not on mucosal surface. It is a locally invasive carcinoma with no lymphatic spread, but it rarely metastasizes. However, pudendal BCCs behave aggressively and seem to metastasize widely. Pudendal BCC has a predilection for the scrotum in males and labia majora in females.[1]

Although ultraviolet radiation may be an important etiologic factor for BCCs on sun-exposed areas, the cause of their occurrence on areas not exposed to sun has not been identified. Chronic skin irritation, previous trauma, exposure to ionizing radiation, coal tar and other carcinogens or immunosuppressive drugs may be responsible for the development of tumors at uncommon sites.[1,2] In our case, advancing age may be responsible for the rare occurrence of BCC over the scrotum.

In view of the aggressive nature of scrotal basal cell carcinoma, all the patients should be kept under surveillance for metastasis for 2 to 5 years after excision of the tumor.

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Oral-esophageal lichen planus associated with oral squamous cell carcinoma

Sir,

Oral lichen planus (OLP) is a chronic inflammatory mucocutaneous condition which affects approximately 0.2% to 3.8% of the general population, being more frequent in females.[1] Among OLP cases, only 1% may involve the esophagus.[2] The rate of malignant transformation in OLP is about 0.4% to 1.74%.[3]

In May 2004, a 64-year-old Italian woman was referred to our hospital with a history of oral white lesions. Clinical examination showed OLP in her oral cavity with reticular and plaque variants. Bilateral buccal mucosae were affected, particularly on the right side, with evidence of atrophic
erosive foci. There were no cutaneous or genital lesions. She had had a sub-total thyroidectomy and suffered from mild hypertension; however, she was not under any medication.

The patient was negative for antibodies to HbsAg and hepatitis C virus. Because of oral lesion extensions accompanied by dysphagia, we performed an endoscopy of the pharynx-esophageal tract. The finding of ulcerative esophagitis necessitated an endoscopic ultrasonography, but there was no evidence of malignancy [Figure 1]. The results of the oral biopsy confirmed the diagnosis of OLP associated with low-degree dysplasia. Therefore, the OLP lesion was subsequently partially removed.

We initiated treatment with a topical corticosteroid and the lesions were examined periodically every 3 months, monitoring the upper digestive tract. During a routine follow-up in February 2006, we performed an oral biopsy and took multiple samples using the toluidine vital staining method. The histopathological report showed a focal severe-degree dysplasia in the right retromolar trigonal region, which was promptly removed.

In May 2006 a new biopsy revealed an oral squamous cell carcinoma (OSCC) on the sublingual area [Figure 2]. The radical removal was carried out after a grade 1 neoplasia staging (according to TNM classification) [Figure 3].

We strongly support the multidisciplinary approach when OLP is associated with esophageal involvement. An esophageal stricture is found in half of these patients, and exceptionally, a squamous cell carcinoma can develop within esophageal lichen planus.[4] It is important to note that within 2 years of follow-up on diagnosis of OSCC, about 10% of the patients develop a new neoplasia in the gastric-respiratory tract.[5]

Our case emphasizes that where OLP affects wide areas, multiple biopsy samples and the toluidine vital staining method should be routinely used to assist in the choice of biopsy sites.

Such procedures allow us to select the lesion areas more likely to develop a malignant process. With an early diagnosis during the process of carcinogenesis, we can improve survival rates for patients with oral cancer.

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Ciprofloxacin-induced generalized bullous fixed drug eruption

Sir,
Ciprofloxacin, a widely used quinolone antibiotic, induces cutaneous adverse drug reactions in about 1% to 2% of treated patients.[1] Urticaria, angioedema, maculopapular exanthem, and photosensitivity are the most frequently documented cutaneous adverse reactions.[2] Only a few cases have been reported in which ciprofloxacin has been implicated in fixed drug eruption (FDE)[3,4] or more severe drug reactions such as Stevens-Johnson syndrome (SJS) [5] or toxic epidermal necrolysis (TEN).[6] We describe a case of generalized bullous FDE induced by ciprofloxacin.

A 57-year-old woman presented with a 3-day history of widespread tender, pigmented patches; and scattered blisters all over her body. The patient stated that these lesions had appeared within a few hours of taking a single dose of oral ciprofloxacin for a respiratory tract infection. She had a previous history of a similar but a more localized reaction after taking the same drug 6 months earlier. She noticed a few residual pigmented macules after that. Her medical history was significant for chronic renal failure, for which she had been on hemodialysis treatment for 5 years. She had been taking perindopril and indapamide for hypertension and congestive heart failure during the same period. The patient denied taking any other new drug in the preceding days.

Physical examination revealed extensive purplish-livid patches covering almost 60% of the total body surface. Multiple well-circumscribed patches were observed on the arms and legs [Figure 1]. Some of the patches were studded with flaccid vesicles and bullae over the buttocks. Eroded areas were also noted on the arms and legs. Pseudo-Nikolsky's sign was positive on some of the purplish-livid patches. The mucous membranes, the palms and soles, and the face were not involved. Her temperature was 37°C, and her other vital signs were within normal range.

The patient was hospitalized with the differential diagnosis of generalized bullous FDE, SJS, and TEN. Histopathological examination of a punch biopsy specimen taken from a flaccid bulla overlying the large purplish-livid patch on the left buttock showed necrosis of epidermal keratinocytes with subepidermal clefting. Perivascular mixed inflammatory infiltrate containing eosinophils and neutrophils, and prominent pigmentary incontinence were also observed within the dermis. Direct immunofluorescence revealed no IgG and C3 deposition at the basement membrane zone. Laboratory investigations showed the following values: C-reactive protein of 65 mg/L (normal range, <10 mg/L); white blood cell count of 20.5×10⁹/L (normal range, 4.5-11×10⁹/L) with 70% neutrophils (normal range, 40%-72%), hemoglobin 8.2 g/dL (normal range, 12-16 g/dL), and blood urea nitrogen 65 mg/dL (normal range, 6-21 mg/dL); and a creatinine level of 6.2 mg/dL (normal range, 0.5-1.3 mg/dL).

A chest x-ray revealed increased opacities in the middle and lower areas, indicating bilateral pneumonic infiltrates. The patient was diagnosed as having ciprofloxacin-induced generalized bullous FDE. We initiated oral prednisolone (1 mg/kg/d) therapy. She was also given oral clarithromycin (1 g/d) treatment for suspicion of atypical pneumonia. The vesicles, bullae, and eroded areas disappeared after 1 week.

References