Optic Neuritis: A Rare Complication of Primary Varicella Infection

Optic neuritis is an extremely rare complication of chickenpox that has been the subject of isolated case reports [1–4]. Selbst et al. reviewed the cases of eight patients aged 3–14 years, and Miller et al. described the first three adult cases [2, 3]. All 11 patients had visual symptoms that occurred during or after the onset of varicella rash and that ranged from 2 to 38 days [2–4]. Visual loss is nearly always bilateral in patients with optic neuritis and can be severe. We describe an unusual case of monocular optic neuritis due to varicella in an adult whose visual symptoms preceded the varicella rash.

A healthy 25-year-old female presented with a 3-week history of progressive blurring of vision of her left eye. She did not have associated eye pain, diplopia, or headache. Five days after the onset of visual blurring, she developed vesicles over her upper lip that spread to the rest of her body. She apparently had not been

His vancomycin-resistant enterococcal bacteremia had been preceded by treatment with iv vancomycin, imipenem, and ciprofloxacin for prior infections. He again had severe neutropenia and mucositis. He never developed signs or symptoms of sepsis. He received no specific therapy, and subsequent blood cultures were negative for enterococci. His neutropenia resolved after 37 days, and his leukemia went into remission.

In September 1995 the patient’s leukemia relapsed, and he again received chemotherapy. A rectal surveillance culture once again yielded the same E. faecium isolate. After two infections (Candida albicans fungemia 10 days before isolation of vancomycin-resistant enterococci [VRE] in blood cultures and Staphylococcus epidermidis bacteremia 20 days before isolation of VRE in blood cultures), blood cultures again yielded the identical E. faecium isolate. Even though his vascular catheter insertion sites were changed, E. faecium bacteremia persisted and he had severe neutropenia and mucositis. On the fifth day of bacteremia, he developed septic shock with hypotension and respiratory and renal failure. He remained bacteremic throughout the course of therapy with doxycycline and novobiocin (the organism was susceptible to these two antibiotics). He was still severely neutropenic at the time of his death 3 days later.

Although vancomycin-resistant enterococci has become a major nosocomial pathogen, little is known about its epidemiology. Leukemia patients offer a unique opportunity to study the epidemiology of VRE since they are admitted to the hospital numerous times and (at our institution) since rectal surveillance cultures are performed weekly while the patients are hospitalized. In our patient’s case, he was colonized with the same vancomycin-resistant enterococcal clone over a 2-year period despite extended periods (>3 months) outside of the hospital and remission of his leukemia. Of 22 follow-up rectal surveillance cultures, all but one yielded VRE.

At our institution, we maximize the chance of isolating VRE by using selective media (colistin nalidixic acid agar supplemented with defibrinated 5% sheep blood, vancomycin [10 /..tg/mL], and amphotericin B [1 /..g/mL]) for surveillance cultures, which may explain the high proportion of positive cultures. However, the potential for long-term carriage suggests that all cancer patients with a history of colonization with VRE should be isolated during every hospitalization.

Our patient had multiple risk factors for infection due to VRE. He consistently became bacteremic with VRE after induction chemotherapy that caused neutropenia and mucositis, which in turn necessitated the use of therapeutic antimicrobial agents. He had received multiple antibiotics (including oral vancomycin) that select for VRE in stool [3]. The VRE organisms isolated from blood cultures were identical to those colonizing his gastrointestinal tract, a finding that strongly implicates the gastrointestinal tract as the source of bacteremia. Oral vancomycin is no longer used for bowel prophylaxis at our institution.

A case-control study comparing cancer patients infected with VRE to those colonized with VRE found that the total number of days of antibiotics was the only independent risk factor for VRE infection in a multivariate analysis [4]. Another case-control study that compared cancer patients infected with VRE to ward controls found that VRE colonization and antibiotics that cover anaerobes were risk factors for infection due to VRE [2]. In a similar study, increasing APACHE scores, mucositis, and receipt of antibiotics for >80% of hospital days [1] were independent risk factors for infection due to VRE in cancer patients.

Mortality in reported outbreaks of VRE infection in cancer patients has been high (57%–73%) [2, 4]. In our experience, the prognosis of bacteremia due to VRE is strongly associated with the prognosis of the underlying disease; infection due to VRE resolved with neutrophil recovery and disease remission. During our patient’s fourth course of induction chemotherapy, when his chance of remission was low, VRE caused persistent bacteremia and, finally, septic shock. Our patient’s case demonstrates that bacteremia due to VRE may not be lethal if the underlying disease is controlled; however, the risk of infection remains if the underlying disease recurs.

References

exposed to varicella, but her son became secondarily infected with varicella about 2 weeks after her illness started. She had the characteristic varicella rash, most of which had already scabbed.

Her visual acuity was 20/20 in her right eye and 20/40 in her left. There was a left afferent pupillary defect. Funduscoppy showed left optic disk edema with no visible retinal or choroidal lesions. Color vision in the left eye was impaired, and there was a demonstrable visual field defect. She did not have any other neurological deficits. A visual evoked response test revealed a conduction defect of the left visual pathway.

The diagnosis of acute varicella infection with left optic neuritis was made. The patient was given iv methylprednisolone (0.5 g q.d. for 5 days) followed by oral prednisolone (45 mg q.d. for 11 days) in tapered doses. At the same time, she was given iv acyclovir (500 mg q8h for 3 days) followed by oral acyclovir (800 mg five times per day for 13 days). Four days after the initial treatment, her visual acuity returned to normal although her color vision was still impaired. Her vision was completely restored after 1 month. She remains well and has not had any further attacks of optic neuritis or other neurological symptoms two years after her admission.

Our case is unique in that the visual complications preceded the onset of the varicella rash. To our knowledge, only one other case with a similar presentation has been reported [1]. The pathogenesis of viral-associated optic neuritis has not been well established. The delayed onset of optic neuritis and the frequent bilateral involvement and near-complete recovery in many cases suggest an immune-mediated process with consequent demyelination [2, 3]. Our observation that the ocular symptoms preceded the systemic infection suggests that the optic neuritis may instead be due to direct viral invasion.

Herpes zoster–associated optic neuritis and necrotizing retinitis are believed to result from viral invasion secondary to varicella reactivation or to dissemination from the dermatomal lesions. These infections are seen in both immunocompetent and HIV-infected patients and are associated with extensive visual loss [5, 6]. Unlike varicella-associated optic neuritis, the outcome for patients with herpes zoster–associated optic neuritis and necrotizing retinitis is extremely poor despite acyclovir treatment. In most cases of varicella-associated optic neuritis, vision is completely restored, although there may be residual optic disk pallor [2]. The use of steroids to treat optic neuritis is controversial. Other patients [1, 2] have recovered without receiving steroid therapy, although it is believed to hasten recovery [3]. Two patients had severe residual visual loss 6 months after the onset of optic neuritis despite receiving steroid therapy [3, 4]. We administered steroids with acyclovir to our patient since the visual symptoms preceded the rash. The patient’s symptoms started to decrease only a few days after the initiation of therapy. The present case illustrates that it is still not understood whether the pathogenesis of varicella-associated complications is due to direct viral invasion, to an immune-mediated process, or to both.

**References**


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**Streptococcal Venereal Edema of the Penis**

The cause of penile edema is often unclear. This condition has been variously described as “idiopathic” in children [1] and as occurring secondary to infection with *Chlamydia trachomatis* or group G streptococci [2]; one case due to group B streptococci in a neonate has been reported [3]. We describe two cases of penile edema and cellulitis in monogamous males; these cases occurred after the patients had engaged in vaginal intercourse, and one was due to *Streptococcus pyogenes*, while the other was due to *Streptococcus agalactiae*. Neither of the couples had engaged in fellatio.

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**Case 1.** A 51-year-old male presented with fever, swelling of the penis, and erythema of the pubis on 2 October 1992. Physical examination revealed bilateral inguinal tenderness and swelling. The patient was treated with amoxicillin for 5 days, and his condition improved. Six months later he presented with the same clinical complex, and specimens for culture were obtained from his urethra as well as from his partner’s vagina (she was asymptomatic). A group B streptococcus (*S. agalactiae*) was isolated from both specimens. The patient and his partner were both treated with amoxicillin, and the patient’s condition improved. He reported no further episodes at a 3-year follow-up assessment.

**Case 2.** A 38-year-old male presented with fever (temperature, to 39°C) and swelling of the penis on 14 May 1996. Examination revealed penile edema and erythema with bilateral enlarged inguinal lymph nodes and overlying erythema of the skin (figure 1). Findings on examination of his wife, who was asymptomatic, were normal except for the presence of minimal vaginal discharge; an IUD was also present. Cultures of specimens from the patient’s urethra were negative. Cultures of vaginal specimens from his wife yielded a group A streptococcus (*S. pyogenes*). Both the patient...