was used to localize the lesion. The lesion was located relatively deep into the brainstem but was closer to the posterior surface and was to the right of the midline. An incision was taken in the region of the pontomedullary junction in the midline at the level of the lesion. Blunt dissection was used in a vertical direction within a limit of about 4-5 mm using number 7 Rhoton microdissector. The lesion was encountered at a depth of about 4 mm. The lesion was then carefully dissected from the surrounding structures using bipolar diathermy, as well as sharp and blunt dissection. With this technique, it was easy to deliver the tissue as a whole through the small opening. Postoperative imaging confirmed complete resection of the lesion (Figure 1b). Following surgery, the patient developed vertical nystagmus and his diplopia persisted. His left hemiparesis and unsteady gait improved.

Intra cranial cavernomas constitute about 5-13% of intracranial vascular malformations and 10-30% of these are located in the posterior cranial fossa.\textsuperscript{[5-6]}

Cavernomas often have a rim of gliosis with haemosiderin deposit surrounding it following previous bleeding, thus making surgical dissection from adjoining brain tissue relatively easy. However, when the lesions are located deep in the brain parenchyma, the dissection has a potential risk of injury. Thus, a safe route of access is crucial. The incision on the brainstem and direction of further dissection within it should take into consideration the orientation of the surrounding structures.

In the presented case, the lesion was located relatively deep in the brainstem from the surface and was to the right of the midline. A direct incision over the site of location of the lesion could have resulted in damage to underlying structures like facial colliculus, vestibular nuclei and hypoglossal nucleus. Considering the location in proximity to the midline, an incision simulating midline myelotomy was made. Neuronavigation technique with intraoperatively acquired MRI images helped to localize the lesion. The incision in the brainstem and the further dissection were done in a vertical direction to protect the adjoining critical neural structures.

\textbf{Augustine A. Adeolu}

\textit{Department of Clinical Neurosciences, Foothills Hospital/University of Calgary, Alberta, Canada}

\section*{References}


\section*{Meningitis due to \textit{Escherichia vulneris}}

\textbf{Sir,}

A right-handed 4-year-old girl presented with a history of trauma as a result of a fall from stairs. Clinical examination revealed an open injury over the left fronto-parietal region with brain herniation and cerebrospinal fluid (CSF) leak. Computed tomography scan showed a compound fracture of the left frontal bone extending to the parietal bone and orbit with an underlying contusion. An emergency surgery was performed with left frontal exploration, removal of loose bone fragments, wound debridement, evacuation of contusion and duraplasty. She was discharged on the 13\textsuperscript{th} post-operative day with a Glasgow Coma Scale (GCS) of E\textsubscript{4}V\textsubscript{4}M\textsubscript{2}.

Two days after discharge, she presented with neck rigidity and purulent discharge from the operative scalp wound. She was febrile, emaciated and dehydrated, had a blood pressure of 100/80 mm Hg and a pulse rate of 100/min. GCS was E\textsubscript{2}V\textsubscript{3}M\textsubscript{1}. She had ptosis of left eye with synecchia formation, right-sided facial nerve paresis and paresis of the right upper and lower limbs. The wound site showed a copious CSF leak with flakes of purulent material.

Laboratory examination revealed hemoglobin 9.9 gm/L, a white blood cell count of 7.4 x 10\textsuperscript{9}/L and platelet count of 110 x 10\textsuperscript{9}/L. Routine blood chemistry and coagulation tests were normal. Cerebrospinal fluid obtained by lumbar puncture revealed a raised leucocyte count of 2500/mm\textsuperscript{3}, elevated protein level of 127 mg/dl and decreased glucose level of 39 mg/dl. The CSF along with the locally purulent material, blood and urine specimens of the patient were sent for culture and empirical therapy was started with parenteral ceftriaxone, amikacin and metronidazole. The CSF (both lumbar puncture and local discharge) culture yielded \textit{Escherichia vulneris} identified by standard biochemical tests\textsuperscript{(1)} and confirmed by API 20E test strips (Bio-Mérieux, Marcy l’ Etoile, France). The organ-
ism was a non-lactose fermenting, motile, gram-negative bacterium, catalase positive, oxidase negative, did not produce indole and did not decarboxylate ornithine. In a standard disk diffusion method,[2] the organism was susceptible to piperacillin, β-lactam–β-lactamase inhibitor combinations (piperacillin-tazobactam, cefoperazone-sulbactam, ticarcillin-clavulanic acid) and meropenem. The strain tested positive for the production of extended-spectrum-β-lactamase.[2] Therapy was changed to parenteral piperacillin-tazobactam combination. However, the patient’s clinical condition did not improve and subsequently therapy with parenteral meropenem was initiated. The patient started to improve neurologically (GCS = E4V5M6), however, the wound site failed to epithelialize. On the 25th day of admission, exploration of the scalp wound site was performed, the underlying necrotic bone fragments were removed and debridement done followed by primary dural closure. A rotation skin flap was utilized for primary skin closure. Antibiotics were continued postoperatively till the patient was discharged on the 35th day of hospitalization with a healthy wound.

In 1982, Brenner et al[3] classified Escherichia vulneris as a new species in the family Enterobacteriaceae on the basis of DNA relatedness studies and biochemical reactions. Most isolates of E. vulneris have been recovered from wounds (vulneris [Latin]: of a wound).[3,4] However, it is questionable whether they are pathogenic or not, since they fail to produce infection in mice.[4] Reports of invasive infections due to E. vulneris are relatively few and include osteomyelitis,[5] urosepsis,[6] bacteremia,[7] and septic shock.[8] No fatal cases have been reported so far. The majority of the patients were adults.[3,4,6,8] A review of literature revealed no previous report of bacterial meningitis due to this organism.

In wounds with E. vulneris infection, co-infection with other bacteria has been observed which may have contributed to the extensive tissue injury seen in such cases.[4,8] However, E. vulneris was the sole pathogen in cases of osteomyelitis,[5] urosepsis,[6] bacteremia[7] and in the present case of meningitis. Thus, E. vulneris may be a potential pathogen and more studies are needed to explore the virulent nature of the organism. Clinical isolates of E. vulneris are reportedly susceptible to commonly used antibiotics.[3,4,7] In contrast, the isolate infecting our patient was multidrug resistant, suggesting that the infection was nosocomial.

Srujana Mohanty, Sharat P. Chandra, Benu Dhawan, Arti Kapil, Bimal K. Das
Department of Microbiology, All India Institute of Medical Sciences, New Delhi - 110 029, India. E-mail: tezpur@yahoo.com

References
2. National Committee for Clinical Laboratory Standards, Performance standards for antimicrobial disk susceptibility testing; Twelfth informational supplement. Wayne PA: NCCLS; (M100-S12), 2002.

Accepted on 23.04.2004.