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 relative afferent pupillary defect. Funduscopy showed left optic disc 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 dermalomalous infections. 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.

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**References**


**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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the condition was self-limited. Cultures for streptococci were not performed for the partners, who were not described as having systemic complaints, in this study.

Carlino and Calzavara [2] described a case of "chronic urethritis" with recurrent attacks of cellulitis. Chlamydiae were isolated at the onset, and group G streptococci were isolated each time the cellulitis recurred. No cultures were performed for the female partner.

We believe that the two cases of penile cellulitis described herein were due to each female partner's vaginal streptococcus. In both cases, the women were asymptomatic. Both men were symptomatic, with penile swelling and erythema and marked tender inguinal adenitis. Both were febrile and responded to treatment with amoxicillin.

The patient in case 1 had a recurrence of his infection, and he and his partner were retreated. Both patients claimed to be monogamous.

How these men became infected is unclear. The patient in case 1 described engaging in vigorous sexual activity before the two episodes. The patient in case 2 noted a few small cuts on the penile skin before he had intercourse and developed the infection.

We believe that this report represents the first description of streptococcal penile cellulitis that occurred following vaginal intercourse. The recurrent nature of the first case underlines the need to counsel such patients when the diagnosis is first made. Perhaps some cases previously described as idiopathic penile edema represent a mild form of streptococcal cellulitis. In situations where penile edema associated with inguinal adenitis and a systemic illness are encountered, cultures of the female partner's vaginal secretions may help elucidate the etiology of the condition, and appropriate therapy may be instituted for both individuals.

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Systemic Paradoxical Response to Antituberculous Drugs: Resolution with Corticosteroid Therapy

The term *paradoxical expansion of tuberculosis* (TB) refers to an infrequent syndrome characterized by the development of previously nonexistent TB lesions or worsening of preexistent lesions during antituberculous treatment [1]. To our knowledge, systemic paradoxical responses to therapy have not been previously reported. We describe an immunocompetent patient who developed a severe, life-threatening systemic syndrome while receiving treatment for miliary tuberculosis.

A 28-year-old man who had previously been in good health was admitted to our hospital because of a 2-week history of fever (temperature, 38.5°-39°C), cough, and constitutional symptoms. He had also had lumbar pain for the last 2 months. Findings on physical examination were unremarkable except for the presence of fever (temperature, 39°C).

The initial results of laboratory studies are presented in table 1. Radiographs of the chest showed a miliary pattern.

Reference


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