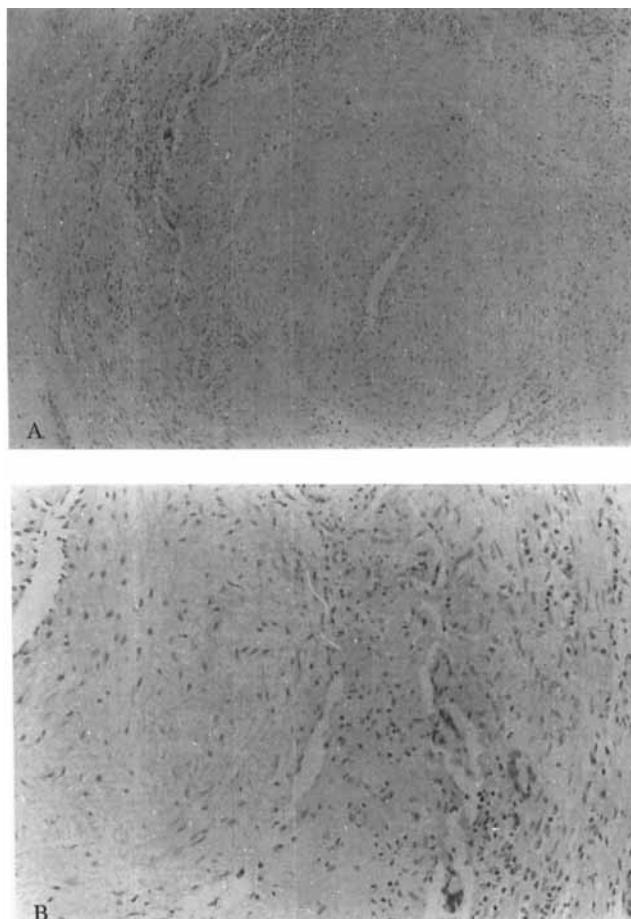
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New onset of headache in an elderly patient is a common presenting symptom of giant cell arteritis. Headache is usually general-

ized, throbbing, and continuous, with focally worse pain in the temporal or, less often, in the occipital regions.<sup>1</sup> We report a patient with a histopathologically confirmed giant cell arteritis whose clinical manifestations mimicked those of cluster headache.

**Case report.** A 74-year-old man without previous personal or familial history of headache was evaluated in our hospital because of a 2-month history that consisted initially of one to two attacks per day and progressed to three to four attacks per day, of severe, boring pain in the left supraorbital area that lasted 15 to 20 minutes. The pain episodes were associated with ipsilateral ptosis, lacrimation, and rhinorrhea, but not with ipsilateral sweating. The patient had no pain between these episodes. Although the headaches did not occur at the same time each day, one of the episodes usually occurred during nocturnal sleep, awaking the patient. He also complained of jaw claudication with chewing and pain in the scapulohumeral joints, although chewing did not precipitate the headache episodes. Neurologic examination, including temporal artery palpation, was normal. The patient was a heavy smoker and had antecedents of chronic obstructive airway disease, cor pulmonale, and a peripheral artery embolism that required left leg amputation.

Blood analyses showed, as the most outstanding features, a mild leukocytosis (12,700 white blood cells/mm<sup>3</sup>) and increased erythrocyte sedimentation rate (83 mm in 1 hour). Brain CT was normal. Histopathologic study of the left temporal artery confirmed the diagnosis of giant cell arteritis (figure). Following initiation of treatment with prednisone 60 mg/day, the patient became asymptomatic. Prednisone dose was tapered gradually in 3 months. After 6 months of follow-up, the patient remains asymptomatic, and erythrocyte sedimentation rate decreased to 11 mm in 1 hour.



**Figure.** Temporal artery shows thickening of the intima, lymphocytic infiltration of the media with multinucleated giant cells, and partial destruction of the internal elastic lamina (H-E, original magnification  $\times 80$  (A) and  $\times 160$  (B) before 42.3% reduction).

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