Arterial Dilatation in Purulent Meningitis*

Case Report

DAVID O. DAVIS, M.D., DOMENICO DILENCE, M.D.,† AND WILLIAM SCHLAEPFER, M.D.
The Edward Mallinckrodt Institute of Radiology, and Department of Pathology,
Washington University Medical School, St. Louis, Missouri

ALTHOUGH angiographically visualized changes in the arteries have been described in patients with tuberculous and fungal meningitis, other types of meningitis have not been studied extensively by this technique. Those patients in whom purulent meningitis is found are usually studied only when there is suspicion of a mass lesion or cerebritis secondary to or causing the meningeal inflammation. Thus, the signs that may be present are little known, and a recent case prompted this report.

Case Report

This 48-year-old woman was admitted to the hospital having been unconscious for 12 hours. There were no symptoms until the evening before admission when she developed a mild headache. Two hours later she was found to be unresponsive, and had a convolution involving the right side.

In 1959 she had been in an automobile accident, was comatose for 3 days, and was left blind. The right eye was removed at this time because of the injury.

Examination. The patient had tachycardia, normal blood pressure, tachypnea, and a rectal temperature of 37.5°C. She was totally unresponsive. The neck was moderately stiff. There was diffuse hyperreflexia. The fundus on the left could not be seen because of scarring. Some involuntary movements of the right side were noted. Spinal puncture revealed purulent fluid containing 1100 mg% of protein and no sugar. Gram stain and culture revealed pneumococci. She was treated with large doses of penicillin, Keflin, and Chloromycetin. There was no neurological improvement, although the spinal fluid eventually cleared. Serial electroencephalograms were abnormal, showing slowing, shifting foci, and terminal flattening. Skull x-ray films revealed the presence of a radiographically dense but non-metallic foreign body overlying a left orbital fracture. Left carotid angiography showed slow transit of contrast material through the cerebrovascular tree with slight narrowing of the major arteries at the base and in the interhemispheric fissure (Figs. 1 and 2). No definite midline shift was identified. The most conspicuous finding was a segmental dilatation involving the cortical branches of the anterior and middle cerebral arteries. Arterial segments enlarged to twice the normal measurement were particularly evident immediately outside of the interhemispheric and Sylvian fissures. These segments, in contrast with other arteries, remained opacified for a long period of time (6 sec), and traces of contrast material were found within their lumen even on the last exposure of the series. The beginning of the venous phase was normal. Presumably, the dilated segments caused a significant slowing of the flow and were responsible for the increase in angiographic circulation time. The patient developed gastrointestinal bleeding, acute renal failure, and tracheobronchial hemorrhage. She died 30 days after admission.

Postmortem Examination. An attempt at angiography showed the vessels to be of relatively normal size, but the filling may have been inadequate. Gross examination of the brain revealed congestion and slight opacification of the leptomeninges. The major cerebral arteries contained some atheromatous deposits but showed no areas of significant narrowing. Encephalomalacia was present in the distal cortical distribution of the left middle cerebral artery, especially in the medial frontal gyrus. Small foci of ischemic necrosis were also detected in the anterior cortical distribution of the right middle cerebral ar-
tery. A slight unilateral enlargement of the right cerebral hemisphere associated with minimal notching of the left uncus was seen; there were no brain-stem hemorrhages or necrosis.

Microscopic examination showed a variable degree of fibrosis and chronic cellular infiltrate in the subarachnoid space. These changes were particularly prominent over the areas of cortical necrosis in the left frontal lobe. In this region the arteries and veins which course through the subarachnoid space were encased within a mantle of fibrous tissue. Although many of the small arteries in this area appeared dilated, no inflammatory infiltrates were observed in the walls of these vessels (Fig. 3). The underlying cortex showed the changes of an organizing infarct with early cystic changes, numerous gitter cells and gemistocytic astrocytes. The small blood vessels were free of thrombotic or embolic material. No evidence of acute inflammation or abscess formation was seen. Multiple sections at successive levels of the left middle cerebral artery revealed scattered chronic inflammatory cells in the surrounding subarachnoid space but no intrinsic structural change in the tunica muscularis or membrana interna elastica of this vessel.

Discussion

Narrowing of the supraclinoid carotid arteries due to granulomatous arteritis has been demonstrated in patients with tuberculous meningitis. In purulent meningitis, however, the etiology of the angiographically demonstrated arterial narrowing is unknown. Presumably the surrounding inflammatory material irritates the arterial wall, causing muscular contraction, which narrows the lumen.

Our case shows, in addition to narrowing of the arteries at the base and in the interhemispheric and Sylvian fissures, diffuse dilatation of the surface arteries. The latter finding, to our knowledge, has not been previously reported. Its cause is obscure. No arteritic changes were identified in the histologic specimen, but the patient had received several weeks of antibiotic therapy between the time of the angiogram and her death.
Fig. 2. Upper: Lateral view (2.0 sec). There is continued flow of the contrast material at a very slow rate into the dilated arteries, which are mostly on the surface of the convexity. Many vessels are not affected, and the circulation through these vessels was not appreciably slowed. The arterial pCO₂ at the time of the angiogram was 29 mm Hg. Lower: Venous phase (7.0 sec). Normally filled veins are well seen but residual contrast is still present in some of the dilated arteries (arrows).
This might have caused resolution of some inflammatory changes. However, residual inflammatory changes were present in the periarterial subarachnoid space.

One hypothesis for the dilatation of the arteries is atony of the musculature caused by severe involvement of the arterial wall by the surrounding infection. Another possibility is the presence of periarterial adhesions holding the walls of the vessel open. Alternatively, regionally increased tissue pH secondary to local necrosis could cause capillary and arterial dilatation, such as occurs in some cases of cerebral infarction. Finally, dilatation of the arteries secondary to obstruction of the capillary bed by extensive thrombosis is a possibility, but such a change was not identified in the pathological specimen. It is our feeling, therefore, that atony of the arterial wall seems the most likely explanation for the findings.

**Summary**

We have reported a patient with purulent meningitis secondary to a fracture of the orbit in whom an angiogram showed marked dilatation of the hemispheric surface arteries. Histological examination showed no arteritis. Atony of the arterial wall may have caused the dilatation.

**References**