Dear Editor,

Intralesional steroid therapy is an effective treatment modality for various dermatological and orthopaedic disorders, but it is associated with a few peculiar side effects. Linear hypopigmentation and depigmentation is a side effect, which is rare and merits discussion. We report three cases of linear depigmentation following intralesional triamcinolone injection for orthopaedics and dermatological indications. Two patients received intralesional triamcinolone acetonide injections for ganglion [Figures 1 and 2] and one patient received it for hypertrophic lichen planus [Figure 3]. The details of their treatment, disease onset and final outcome till last follow-up visit have been summarized in Table 1. First two patients refused to get biopsy. In the last patient, histopathology examination (hematoxylin and eosin stain, 200×) from depigmented macule showed normal number of melanocytes with decreased melanin content [Figure 4]. All patients were counseled about the condition, avoidance of intralesional steroid treatment and possibility of partial or complete repigmentation. Injectable steroids offer many advantages over systemic or topical preparations; causing less irritation and minimal side effects. Moreover, potent insoluble steroid compounds are longer acting and more effective due to significantly reduced dissolution.
Correspondence

Table 1: Enumerating cases of linear leucoderma following intralesional steroid treatment

| Case no.                  | Indication                             | Intralesional triamcinolone acetonide (mg/mL) | No. of injections received and volume of steroid injected | Onset of lesions after intralesional injection/s (months) | Final outcome                                      |
|---------------------------|----------------------------------------|----------------------------------------------|----------------------------------------------------------|--------------------------------------------------------|---------------------------------------------------|
| Case 1 (40 years/female)  | Ganglion over left wrist               | Injection triamcinolone acetonide (40 mg/mL) | 2 times, 3 months apart, 0.4 and 0.3 mL, respectively    | 5                                                      | Partial repigmentation at 2 months follow up, subsequently lost to follow up |
| Case 2 (23 years/female)  | Ganglion over left wrist               | Injection triamcinolone 1 injection, 0.3 mL acetonide (40 mg/mL) | 1                                                      | 1                                                      | No repigmentation till 6 months.                  |
| Case 3 (30 years/female)  | Hypertrophic lichen planus             | Injection triamcinolone 3 times, 0.3, 0.2, 0.2 mL, acetonide (10 mg/mL) respectively | 6                                                      | 6                                                      | No repigmentation till 2 months, subsequently lost to follow up |

at the site of injection and are therefore, preferred for chronic conditions. Triamcinolone acetonide is a macrocrystalline molecule and is a potent steroid. It has been used effectively intralesionally for the treatment of hypertrophic scars, keloids, alopecia areata, hypertrophic lichen planus and various non-dermatological conditions such as ganglion, tenosynovitis and intra-articularly for arthritis. The concentration of triamcinolone acetonide varies from 2.5 to 40 mg depending upon the indication.

Intralesional injections are an effective treatment modality; however, perilesional hypopigmentation, depigmentation, cutaneous atrophy, alopecia, infection, ulceration and localized dystrophic calcification are commonly observed local side effects. Linear hypopigmentation or depigmentation following intralesional steroid is a rarely seen side effect with few reports in the literature and even fewer reports presenting in a linear distribution. It may develop after a variable latency period ranging from a few weeks to months and may develop following a single, a few or even after several intralesional corticosteroid injections.

The exact pathogenesis of corticosteroid-induced hypopigmentation is unknown. The hypothesized etiology relates to corticosteroids-induced inhibition of prostaglandin or cytokine production in various epidermal cells, thereby altering the melanocyte function by suppressing the secretory metabolic products from melanocytes without causing their destruction. Friedman et al. and Venkatesan et al. demonstrated reduced melanin pigment and activity of melanocytes in the presence of normal melanocyte number, by histopathology added with histochemical staining. In addition, the exact pathogenesis of linear hypopigmentation or atrophy is also unknown. The most widely accepted mechanism is the lymphatic spread of the corticosteroid suspension along the lymphatics. Lymphatic vessels run in a unidirectional manner and are responsible for removal of macromolecules and proteins. Kikuchi et al. proved the relationship between these linear lesions with the lymphatic vessels after injecting Evans Blue Dye or Alphazurine 2 G (Patent Blue) into atrophic lesions.

However, there are a few factors which predispose to this corticosteroid-induced hypopigmentation or depigmentation. The concentration of the injected corticosteroid seems to be important. When the concentration increases, more of the steroid remains in the unbound state because the protein-binding capacity exceeds. This freely available steroid then enters cells and mediates effects. Moreover, depigmentation seems to be more likely with triamcinolone due to larger size, higher tendency to aggregate and higher density.

There is no specific treatment for this condition and repigmentation, which may be partial in some cases might take several months. Further injections should be withheld and the patients should be kept under follow up.

To conclude, this case report highlights the potential side effect of depigmentation or hypopigmentation following injectable steroids and the related cosmetic distress to the patient. Therefore, it is emphasized that care should be taken to minimize this side effect. Appropriate concentration of steroid should be injected and excess drug and deeper injections into the underlying dermis and subcutaneous tissue should be avoided especially in hyperpigmented individuals.

Amit Kumar Dhawan, Kavita Bisherwal, Chander Grover, Nadeem Tanveer
Departments of Dermatology and STD and Pathology, University College of Medical Sciences (UCMS), Guru Teg Bahadur (GTB) Hospital, Delhi, India
E-mail: amitkumardhawan@gmail.com

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Dear Editor,

Acrochordon (fibroepithelial polyp or skin tag) is a common benign neoplasm seen predominantly in obese individuals with an average of 46% incidence in the general population. Measuring about 1 to 5 mm in size, they are usually located in the intertriginous areas. We report an unusual presentation of acrochordon.

A 50-year-old postmenopausal woman presented with a mass hanging from labia majora since 3 years. While cleaning her private parts she first noticed a lemon sized mass which gradually increased in size. Patient experienced discomfort while walking due to the weight and rubbing of the mass between her thighs and became apprehensive of its growing size. There was no pain, pruritus, fever, redness, ulceration, bleeding, discharge, sudden increase in size, aggravating/relieving factors or diurnal change in size. Patient was overweight (body mass index 27.3 kg/m²). Dermatological examination revealed a single, skin-coloured, pedunculated, pear-shaped, non-tender, soft fleshy mass with wrinkled surface measuring 16 × 9 cm and arising from a thin stalk from posterior part of left labia majora [Figure 1] and distorting its shape. There was no redness, discharge or ulceration. The mass was nonpulsatile, nonreducible, with no impulse on coughing, no palpable thrill or bruit with no regional lymphadenopathy. Systemic and genitourinary examination was normal. Blood sugar and lipid profile were normal. The mass was diagnosed as acrochordon due to its pedunculated attachment, soft consistency and free mobility.

The lesion was elliptically excised. Histopathological examination revealed mature stratified squamous epithelium, increased fibrocollagenous tissue in the stroma, thickened blood vessels, stellate fibroblast and sparse perivascular chronic mononuclear inflammatory infiltrate with no evidence of malignancy [Figure 1].

In women, genital acrochordon is more common in the vagina than vulva and cervix with peak incidence at 20-40 years of age. It is rare in postmenopausal women. Large lesions may arise due to proliferation of mesenchymal cells within the hormonally sensitive subepithelial stromal layer of the lower genital tract. Acrochordons are associated with type 2 diabetes mellitus, insulin resistance, obesity, dyslipidemias, pregnancy, genetic predisposition, human papilloma.