Meningitis Due to *Enterobacter aerogenes* Subsequent to Resection of an Acoustic Neuroma and Abdominal Fat Graft to the Mastoid

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Meningitis is an uncommon complication of neurosurgical procedures, with an incidence of 1.1% to 2.5%. Although unusual, the frequency of nosocomial Gram-negative meningitis appears to be increasing. Gram-negative meningitis has been documented following disruption of the dura-arachnoid barrier secondary to trauma or surgery. The association of Gram-negative bacillary meningitis with neurosurgical procedures was first reported in the 1940's. Wolff et al. described the association between *Enterobacter* species and post-neurosurgical infection. More recently, risk factors for nosocomial *Enterobacter* meningitis have been characterized by Parodi et al. Adipose graft, as an independent risk factor has not yet been reported. A patient with acoustic neuroma resection, who developed bacterial meningitis from an abdominal fat pad graft to a mastoidectomy bed is described. A brief overview was made of post-neurosurgical Gram-negative meningitis.

Key Words: Enterobacter, meningitis, neuroma and translabyrinthine resection.

Case Report

A 47-year-old male underwent translabyrinthine resection of a left acoustic neuroma, with an abdominal fat graft to the mastoid. Perioperatively, the patient received 1g of nafcillin intravenously every four hours.

On postoperative day 4, patient started to run a temperature of as high as 39.7° C, along with neck stiffness. On postoperative day 7, a spinal tap was performed; it revealed cloudy cerebrospinal fluid, with a profile consistent with pyogenic meningitis. *Enterobacter aerogenes* was isolated from the spinal fluid and from an anaerobic culture of one set of blood cultures. His antimicrobial regimen was changed to 2g ceftriaxone, intravenously every eight hours. However, the patient continued to spike high-grade fevers over the next 48 hours, at which juncture ceftriaxone was substituted with meropenem, metronidazole and vancomycin.

There was no radiological evidence of intracranial thrombosis or CSF leakage. However, there was a 4cm x 1.5cm region of soft tissue type attenuation, with gas, adjacent to the mastoidectomy surgical bed, along the inferior portion of the fat packing, extending from the region of the mastoid tip to the level of C2 on MRI. This was interpreted as post-operative changes by the surgical teams. Despite sterilization of cerebrospinal fluid upon serial lumbar punctures, no growth from blood cultures, and negative chest films, the patient continued to have low-grade fevers. Repeat MRIs of the brain revealed inflammatory changes within the left mastoid/petrous bone, with enhancement at the periphery of the fat packing.

Discussion

Post operative infections of the meninges, following resection of an acoustic neuroma, result from iatrogenic contamination of the cerebrospinal fluid, either directly

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during surgery, or indirectly by creating a defect through an infection defense barrier, providing a route of entry for organisms that invade the meninges, inciting immune inflammatory response associated with vasculitis and infarction [1-3].

Gram-negative bacilli are rarely a cause of central nervous system infection in adults, but they have become increasingly common in patients with a history of head trauma, neurosurgical operations, and impaired host defenses [1,2,4]. Meningitis was seen in 8.3% of patients who underwent a trans-labyrinthine approach to acoustic neuroma resection [5].

Table 1. Etiologic agents in patients with Gram-negative bacillary meningitis [2]

Organisms	Number of cases
Klebsiella species	6
Escheridia coli	4
Acinetobacter baumannii	5
Morganella morganii	1
Serratia marcescens	1
Citrobacter freundii	1
Enterobacter cloacae	1
Proteus mirabilis	1

Korinek et al., in a prospective trial of 3,000 patients, described meningitis or deep abscess in 2.5% of craniotomy patients, with symptoms developing on average by postoperative day 10, with a range of 2 to 18 days. They reported *Staphylococcus aureus* as the most common cause, followed by *Enterobacter* in 24% of the cases [6]. *Enterobacter* species are increasingly the cause of nosocomial meningitis among neurosurgery patients, and this has been documented following disruption of the dura-arachnoid barrier, secondary to trauma or surgery [7,11].

Parodi et al., in their eight-year study, found 15 post-neurosurgical cases of *Enterobacter* meningitis, *E. aerogenes* species, being isolated from CSF samples of 16% of the study population.

Table 2. Cerebrospinal fluid (CSF) microbiological data from 20 neurosurgical patients [8]

Organisms isolated from CSF samples	Number of cases
Enterobacter aerogenes	9
Enterobacter cloacae	6
Coagulase-negative *staphylococci*	3
Propionibacterium acnes	1
Klebsiella pneumonia	1

Risk factors for these types of infections are not clearly defined. A recent matched-case-control study, comparing 13 case patients with 26 controls, indicated external CSF drainage devices, prolonged administration of antimicrobial drugs prior to the diagnosis of meningitis, and isolation of *Enterobacter* from a non-CSF culture, as independent risk factors for *Enterobacter* meningitis [8]. *Enterococcal* meningitis following PEG tube placement has also been described [9]. To date there have been no studies postulating the role of abdominal fat graft as an independent risk factor for the development of *Enterobacter* meningitis. There is a potential risk of contamination of fat cells when exposed to air and the location of the harvesting, but there are no published data [10].

Unless the surgeons decide to biopsy the adipose graft in our patient, it would be impossible to know whether the contamination was indeed from the transplantation of the adipose tissue from the abdomen, as *Enterobacter* is normally found in the gastrointestinal region. However, as there is no alternative explanation, this seems the likely scenario.

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