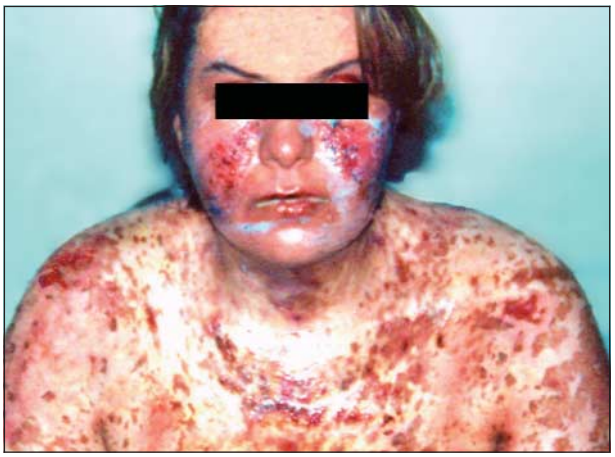

Figure 1. Skin detachment and mucosal erosions.

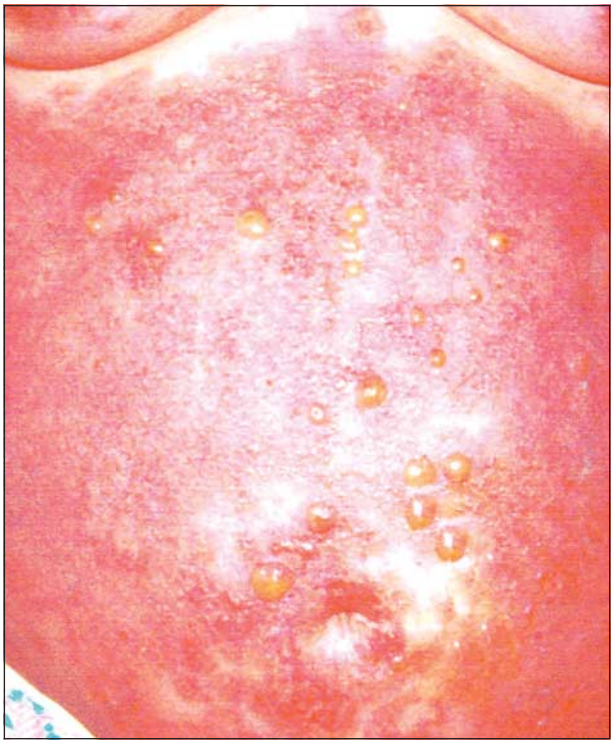

Figure 2. Bullous eruption.

and treatment of infection with antibiotic therapy as a prophylaxis, and systemic corticosteroid administration. The signs and symptoms progressively resolved in 23 days, and she recovered completely and was discharged.

Comment. The association between TEN and the use of lamotrigine in patients has been previously reported with concomitant use of valproic acid and lamotrigine.[3,4] It has been hypothesized that valproic acid may interfere with the metabolism of lamotrigine, leading to increased lamotrigine blood levels.[4] Our patient acquired TEN as a result of lamotrigine being concomitantly used with carbamazepine. Carbamazepine is one of the drugs most frequently implicated in TEN. It decreases lamotrigine blood levels by increasing its clearance.[5] Also, the patient reported prior use of carbamazepine for the previous 3 years with no skin rash, so lamotrigine appears to be the causative agent. However, because of the temporal relationship of the onset of the patient's rash and use of 2 antiepileptic drugs that are known to cause severe rashes, it is not certain which drug was the definite culprit. Further epidemiologic studies are needed to identify the incidence of lamotrigine-induced severe cutaneous reactions and the relative risk in concomitant use with other antiepileptic drugs.

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Plasma Cell Vulvitis: A Rare Cause of Intractable Vulvar Pruritis

In 1955, Zoon[1] described a benign condition of the vulva that was characterized by erythematous plaques composed of predominantly plasma cells. Since his initial case report, there have been 32 additional cases reported, using 3 synonyms: plasma cell vulvitis (PCV), Zoon vulvitis, and vulvitis circumscripta plasmacellularis.[2,3] The most common symptoms associated with PCV are pruritis, pain, burning, and dyspareunia.[2] While some case reports describe conservative medical treatments that have been effective, we report a case of PCV that was recalcitrant to all treatments except surgical resection.

Report of a Case. A 28-year-old woman presented to a referral center for vulvovaginal diseases with a 3-year history of intractable pruritis that was localized to the vulvar vestibule. She also complained of intermittent introital dyspareunia. She had been treated with fluconazole, terconazole, clobetasol, estradiol, and intralesional triamcinolone, without symptom relief. Colposcopic examination of the vulva revealed a glistening plaque in her vulvar vestibule (**Figure 1**). A biopsy specimen of the lesion demonstrated spongiosis and a lichenoid infiltrate containing a predominance of plasma cells

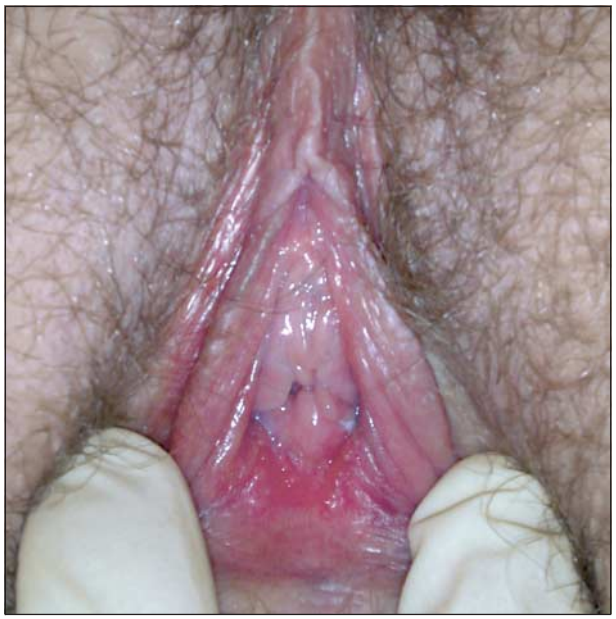

Figure 1. Shiny, erythematous plaque in the vulvar vestibule.

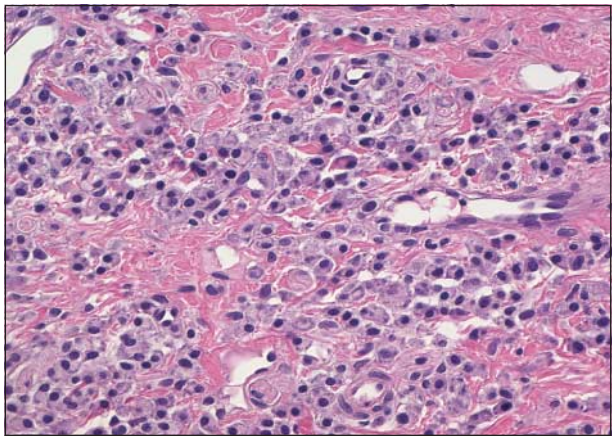

Figure 2. Infiltrate consisting of plasma cells and lymphocytes (hematoxylin-eosin, original magnification ×100).

(**Figure 2**). Her symptoms and the histologic features were consistent with the diagnosis of PCV. She underwent a series of 6 intralesional injections of recombinant interferon alfa-2b (Intron A), without improvement in her symptoms. She then underwent surgical resection of the lesion and has remained symptom-free since then.

Comment. Plasma cell vulvitis is a rare cause of intractable vulvar pruritis. The physical findings consist of atrophic, glistening, erythematous plaques or patches. Infrequently, there can be erosions or areas of friability. Histologic examination reveals a lichenoid infiltrate that is composed of plasma cells and lymphocytes. Additional findings include "diamond-shaped" keratinocytes, vascular dilatation, and intracellular edema. Hemosiderin deposits give the lesions their characteristic red or brownish color.[2,4]

While the etiology of PCV is unknown, authors have suggested that trauma, chronic irritation, or an autoimmune response to an unidentified mucosal antigen may play a role in the pathogenesis of this disorder.[4] Response to therapy is inconsistent. Reported treatments include estrogens, topical and intralesional corticosteroids, antifungal agents, antibiotics, fulguration, caudal nerve blocks, laser ablation, cryotherapy, interferon alfa, etretinate, and surgical resection.[2,5] In the present case, several conservative methods of treatment failed before the patient underwent successful surgical resection.

The differential diagnosis of these erythematous lesions should include Paget disease, pemphigus vulgaris, lichen planus, fixed drug eruption, squamous carcinoma, and herpes simplex infection.[4] The diagnosis of PCV should be considered in any patient with an erythematous lesion and intractable vulvar pruritis that is nonresponsive to treatment with topical steroids.

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Vulvovaginitis and Perineal Cellulitis Due to Group A Streptococcus in an Adult Woman

We describe a woman of childbearing age with group A streptococcal vulvovaginitis and perineal cellulitis, a condition usually encountered in prepubertal girls.[1-3]

Report of a Case. A 41-year-old woman was admitted for a 2-week history of copious vaginal discharge with severe and progressive vaginal and perineal pruritus, edema, and erythema. Prior to admission, the patient had multiple visits to community gynecologists and received, without benefit, topical antifungal agents, metronidazole vaginal gel, oral antiviral agents, and antihistamines.

The patient had a ParaGard copper intrauterine device (Ortho Pharmaceutical Corporation, Raritan, NJ) in place since 1999. The remainder of her history was unremarkable. However, her daughter had been diagnosed with group A streptococcal pharyngitis 1 week before onset of her symptoms.

On physical examination, the patient was afebrile, and extremely uncomfortable due to severe pruritus in the