Escherichia Coli Meningitis in Adults: Neurosurgical and Neuropathological Considerations*

FRANKLIN ROBINSON, M.D., JACQUES B. LAMARCHE, M.D., AND GILBERT B. SOLITARE, M.D.
Department of Pathology, Yale University School of Medicine, New Haven, Connecticut

Meningitis resulting from infection with Escherichia coli is uncommon in adults1,18,22,24 when compared to the relative frequency of occurrence in infants.20 In adults E. coli meningitis has been reported associated with urinary tract infection,18,23,26,27 abortion,15,22 and liver disease,10,20 as well as in instances without a readily demonstrable portal of entry or primary focus of infection.21,29 Meningeal infection is probably blood-borne in most cases2,5,14 excluding those infections resulting from penetrating head wounds of combat,7,16 compound fracture of the skull,22 or contamination of the cerebrospinal fluid during lumbar puncture.12,17

The clinical findings in three adults with E. coli meningitis are presented with emphasis upon certain neurosurgical implications arising during the course of the illness. Two of these patients died, one entirely unsuspected of harboring this intracranial infection; the third developed persistent hydrocephalus, which was ultimately relieved by a cerebrospinal fluid shunt. Postmortem observations are also presented to further our awareness of this organism as it affects the nervous system of the older individual.

Case Reports

Case 1. A 57-year-old white housewife was admitted on August 9, 1965, in a confused, tremulous state. Nine days earlier she had sustained contusions of the right orbital region. There was a history of excessive alcohol intake for many years, with several episodes of delirium tremens.

Examination. The patient was restless and incoherent and had visual hallucinations. Her temperature was 103.8°F. There were extensive ecchymoses of the right peri orbital and temporal regions and a stiff neck. The lower border of the liver was 3-fingers’ breadth below the right costal margin. There was good muscle strength of the extremities, and the deep tendon reflexes were symmetrically active except for the ankle jerks, which were absent. A Babinski sign was observed on the right side. The patient perceived painful stimuli, but finer sensory testing could not be evaluated.

The clinical impression was meningitis, although subdural hematoma, delirium tremens, and hepatic encephalopathy were also considered. The white blood cell count was 5200 per cu mm. X-rays of the skull and chest and an echoencephalogram were negative. Lumbar puncture yielded cloudy-yellow fluid under a pressure of 80 mm of water; the white blood cell count was 3000 per cu mm, virtually all polymorphonuclear leucocytes, and the red blood cell count 8000 per cu mm. The CSF protein was 730 mg%, and glucose was 49 mg%. A spinal fluid smear revealed gram-negative rods, and both blood and CSF cultures grew E. coli. The patient was started on treatment with intravenous cephalothin, chloramphenicol, and colistimethate sodium. Corticosteroids were given, and a hypothermic blanket reduced her body temperature to normal. Lumbar puncture on the third day revealed cloudy xanthochromic CSF under pressure of 140 mm of water, a protein of 920 mg%, a glucose of 103 mg%, and a white blood cell count of 3790 per cu mm with 80% polymorphonuclear leucocytes. An electroencephalogram showed diffuse slow-wave activity.

The patient remained in a coma, deteriorating gradually without localizing neurological signs. She died on the seventh hospital day.

General postmortem findings. There were ecchymoses over the right temporal and periorbital regions. No fracture of the skull or nose was found. The liver showed fatty change and portal fibrosis, the pancreas was diffusely fibrotic. E. coli were cultured from the CSF and blood.

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Neuropathologic observations. There was no blood within the subdural space. The brain weighed 1200 gm. The leptomeninges over the convexity and basilar surfaces of the brain were clouded by yellow-green exudate. The gyri were flattened and the sulci narrowed. There was minimal arteriosclerosis.

After fixation in 10% formalin for 1 week the brain was coronally sectioned. The ventricular system was moderately and uniformly dilated. The ventricular surfaces were covered with a thick, shaggy, yellow-green exudate. No other lesions were observed grossly within the cerebrum, cerebellum, brain stem, or spinal cord. Blocks of tissue from the cerebrum, including basal ganglia, pons, medulla, cerebellum, and spinal cord were embedded in paraffin, sectioned, and stained with hematoxylin and eosin, and with special stains for bacteria and fungi. A heavy exudate, composed predominantly of polymorphonuclear leucocytes with some mononuclear cells, erythrocytes, and fibrin, was seen within the subarachnoid space extending deeply along the perivascular spaces of the cerebrum. The exudate was most prominent in the cerebellar and medullary subarachnoid space, where there was an acute arteritis and phlebitis (Fig. 1). Similar vascular lesions were observed in the subependymal regions. The ependymal lining was coated with a purulent exudate which was also present in the choroid plexus and among the cranial nerve roots. There were foci of astrocytic and microglial proliferation in the superficial layers of the cerebral cortex and focal loss of neurons in the cerebellum. The special stains for fungi and bacteria were unrevealing.

Case 2. A 69-year-old white man was admitted on June 27, 1966, in an unconscious state. He had fallen and had sustained a head injury at home following an episode of heavy drinking 3 days before. He had remained at home in a dull state of consciousness but partially responsive for 1½ days, and was then admitted to another hospital where x-rays revealed a skull fracture. He developed increasing depth of coma; focal seizures of the right side of the face and right arm appeared 12 hours before his transfer to this hospital. For the preceding 10 years he had had diabetes mellitus which was being treated with Orinase.

Examination. The patient was unresponsive to painful stimulation. His temperature was 102.8°F. There were no localizing neurologi-