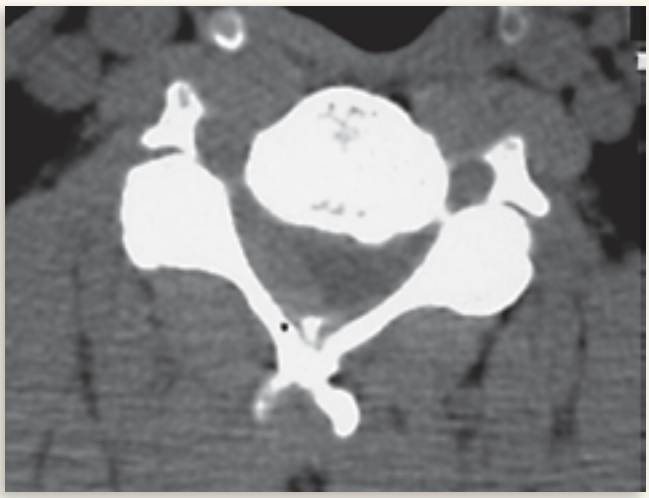
Emergency Case

A major pain in the neck

Anthony M. Herd, MD, CFPC, CFPC(EM)



A 54-year-old man presented to our emergency department with sudden spontaneous onset of pain in his neck. The pain was sharp and radiated into both arms. He had never had this pain before. Results of examination were unremarkable; bowel and bladder function were normal. He had undergone an aortic valve replacement 2 years earlier and was taking prophylactic warfarin.

He was in some distress from pain but was afebrile, and his vital signs were normal. His blood pressure was similar in both arms, and he had no cardiac murmurs. There was generalized tenderness of the paraspinal muscles of his neck and upper back, and he had a slight weakness in his right arm when he extended his elbow. Over the course of a few hours, this progressed to weakness in both arms. Laboratory studies showed his international normalized ratio to be 2.9, but all other results were normal.

What unusual diagnosis must be considered in this case?

1. Transverse myelitis
2. Spinal epidural hematoma
3. Pathologic cervical fracture
4. Spinal epidural abscess

Answer on page 506

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Dermacase

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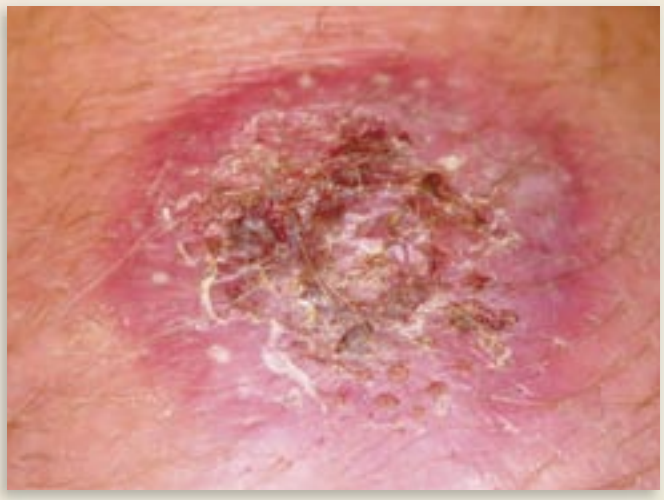

CAN YOU IDENTIFY THIS CONDITION?

A 57-year-old farmer presents with edema, erythema, and a cutaneous lesion on the back of his right hand. The lesion first appeared 10 days before as a small, erythematous, pruritic macule and disappeared shortly thereafter. It reappeared 8 days later and became bigger. The farmer was otherwise healthy and was taking no medication. He had no known drug allergies. On examination, his right hand appeared erythematous, swollen, and warm, and it had a 3-cm erythematous scaly plaque on the back. Results of his examination were otherwise normal.

The most likely diagnosis is:

1. Spider-bite reaction
2. Psoriasis with inflammation secondary to bacterial infection
3. Dermatophyte infection (tinea)
4. Rickettsial infection (tick-bite eschar)
5. Granuloma annulare

Answer on page 500

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Answer to Dermacase

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3. Dermatophyte infection (tinea)

The dermatophytes are a group of fungi that invade the superficial layer of the epidermis and survive on the keratin of skin, hair, and nails. A dermatophyte infection results in increased skin proliferation, scaling, and epidermal thickening.[1] Cutaneous dermatophyte infections are common in the general population; up to 20% of people are infected at any time.[2] Most of these infections are not life threatening, but they can cause morbidity in immunocompromised and diabetic patients. It is important, therefore, for primary care physicians to recognize and treat these infections appropriately.

Dermatophyte infections involving the skin are called epidermal dermatophytoses. These infections are the most common dermatophytic infections[2] and can involve the feet, hands, groin, face, trunk, and extremities (**Table 1**). Infection can last from months to years. Patients might have no symptoms or seek help for pruritus.[1] Tinea pedis affects up to 70% of adults worldwide.[2]

Dermatophyte infections involving the nails are called onychomycoses or tinea unguium. Adults are most commonly affected; children are rarely affected. About 40% of those older than 70 years are affected.[2] Infection does not usually remit spontaneously.

Risk factors for developing onychomycosis include atopy, diabetes mellitus, immunosuppression, occlusive footwear, nail trauma, and cutaneous diseases affecting the nails, such as psoriasis.[2] Differential diagnosis can include psoriasis, lichen planus, traumatic injury, and congenital nail dystrophies.[2] Clinical appearance can vary with site, fungal species involved, and host response. Appearance alters with inappropriate use of steroid creams. Zoophilic infections (fungi from animal sources) often have a more inflammatory presentation.[2]

Trichomycoses are dermatophyte infections involving the hair. Tinea capitis is most common in preschool-aged black children.[2] *Trichophyton tonsurans* is a causative agent in most cases; it does not fluoresce on Wood's lamp examination.[2] Lesions usually last weeks to months. Tinea barbae is a dermatophytic trichomycosis involving the beard and moustache area. It resembles tinea capitis and invades the hair shaft. It occurs only in adult men and is more common among farmers.[1]

Table 1. Clinical classification of cutaneous dermatophyte infections according to body area involved

BODY AREA INVOLVED	CLASSIFICATION	COMMON PRESENTATION
Scalp hair	Tinea capitis	Alopecia, broken hairs at the scalp, painful inflammation of the scalp with boggy tender nodules that drain pus
Facial hair (beard) and neck	Tinea barbae	Erythema, scaling, pustules, broken hairs
Face	Tinea faciale	Annular scaly plaques with raised edges, pustules, and vesicles
Trunk and extremities	Tinea corporis	Annular scaly plaques with raised edges, pustules, and vesicles
Palms	Tinea manus	Scaling, erythema, usually one hand only, commonly in association with tinea pedis
Soles and interdigital spaces	Tinea pedis	**Interdigital:** dry scaling or maceration and fissuring of toe web spaces **Moccasin:** well demarcated erythema with fine white scaling and hyperkeratosis involving soles of one or both feet **Bullous:** vesicles or bullae filled with clear fluid on the sole, instep, or web spaces; after rupturing, erosions with ragged ringlike border
Groin	Tinea cruris	Erythematous plaques with central clearing and raised borders
Nails	Tinea unguium	Onycholysis, thickened, discoloured, dystrophic nails

Adapted from Martin et al.[1]

Diagnosis

Diagnosis of dermatophytosis relies on history and characteristic cutaneous findings, but can be confirmed by laboratory investigations that include potassium hydroxide (KOH) microscopy, fungal culture, and skin or nail biopsy.[2] Wood's lamp examination can be useful in confirming a hair infection with zoophilic dermatophytes (eg, *Microsporum canis* and *Microsporum audouinii*) that produce blue-green fluorescence.[2] In general,

this investigation is of limited diagnostic value because most dermatophytes in North America do not fluoresce.

Treatment

Dermatophyte infections can be treated with topical or systemic antifungal agents (**Table 2**). Topical therapy is seldom effective for treatment of onychomycosis and tinea capitis,[3] but can be used for treatment of tinea corporis, faciale, cruris, and pedis. Aluminum chloride hexahydrate (20% to 25%) is a useful adjunct to topical antifungal therapy in chronic macerated interdigital tinea pedis.[1] Keratolytic agents (eg, salicylic acid and lactic acid) can be useful in reducing hyperkeratosis in the moccasin type of tinea pedis. In this case, the patient was treated with oral fluconazole with no adverse effects, and had complete resolution of the dermatophytosis on his hand (tinea manus) within 2 months.

Diagnosis of dermatophytosis should be confirmed before initiating systemic therapy. Systemic antifungal medications can interact with other therapeutic agents,[3-5] so consultation with a pharmacist or review of prescribing information is recommended if patients are taking more than one medication. Monitoring complete blood count with differential, creatinine, and liver function tests is advised. Patients should be told to discontinue their medication at the first sign of acute hepatitis symptoms, such as nausea, malaise, or fatigue.[4] Consultation with a dermatologist might be beneficial for chronic disease and for consideration of systemic therapy.

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Table 2. Topical and systemic antifungal treatment in management of dermatophytosis

TYPE OF INFECTION	TOPICAL THERAPY	SYSTEMIC THERAPY
Tinea capitis (usually pediatric) and barbae (use adult dose)	Ketoconazole cream or shampoo (adjunct only, reduces fungal shedding). Apply daily for 5 min	Terbinafine* 3-6 mg/kg/d for 4-8 wk; itraconazole† tablets 5 mg/kg/d for 4-6 wk; fluconazole‡ 3-6 mg/kg/d for 6 wk (oral solution); griseofulvin§ 20-25 mg/kg/d for 8 wk and continue for 2 wk beyond cure (approved for use in children)
Tinea corporis, faciale, cruris, pedis	Clotrimazole, ketoconazole, miconazole, ciclopirox,¶ naftifine¶ (cream, ointment, or spray). Apply to affected area bid for 2-4 wk. Continue for 1-2 wk after symptoms resolve	Terbinafine* 250 mg qd for 2–6 wk; itraconazole† 200-400 mg qd for 1 wk; fluconazole‡ 150-300 mg and one dose repeat for 4-6 wk; griseofulvin§ 500 mg qd for 4-8 wk
Tinea unguium	Ciclopirox nail lacquer qd; remove each wk with ethyl alcohol. Cure rates range from 5% to 50% at 48 wk	Terbinafine* 250 mg orally for 6 (fingernails) to 12 (toenails) wk; itraconazole† 200 mg qd for 2 (fingernails) to 3 (toenails) mo; fluconazole‡ 150-300 mg/wk for 3-6 (fingernails) or 6-12 (toenails) mo; griseofulvin§ 500 mg qd for > 6 (fingernails) to > 12 (toenails) mo

bid—twice daily, qd—each day.

**Patients taking terbinafine should be followed up for hepatotoxicity; avoid in active or chronic liver disease.*

†Itraconazole use has been associated with cardiac complications and neuropathy. Hypoglycemia might occur with sulfonylureas; rhabdomyolysis might occur with 3 hydroxy-3-methylglutaryl coenzyme A reductase inhibitors; absorption is impaired with administration of antacids. Use with caution in hepatic insufficiency.

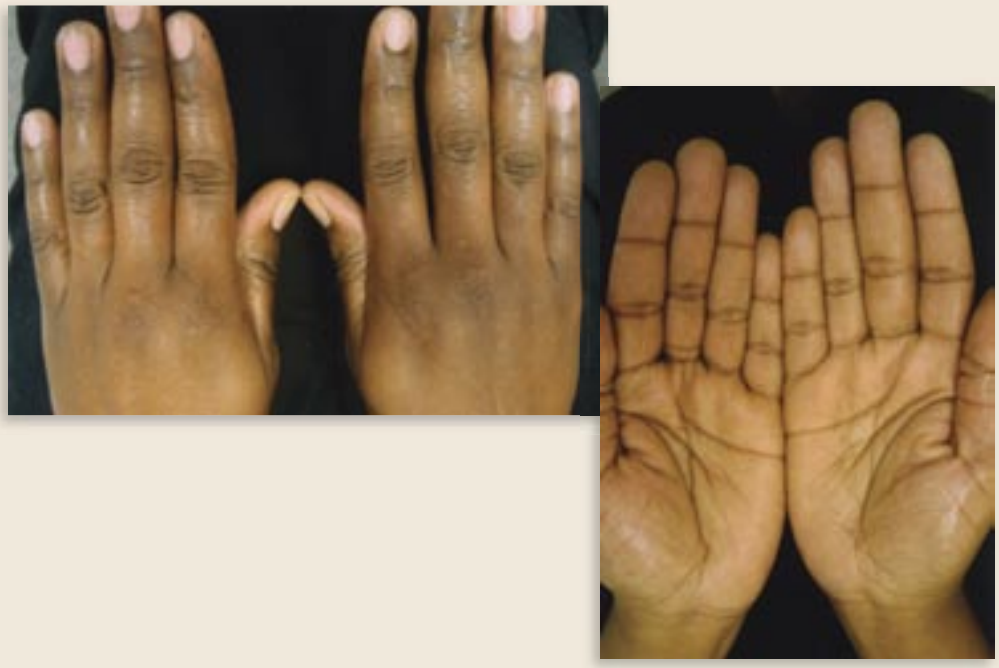
‡Fluconazole might cause hepatic complications, especially with underlying AIDS and malignancy. Hydrochlorothiazide diuretics might increase fluconazole plasma levels.

§Griseofulvin could decrease warfarin activity, serum salicylate concentrations, and the effectiveness of oral contraceptives and cyclosporine. If therapy is prolonged, monitor complete blood count and renal and hepatic function. Photosensitivity is a possibility.

¶Apply to moist web spaces.

Dermacase

Irina Turchin, MD **Benjamin Barankin,** MD



CAN YOU IDENTIFY THIS CONDITION?

A 30-year-old black woman presents with profuse sweating on her palms, soles, and axillae. She developed this condition about 15 years ago and now is concerned about having difficulty grasping objects and being embarrassed when shaking hands. She is otherwise healthy and is taking no medication.

The most likely diagnosis is:

1. Hyperhidrosis secondary to thyrotoxicosis
2. Dyshidrotic eczema
3. Essential or primary hyperhidrosis
4. Riley-Day syndrome (familial dysautonomia)
5. Hidradenitis suppurativa

Answer on page 505

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3. Essential or primary hyperhidrosis

Hyperhidrosis is excessive sweating beyond what is necessary for normal thermoregulation. It affects up to 1% of the population[1] and has been associated with social embarrassment, withdrawal, depression, and disruption of work.[2]

Several medical conditions are associated with hyperhidrosis. Primary or essential hyperhidrosis is idiopathic. It often begins in childhood or adolescence and persists throughout adult life.[3]

Secondary hyperhidrosis is a consequence of some other medical condition, such as chronic infections (eg, tuberculosis), endocrinopathies (eg, thyrotoxicosis or hypoglycemia), neoplasms (eg, carcinoid tumours, Hodgkin disease, or pheochromocytoma), medications (eg, antiemetics or antidepressants), menopause, and neurologic disorders (eg, syringomyelia, focal brain lesions, or autonomic dysregulation).[3]

Hyperhidrosis occurs in three different forms: localized, generalized (>100 cm^2), and emotionally induced. Emotionally induced hyperhidrosis affects the palms, the soles, and occasionally the axillae and can be associated with tachycardia and vasomotor instability.

Diagnosis

Hyperhidrosis can often be diagnosed by history and physical examination.[1] Diagnosis can be confirmed by an iodine-starch reaction that involves spraying iodinated starch powder onto the affected area, which will turn black in the presence of sweat.[4] This test helps to identify areas with excessive sweat production and indicate certain treatments. Appropriate investigations should be pursued if secondary hyperhidrosis is suspected.[1]

Treatment

Several treatments can be used to manage hyperhidrosis. Topical agents can be effective for localized hyperhidrosis. Over-the-counter antiperspirants containing aluminum chloride could suffice for most uncomplicated cases of axillary hyperhidrosis.[5] More severe cases of localized axillary or palmoplantar hyperhidrosis can be appropriately managed with aluminum chloride hexahydrate (20%) dissolved in anhydrous ethyl alcohol (Drysol). The preparation should be applied to dry skin every night and washed off in the morning.[5] Application can be gradually spaced out to once a week or as required once the medication is effective.[1]

Several systemic agents have been found effective for various forms of hyperhidrosis. Systemic anticholinergic agents (glycopyrrolate, oxybutynin) can be used for patients with localized and generalized hyperhidrosis. Dry mouth, constipation, urinary hesitancy, nausea, abdominal cramps, weakness, headache, dizziness, tachycardia, and blurred vision are well recognized side effects of anticholinergic therapy, but these side effects are usually well tolerated, especially by young, healthy patients. Clonidine has been shown to be effective for hyperhidrosis secondary to menopause and tricyclic antidepressants. Fludrocortisone acetate, 0.3 mg daily, might control sweating in quadriplegics with orthostatic hypotension.[5] Consultation with dermatologists and other specialists is highly encouraged before initiating systemic therapy.

Psychotherapy is an unconventional therapy for managing emotionally induced hyperhidrosis. Biofeedback for relaxation, desensitization training, and hypnosis might be useful as adjunct therapies.[6] Iontophoresis, passing of an anodal current through intact skin, is an effective treatment for patients with palmoplantar hyperhidrosis. Devices can be ordered directly from companies that produce them; they are often found in dermatologists' offices. Tap water iontophoresis for 30 minutes daily is effective for most patients within a week.

Botulinus toxin is one of the newer therapies for managing localized hyperhidrosis of the palms, soles, and axillae, and gustatory sweating. The toxin must be injected many times into the affected area within approximately 1 week of onset of anhidrosis; the therapeutic effect lasts between 4 and 12 months depending on patients' response and dose administered. Major drawbacks to this method are the high cost and mode of administration. Some patients

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experience transient weakness of the small muscles of the hand that lasts 2 to 3 weeks.[7] Botulinus toxin therapy is becoming increasingly popular among patients with localized hyperhidrosis despite its high cost and side effects.

Surgical options in management of hyperhidrosis include excision of axillary tissue, axillary liposuction, and thoracic sympathectomy.[3] Excision of axillary tissue aims to eliminate most of the axillary sweat glands. Complications of this method include wound infection, slow healing, wound hematoma, wound dehiscence, hidradenitis, and necrosis at the edge of the skin.[3]

Axillary liposuction involves removal or destruction of the apocrine glands along with disruption of nerve supply to the sweat glands.[3] Axillary liposuction will likely be the surgical treatment of choice for axillary hyperhidrosis refractory to more conservative measures.

One of the final options in managing hyperhidrosis is thoracic sympathectomy, which is done under general anesthesia as day surgery. It involves disruption of the second, third, and fourth thoracic ganglia and is successful in 87% to 98% of patients with palmar-axillary hyperhidrosis. Patients with respiratory impairment and pleural adhesions are not suitable for this surgery. Complications of thoracic sympathectomy include compensatory sweating, Horner syndrome, pneumothorax, hemothorax, thoracic duct injury, and phrenic nerve injury.

Family physicians can have an important role in recognition and treatment of hyperhidrosis, thus relieving patients of the psychosocial burden of this condition.

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Answer to Emergency Case

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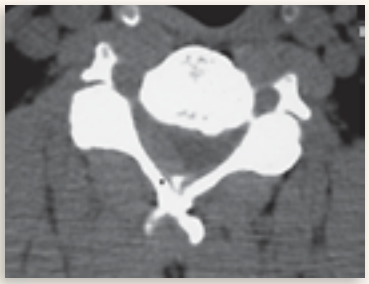

Figure 1. Computed tomography scan at the level of C4 vertebra showing an epidural hematoma pushing the spinal cord to the left and forward

2. Spinal epidural hematoma

This patient has a spontaneous (ie, nontraumatic) spinal epidural hematoma (SEH) from approximately the second to fifth cervical vertebrae (**Figure 1**). He immediately underwent laminectomy and decompression, made a full recovery, and was discharged 9 days later.

Traumatic SEH is well recognized and, when it causes spinal cord compression, is a true neurosurgical emergency. Although most commonly associated with major blunt trauma, it has also been described in association with spinal surgery,[1] epidural anesthesia,[2] and spinal manipulation.[3]

Spontaneous or nontraumatic SEH has also been reported[4] in association with fibrinolytic and anticoagulant agents[5] and with congenital and acquired coagulopathies.[6,7] The incidence of nontraumatic SEH is unknown. Although uncommon, this diagnosis needs to be remembered when any patient has spontaneous onset of back pain that is otherwise unexplained. Patients' use of anticoagulants should increase suspicion. Most importantly, any patient with unexplained back pain and neurologic findings urgently needs imaging studies and specialty consultation.

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Dermacase

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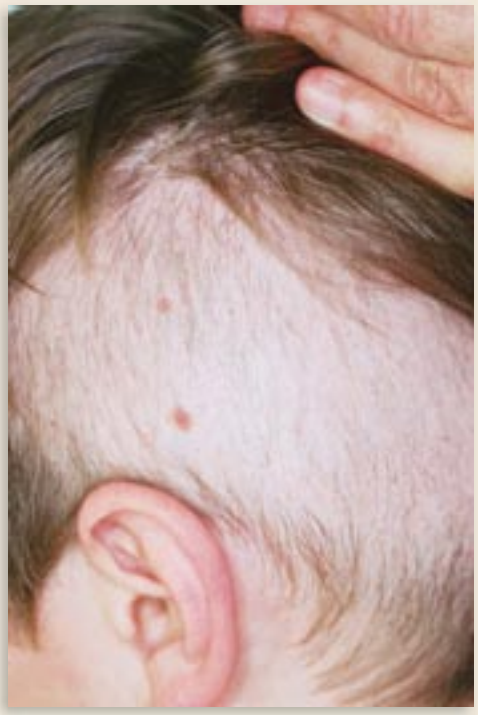

CAN YOU IDENTIFY THIS CONDITION?

A patient presents with hair loss on his scalp. The hair that remains is of unequal length and fractured.

The most likely diagnosis is:

1. Trichotillomania
2. Alopecia areata
3. Pseudopelade
4. Telogen effluvium

Answer on page 510

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1. Trichotillomania

Trichotillomania is a psychiatric disorder resulting in a characteristic hair loss pattern. Hair is lost through repetitive pulling, twisting, and rubbing. Hair loss might also be due to use of tweezers, scissors, or razors. Hair pulling with early onset (younger than age 6) is reported to end of its own accord more frequently than when it appears later in life.[1] Incidence of trichotillomania is higher among women than among men. The scalp is most frequently involved, followed by the eyebrows, eyelashes, facial hairs, and pubic and chest hairs. Patients feel a tension that is relieved by pulling out hair. Diagnosis can be complicated by patients' denial of hair pulling.

Certain clinical features differentiate trichotillomania from other hair disorders. The most distinguishable characteristic is an irregular pattern of fractured hairs of unequal length.[3] Alopecia areata appears as a nonscarring, well-defined oval area of scalp with no hair (or hair of equal length if growth has occurred) surrounded by "exclamation hairs." With trichotillomania, areas of hair loss are sharply or poorly defined, and there is no scarring. There is no inflammation at the site with either condition. In difficult cases, trichotillomania can be differentiated through histopathology.[4]

Treatment can be difficult. Patients with trichotillomania should be referred to psychiatrists. Appropriate therapies include psychotherapy, behavioural therapy, or appropriate medication.[5] The tricyclic antidepressant clomipramine has been shown to be somewhat effective, but patients' compliance is often poor. Highly selective serotoninergic receptor reuptake inhibitors (SSRIs), such as citalopram, paroxetine, and fluvoxamine, might be effective. First-line therapy includes clomipramine or SSRIs. Lithium carbonate, which decreases neuronal excitability, and naltrexone, an opiate antagonist, might also be helpful. Habit reversal training is the most effective behavioural therapy; it targets the obsessive-compulsive disorder that causes patients to tear out their hair.

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Dermacase

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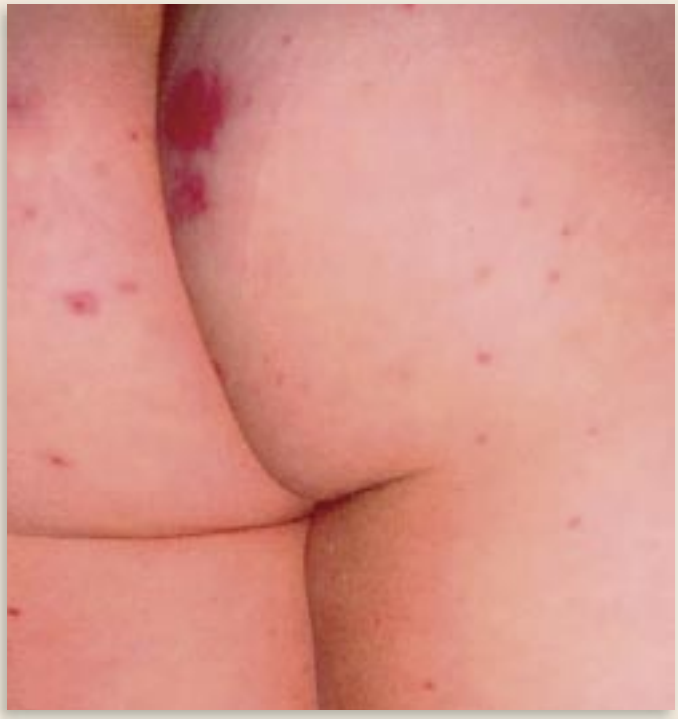

CAN YOU IDENTIFY THIS CONDITION?

An 11-year-old boy presented complaining that he had had a non-itchy rash on his legs and buttocks for 3 days. He had slight abdominal discomfort, but otherwise felt well. Examination revealed purpuric papules scattered over his posterior thighs. His right ankle was mildly edematous, but the rest of his examination was unremarkable.

The most likely diagnosis is:

1. Papular urticaria
2. Systemic lupus erythematosus
3. Henoch-Schönlein purpura
4. Meningococcemia
5. Dermatitis herpetiformis

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Dr Freiman *is a dermatology resident at the McGill University Health Centre in Montreal, Que.*

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3. Henoch-Schönlein purpura

Henoch-Schönlein purpura (HSP) is one of the most common forms of vasculitis of childhood. It is characterized by immunoglobulin A deposits in the walls of small blood vessels.[1] The estimated annual incidence of HSP is 20.4/100 000 people and is highest in boys between the ages of 4 and 6.[2]

Clinically, HSP presents with a classic tetrad of symptoms that have documented relative frequencies: cutaneous purpura (100%), arthralgia or arthritis (82%), abdominal pain (63%), and gastrointestinal bleeding (33%).[3] Characteristic primary skin lesions are purpuric papules symmetrically distributed on the buttocks, legs, and extensor extremities. Arthralgia commonly affects the knees and ankles, but resolves without permanent damage to joints. Gastrointestinal bleeding and intussusception are rarer complications of HSP and are presumably initiated by edematous vasculitis of the small-bowel mucosa.[4] Occasionally, the central nervous system and the respiratory system are also affected.

The serious sequela of HSP is its renal involvement, which typically takes place within a few days to several weeks after onset of systemic symptoms. Heralded by asymptomatic microscopic hematuria and proteinuria, it occurs in 30% to 70% of patients, but it is usually mild and self-limiting. With increasing age at presentation, however, more marked complications can arise, including nephrotic syndrome, hypertension, and acute renal failure. Children diagnosed with HSP need their renal status closely monitored with repeat urinalyses and renal function tests and referral to a pediatric nephrologist if necessary.

The etiology of HSP is unclear. It is frequently associated with upper respiratory tract infection, which is consistent with its peak occurrence in the winter and fall. Patients usually present with a 2- to 3-week history of fever, headache, myalgia, arthralgia, and abdominal pain preceding the typical cutaneous purpura. Group A streptococci, mycoplasma, and a variety of other infectious agents and drugs have been reported as potential triggers. Diagnosis of HSP is often made on the basis of clinical signs and symptoms and can be confirmed with direct immunofluorescence of the skin biopsy sample, which demonstrates leukocytoclastic vasculitis with perivascular immunoglobulin A, immunoglobulin C3, and fibrin deposits.

Henoch-Schönlein purpura is usually benign and resolves spontaneously, so treatment is mostly supportive, including adequate hydration. Long-term prognosis largely depends on the severity of renal involvement. About 94% of children and 89% of adults recover completely.[5] Nonsteroidal anti-inflammatory drugs can be used to treat arthralgia associated with HSP. While systemic corticosteroids might help with some symptoms, such as arthritis and abdominal pain, no form of therapy has yet been shown in randomized trials to have an effect on the duration of illness or recurrences. Corticosteroids, cytotoxic agents, and intravenous immunoglobulin therapy might be of benefit in advanced disease, defined as crescentic nephritis.[6]

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