

Case Report

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Eruptive xanthomas revealing severe hypertriglyceridemia: A dermatologic window into metabolic crisis

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Abstract

Eruptive xanthomas are rare cutaneous manifestations characterized by lipid deposition in the skin. They are often underrecognized but may represent the first clinical sign of an underlying lipid metabolism disorder, such as severe hypertriglyceridemia. Clinically, they appear as yellowish papules or nodules, frequently surrounded by an erythematous halo, and are predominantly located in areas of friction, extensor surfaces, the back, and the gluteal region. Histopathological examination reveals numerous foamy histiocytes within the dermis. Management is centered on metabolic control through a multidisciplinary approach. We report the case of a 40-year-old male who presented to the dermatology clinic with yellowish papuloid lesions. Laboratory findings revealed marked hypertriglyceridemia (7450 mg/dL), and the diagnosis of eruptive xanthomas was confirmed via histopathological analysis. The patient was treated with subcutaneous insulin and lifestyle modifications, leading to clinical improvement.

Keywords: Dermatological manifestations; Lipid-laden foamy Histiocytes; Hyperlipidemia; Disorder of lipid metabolism.

Introduction

Eruptive xanthomas are uncommon dermatological manifestations caused by dermal deposition of lipids, primarily triglycerides, in the context of severe dyslipidemia [1]. The pathophysiology of this condition involves a marked elevation of chylomicrons and plasma lipoproteins, influenced by genetic factors that are classified into various subtypes (Table 1) [2-4]. Lipoproteins, responsible for lipid transport throughout the body, are classified into five types based on their density and follow two main metabolic pathways. The exogenous pathway begins in the intestine with the formation of chylomicrons, which are processed by the enzyme Lipoprotein Lipase (LPL) to release free fatty acids. The remnants are subsequently taken up by the liver. In the endogenous pathway, the liver synthesizes Very-Low-Density Lipoproteins (VLDL), which are hydrolyzed by LPL to produce Intermediate-Density Lipoproteins (IDL), and

eventually Low-Density Lipoproteins (LDL). These particles may be cleared or deposited in various tissues, including the dermis, where they are phagocytosed by cutaneous macrophages that transform into foamy cells [5]. The localized accumulation of these cells in the superficial dermis gives rise to sudden-onset, yellowish papular lesions, typically surrounded by an erythematous halo and associated with local inflammation [6]. This condition may coexist with poorly controlled diabetes mellitus, metabolic syndrome, and familial dyslipidemias [7]. Due to its rarity, eruptive xanthomatosis is often underrecognized in clinical practice, potentially delaying the diagnosis of primary or secondary dyslipidemias [8]. Under this background, the presentation of clinical cases is essential to promote early recognition and comprehensive management through a multidisciplinary approach involving internal medicine, endocrinology, nutrition, and dermatology [9].

Case history

A 40-year-old male patient, with no known personal pathological history, presented with disseminated cutaneous lesions. Physical examination revealed a widespread dermatosis involving the trunk, shoulders, and extremities, with a bilateral distribution and predominance on extensor surfaces. The lesions consisted of multiple exophytic yellowish papular and nodular formations, set on an erythematous base, with regular borders, ranging in size from 0.2 to 1 cm in diameter. The Koebner phenomenon was observed. The lesions were asymptomatic (Figure 1). Based on the clinical presentation, differential diagnoses included eruptive histiocytosis, sarcoidosis, disseminated granuloma annulare, and eruptive xanthomas. Laboratory investigations were ordered, revealing: complete blood count within normal limits, serum glucose of 165 mg/dL, creatinine 0.95 mg/dL, uric acid 11.5 mg/dL, triglycerides 7450 mg/dL, and total cholesterol 658 mg/dL. An excisional biopsy of one nodular le-

sion was performed. Histopathological examination showed vacuolated histiocytes with a foamy appearance and multinucleated Touton giant cells in the superficial and mid-dermis (Figure 2). Based on clinical, biochemical, and histopathological findings, a diagnosis of eruptive xanthomas secondary to severe hypertriglyceridemia and hypercholesterolemia was confirmed. The patient was referred to the internal medicine department, where treatment was initiated with intravenous insulin infusion and a strict sugar-free diet. Subsequently, pharmacological therapy was instituted with bezafibrate (200 mg every 12 hours), subcutaneous insulin glargine (10 IU daily), and atorvastatin (80 mg daily), resulting in excellent therapeutic response. At the one-month follow-up, laboratory results showed: glucose 110 mg/dL, creatinine 0.96 mg/dL, uric acid 8.8 mg/dL, total cholesterol 366 mg/dL, and triglycerides 1870 mg/dL. After one year of treatment, triglyceride levels had decreased to 108 mg/dL (Table 2), with complete resolution of the cutaneous lesions.

Table 1: Classification of hyperlipidemias and association with Xanthomonas.

Type	Name	Elevated lipoproteins	Main mechanism	Involved genes/causes	Association with xanthomas
Type I [16]	Hyperchylomicronemia	Chylomicrons	LPL deficiency or cofactors preventing triglyceride hydrolysis	LPL, APOC2, APOA5, LMF1, GPIHBP1	Frequent eruptive xanthomas
Type IIa [17]	Familial hypercholesterolemia	LDL	Decreased LDL clearance due to receptor defects	LDLR, APOB, PCSK9	Tendinous and tuberous xanthomas
Type IIb [18]	Familial combined hyperlipidemia	LDL and VLDL	Overproduction of apoB100 and VLDL	Multifactorial (genetic + lifestyle)	Occasional eruptive xanthomas
Type III [19]	Dysbetalipoproteinemia	Remnants of VLDL and chylomicrons	Defective ApoE binding to LDL receptor	APOE (homozygosity E2/E2)	Tuberous, palmar, and eruptive xanthomas
Type IV [20]	Endogenous hypertriglyceridemia	VLDL	Hepatic overproduction of VLDL	Obesity, diabetes, insulin resistance	Eruptive xanthomas in severe cases
Type V [21]	Mixed hyperlipidemia	Chylomicrons and VLDL	Combined alterations in exogenous and endogenous pathways	Genetic + secondary factors (alcohol, diet, diabetes)	Frequent eruptive xanthomas and pancreatitis

LDL: Low-Density Lipoproteins; VLDL: Very-Low-Density Lipoproteins; LPL: Lipoprotein Lipase; Ape: Apolipoprotein E.



Figure 1: Yellowish exophytic neoformations with papular and nodular appearance, showing well-defined.

Table 2: Monitoring of triglyceride and cholesterol levels at different time intervals.

Clinical moment	Triglycerides (mg/dL)	Total, colessterol (mg/dL)
Beginning	7450	658
One month	1870	366
A year	108	147

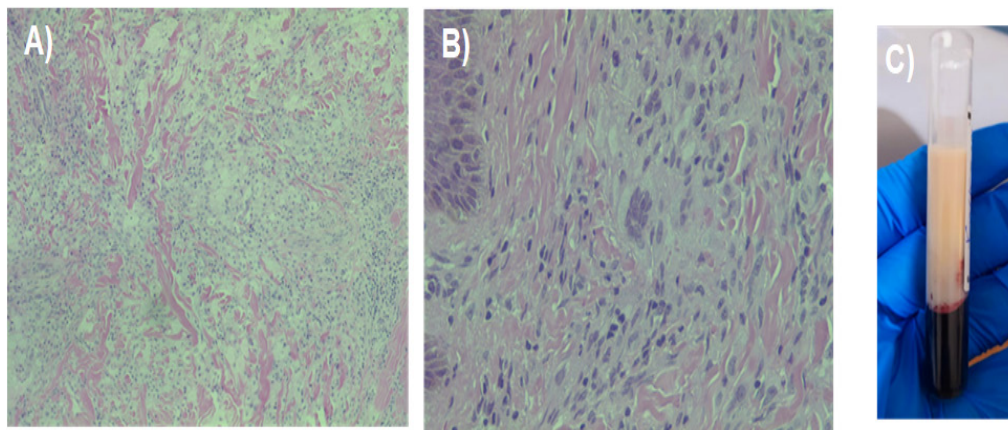


Figure 2: (A) H&E staining showing vacuolated histiocytes with a foamy appearance and wide, pale cytoplasm. (B) H&E staining revealing vacuolated histiocytes and formation of sparse multinucleated Touton-type giant cells. (C) Lipemic sample obtained from the patient.

Discussion

In the case presented, a 40-year-old male patient exhibited characteristic cutaneous lesions and markedly elevated triglyceride levels of 7,450 mg/dL, placing him within the range associated with a high risk for pancreatitis and cardiovascular events. Numerous reports have highlighted the association between eruptive xanthomas and severe hypertriglyceridemia [10-13]. Tang et al. described a case involving a male patient with triglyceride levels of 6,697 mg/dL and total cholesterol of 943 mg/dL, where the lesions were distributed over extensor surfaces and showed resolution following intensive metabolic control [10]. Similarly, Muñoz et al. reported a patient with initial triglyceride levels of 6,284 mg/dL and extensive cutaneous manifestations, whose lesions resolved after treatment with insulin and fibrates [11]. Both cases are comparable to the present report in terms of laboratory findings and clinical presentation. Pierce et al. and Nayak et al. also documented cases involving young patients with yellowish-erythematous lesions affecting the trunk and extremities, associated with triglyceride levels exceeding 3,000 mg/dL. In both cases, favorable outcomes were achieved through lipid-lowering interventions [12]. Saoji et al., from India, reported a patient with lesions on the palms and elbows, and extremely elevated triglyceride levels (7,754 mg/dL), attributed to type V hyperlipidemia. Clinical remission was achieved with a combination of statins, omega-3 fatty acids, and cefloglitazar [13]. Although the histopathological characteristics of eruptive xanthomas (i.e., foamy histiocytes in the reticular dermis) and their anatomical distribution tend to be consistent, the precipitating factors may vary. Some authors have identified poorly controlled diabetes as the primary trigger [14], while others have reported associations with alcohol consumption [15], genetic predisposition, and the use of specific medications [16]. In the present case, an interdisciplinary approach resulted in a progressive reduction of lipid levels—from 7,450 mg/dL to 108 mg/dL within one year (Table 2) accompanied by complete resolution of the cutaneous lesions. This clinical course supports existing literature indicating that appropriate management of dyslipidemia, combined with nutritional and pharmacologic interventions, is crucial for the remission of eruptive xanthomas [8,13]. Despite being a rare condition, eruptive xanthomas should be recognized as important clinical indicators of potentially serious metabolic disorders.

Conclusion

Eruptive xanthomas, while infrequent, represent a critical dermatological sign that may be the first indication of severe underlying metabolic disorders. The dermatologist plays a pivotal role in recognizing these lesions and initiating a timely and comprehensive diagnostic approach. As demonstrated in this case, cutaneous manifestations can precede or coincide with life-threatening conditions such as acute pancreatitis, premature cardiovascular disease, and, in extreme cases, sudden death. Early identification and multidisciplinary management are essential to reduce morbidity and improve long-term outcomes. Therefore, awareness and prompt referral by dermatologists can significantly influence patient prognosis.

Declarations

Competing interests: The authors declare no competing interest.

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