

H&E, original magnification $\times 200$.



H&E, original magnification \times 400.

The best diagnosis is:

- a. eruptive xanthoma
- b. lepromatous leprosy
- c. tuberous xanthoma
- d. verruciform xanthoma
- e. xanthelasma

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Eruptive Xanthoma

E ruptive xanthomas arise in the setting of elevated levels of chylomicrons,¹ often occurring in cases of uncontrolled diabetes mellitus, alcohol dependence, medication use (eg, isotretinoin),² and familial hyperlipidemias (Frederickson types I, IV, and V).¹ Classically, lesions present as multiple smooth red-yellow papules that occur in eruptive crops on the buttocks and extensor surfaces of the extremities and may koebnerize.³ Patients who present with eruptive xanthomas should have their lipid panels checked, as they may have extremely elevated levels of triglycerides that would predispose them to acute pancreatitis. Characteristically, eruptive xanthomas show collections of foamy histiocytes in the dermis on histology along with extracellular lipids (Figure 1). In contrast, xanthelasma has a characteristic clinical distribution around the eyes with histology demonstrating vellus hair follicles, thin loose dermal collagen, and skeletal muscle bundles. Although similar collections of foamy histiocytes are seen in xanthelasma, extracellular lipid accumulation is not prominent (Figure 2). Lepromatous leprosy typically shows a grenz zone with sheets of foamy histiocytes known as Virchow cells (Figure 3).



Figure 1. Both intracellular and extracellular lipid deposition (A and B)(H&E; original magnifications \times 200 and \times 400).



Figure 2. Xanthoma cells without prominent extracellular lipid deposition in a case of xanthelasma (H&E, original magnification $\times 100$).



Figure 3. Lepromatous leprosy with sheets of foamy histiocytes in the dermis and some globi. Arrow points to the epidermis (H&E, original magnification ×200).

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Figure 4. Tuberous xanthoma with pronounced fibrosis in addition to xanthomatous cells with cholesterol cleft formation (H&E, original magnification ×100).



Figure 5. Verruciform xanthoma with characteristic orange parakeratosis with a papillomatous and acanthotic epidermis (left)(H&E, original magnification ×100) along with foamy histiocytes in the dermal papillae (right)(H&E, original magnification ×400).

Globi consisting of collections of Mycobacterium leprae sometimes can be identified (Figure 3) and are acid-fast positive.⁴ Patients with lepromatous leprosy present with numerous anesthetic lesions that are symmetrically distributed² and may present with leonine facies. Tuberous xanthoma also may be seen in several secondary and hereditary hyperlipidemias, most characteristically in familial dysbetalipoproteinemia, as well as in homozygous familial hypercholesterolemia, cerebrotendinous xanthomatosis, β-sitosterolemia, and type IV familial hyperlipoproteinemia.¹ The process is chronic rather than acute, which is revealed by the presence of fibrosis accompanying the collections of xanthoma cells; additionally, cholesterol clefts may be seen (Figure 4). Verruciform xanthoma predominantly occurs in the oral cavity or genitalia.¹ Most patients do not have an association with hyperlipidemia. The etiology of verruciform xanthoma is unknown, though it has been suggested that keratinocyte necrosis may play a role.¹ It is characterized by a vertucous surface with acanthosis and parakeratosis and an orange hue. The dermal papillae contain foamy histiocytes (Figure 5).

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