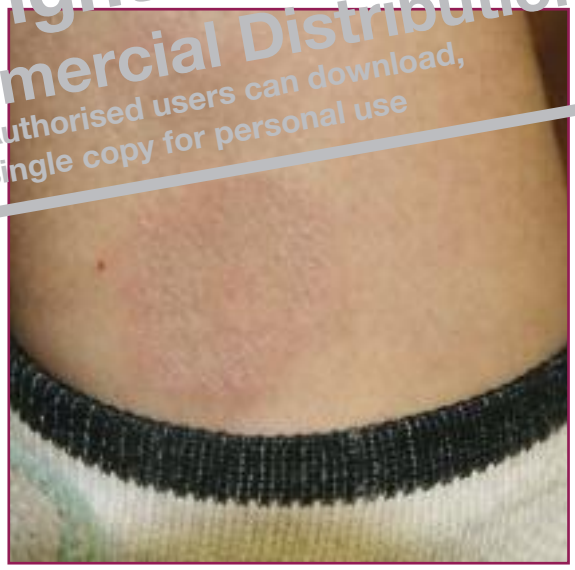
Tender, Flesh-coloured Nodules

By Simon Lee, MD, FRCPC

A healthy 5-year-old girl presents to the office with a persistent asymptomatic eruption of two months duration. On examination, slightly tender, flesh-coloured nodules are noted on the left medial ankle forming an annular plaque.



What is your diagnosis?

a. Lichen planus
b. Discoid lupus erythematosus
c. Milia
d. Granuloma annulare
e. Lichen striatus

Answer: Granuloma annulare

Granuloma annulare (GA) is a benign, cutaneous disorder of unknown etiology. It is characterized by numerous flesh-coloured, dermal papules that are often arranged in a distinctive, annular pattern. Microscopically, there are focal areas of degenerative collagen with granulomatous inflammation, hence the name granuloma annulare.

Although the true incidence of GA is not known in the general population, it is not an uncommon disorder in a general dermatology practice. There is no geographic or racial predilection; however, females are more affected.

Four major clinical variants are encountered. First, localized GA is the most common subtype. Plaque formation may reach up to 5 cm in diameter. Individual lesions do not have any significant surface alteration. Typically, localized GA occurs on the extremities, especially the lower legs.

Second, the generalized form consists of numerous plaques on the trunk and extremities. It is more commonly seen in adults. There is a loose association with type 1 diabetes mellitus.

Third, subcutaneous GA may also occur in adults. Pathology resembles the rheumatoid nodules often seen in patients with rheumatoid arthritis; therefore, a rheumatoid factor may be requested as part of investigations. More frequently, subcutaneous GA occurs in otherwise healthy children. Somewhat confusingly, subcutaneous GA may be referred to as "rheumatoid nodules" in pediatric literature.

Finally, perforating GA may also be seen in children and typically the lesions display surface ulceration.

Various immunologic theories, including cell mediated (type IV) immunity as well as immune complex vasculitis have been proposed; however, the precise mechanism of change remains to be determined. Some authors favour a primary

degeneration of collagen, triggering a reactive immunologic phenomenon. Initial, associated triggering events include insect bites, trauma, sun exposure, vaccinations, and viral infections, including hepatitis B and C, herpes, and Epstein-Barr virus.

In the great majority of cases, the history and clinical findings are sufficient for diagnosis. Painful or rapidly enlarging lesions with atypical presentation may warrant a skin biopsy. Selective blood work, such as fasting glucose, urinalysis, CBC count, and rheumatoid factor, may be obtained depending on clinical circumstances.

The prognosis for GA is excellent, especially for children, since spontaneous resolution is the natural course of history. Occasionally, lesions may be tender, painful, or cause significant emotional distress. Potent topical steroid preparations may hasten resolution and alleviate concerns. Options include cryotherapy and intralesional steroid injection; however, patient reluctance may limit therapeutic success. There have also been some reports of success using topical calcineurin inhibitors, such as tacrolimus, and imiquimod cream.

In patients with unsightly disseminated GA, isotretinoin, pentoxyfilline, and ultraviolet light therapy combined with psoralens (PUVA) may be employed to control symptoms of pruritus and improve cosmetic appearance. Judicious use of oral steroids may also be reserved for recalcitrant cases. Approximately one half of all cases will resolve spontaneously within two years of onset.

Dx

References
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Dr. Simon Lee is a Dermatologist in Richmond Hill, Ontario.

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Our mailing address:
6500 Trans-Canada
Suite 310
Pointe-Claire, Québec
H9R 0A5

Our fax number:
(514) 695-8554

Our e-mail address:
diagnosis@sta.ca