Eruptive Xanthoma: A Case of Extreme Hypertriglyceridemia

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Abstract

Introduction: Eruptive xanthoma (EX) is a cutaneous marker of severe hypertriglyceridemia and uncontrolled diabetes mellitus, often preceding life-threatening complications such as acute pancreatitis and cardiovascular disease.

Case presentation: A 30-year-old man with type 2 diabetes mellitus (T2DM) and recent heavy alcohol intake (> 200 g/day) presented with a 3-day history of generalized, non-pruritic papules. Examination revealed erythematous-to-yellowish papules on the trunk and extremities, predominantly on extensor surfaces. Dermoscopy revealed a central yellowish structureless area surrounded by an erythematous brown halo. Biopsy showed dermal foam cells and cholesterol clefts, confirming EX. Laboratory findings included fasting glucose 28.00 mmol/L, glycated hemoglobin (HbA1c) 13.9%, triglycerides 77.10 mmol/L, and total cholesterol 19.03 mmol/L.

Results: The patient was managed with intravenous continuous infusion of regular insulin, prophylactic subcutaneous unfractionated heparin, oral atorvastatin 20 mg daily plus fenofibrate 200 mg once daily, and a strict low-fat diet. On day 7, insulin was transitioned to oral metformin 850 mg twice daily plus sitagliptin 100 mg once daily. At discharge, fasting glucose (5.70 mmol/L), total cholesterol (5.14 mmol/L), and triglycerides (9.77 mmol/L) had improved substantially, with triglycerides remaining elevated. The cutaneous lesions began to involute within 3 weeks, with near-complete resolution documented at 3-month follow-up.

Discussion: EX serves as an early cutaneous biomarker of severe dyslipidemia, most commonly associated with uncontrolled diabetes and hypertriglyceridemia. Histopathology remains the diagnostic gold standard when clinical features are equivocal. In patients without hypertriglyceridemia, a paraneoplastic syndrome should be considered. Timely management of the underlying metabolic disorder not only resolves skin lesions but also reduces the risk of acute pancreatitis and cardiovascular events.

Conclusion: This case highlights that systematic evaluation of metabolic parameters, including glucose, lipid profile, and family history, enables prompt diagnosis of EX. Effective control of hypertriglyceridemia and diabetes leads to complete resolution of cutaneous manifestations and prevents serious systemic complications.

Keywords: eruptive xanthoma; hypertriglyceridemia; type 2 diabetes mellitus; alcohol

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Introduction

Xanthomas are cutaneous lesions resulting from localized lipid deposition, classified according to clinical morphology and pattern of onset. Eruptive xanthoma (EX) represents a critical dermatologic marker of profound metabolic derangement[1]. EX manifests as rapidly evolving, erythematous-to-yellowish papules, typically 1–5 mm in diameter, distributed over the extensor surfaces of the extremities and buttocks, with occasional involvement of palmar and plantar creases. Lesions may be tender, and pruritus may accompany early-stage disease[2]. EX is frequently associated with diabetes mellitus, hypercholesterolemia, severe hypertriglyceridemia, lipemia retinalis, and hepatic steatosis. Recognition of the underlying metabolic disorder is paramount, as it enables prevention of potentially life-threatening complications, including coronary artery disease and acute pancreatitis[3].

Case presentation

A 30-year-old man presented with a 3-day history of generalized, non-pruritic papules. His medical history was notable for type 2 diabetes mellitus (T2DM) of 3 years' duration. Over the past 3 years, he had been taking metformin 500 mg twice daily irregularly with poor adherence. He reported polyuria and polydipsia for 1 week prior to presentation but denied weight loss. He also reported consuming more than 200 g of alcohol daily during the preceding week. The patient had a smoking history of approximately 10 years. Family history was positive for diabetes mellitus (father) but negative for hyperlipidemia, premature cardiovascular disease, or recurrent pancreatitis. On physical examination, the patient was 180 cm tall with a body weight of 83.1 kg (body mass index [BMI] 25.66 kg/m^2). Dermoscopy revealed a central yellowish structureless area surrounded by an erythematous brown halo. Scattered erythematous-to-yellowish papules, ranging from several millimeters to 1 cm, were observed on the trunk and extremities in a linear arrangement (Fig. 1A). The lesions predominated on the extensor surfaces of the upper arms, back, and lower legs. Blood pressure was normal. Chest computed tomography (CT) was performed as part of routine infection screening in the setting of severe hyperglycemia and to screen for paraneoplastic syndrome, given that EX may occasionally be associated with underlying malignancy or paraneoplastic conditions, and showed no significant abnormalities. Abdominal ultrasonography revealed mild hepatic steatosis. A punch biopsy from a representative lesion on the upper arm demonstrated dermal aggregates of histiocytes with cholesterol cleft deposition and foam cell formation (Fig. 1B), confirming EX.

Laboratory investigations uncovered markedly deranged metabolic parameters. Fasting plasma glucose was 28.00 mmol/L, and glycated hemoglobin (HbA1c) was 13.9% (0%–6%). Total cholesterol was 19.03 mmol/L (< 5.20 mmol/L), triglycerides 77.10 mmol/L (< 1.70 mmol/L), and phospholipids 8.77 mmol/L (1.90–3.20 mmol/L).

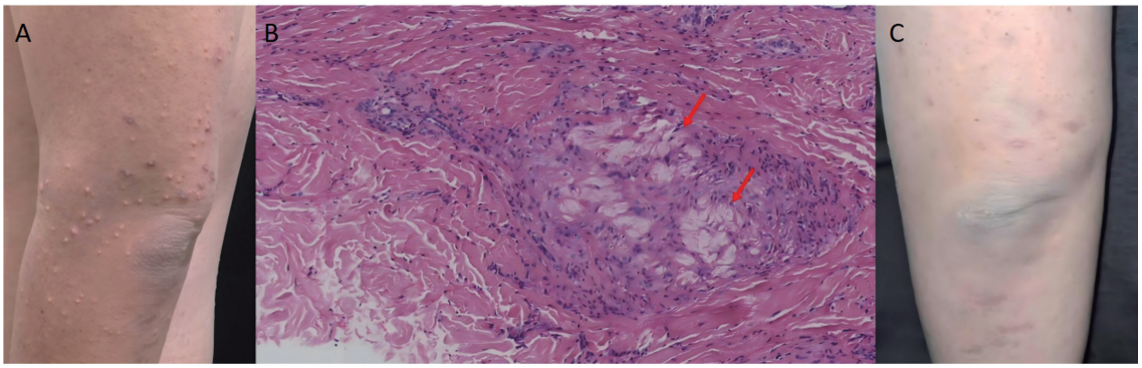

Figure 1: Clinical and histopathological features of eruptive xanthoma.

(A) Clinical photograph of the elbow at presentation, demonstrating erythematous-to-yellowish papules arranged in a linear pattern over the extensor surface. (B) Histopathology (hematoxylin and eosin stain, original magnification 40×; eyepiece 10×) shows dermal cholesterol cleft deposition. (C) Three-month follow-up photograph demonstrating near-complete resolution of xanthomatous lesions.

Diabetic ketoacidosis (DKA) and hyperosmolar hyperglycemic state (HHS) were excluded based on arterial blood gas (potential of hydrogen [pH] 7.40, HCO_3^- 24.00 mmol/L, base excess +1.00 mmol/L), anion gap of 12.00 mmol/L, serum potassium 4.30 mmol/L, serum sodium 140.00 mmol/L, plasma osmolality 300 mOsm/kg, and serum creatinine 78.00 μmol/L with estimated glomerular filtration rate (eGFR) > 90 mL/min/1.73 m^2. Urinalysis showed urine glucose +3 (28.00 mmol/L) and urine ketones +1 (1.50 mmol/L); in the context of normal blood pH, normal anion gap, and normal plasma osmolality, this mild ketonuria was attributed to alcohol-related ketonuria rather than ketoacidosis. The patient was fully alert with no signs of dehydration or hemodynamic compromise. Thyroid-stimulating hormone (TSH) was 2.10 mIU/L (0.27–4.20 mIU/L). High-density lipoprotein cholesterol (HDL-C) was 5.92 mmol/L (1.00–2.20 mmol/L); however, this result may be artifactually elevated due to interference from severe chylomicronemia and should be interpreted with caution, as it is inconsistent with the expected metabolic profile of severe hypertriglyceridemia, in which HDL-C is typically low. Low-density lipoprotein cholesterol (LDL-C) was 1.43 mmol/L (< 3.40 mmol/L), directly measured by a direct homogeneous assay. Non-high-density lipoprotein cholesterol (non-HDL-C) was 13.11 mmol/L [calculated as total cholesterol (19.03) – HDL-C (5.92)]. Apolipoprotein A1 (ApoA1) was 1.11 g/L (1.12–1.62 g/L), apolipoprotein A2 (ApoA2) was 0.13 g/L (0.25–0.52 g/L), apolipoprotein B (ApoB) was 1.23 g/L (0.69–1.05 g/L), free fatty acid (FFA) was 0.44 mmol/L (0.10–0.90 mmol/L), and apolipoprotein E (ApoE) was 2.92 mg/dL (2.70–4.90 mg/dL). Lipoprotein(a) [Lp(a)] testing was not performed due to severe chylomicronemia. Serum amylase and lipase were within normal limits, and the patient denied abdominal pain, excluding acute pancreatitis. Complete blood count, hepatic enzymes, and coagulation parameters were within normal limits.

The patient was managed through a multidisciplinary approach targeting both type 2 diabetes and severe dyslipidemia. Because DKA/HHS had been definitively excluded, a continuous intravenous infusion of low-dose regular insulin was initiated at an initial rate of 2 U/h. with hourly point-of-care blood glucose monitoring. This approach is appropriate for non-ketotic severe hyperglycemia and avoids the risks of hypokalemia or cerebral edema associated with high-dose insulin protocols. Concurrently, given the patient's chylomicronemic state and immobility-related risk, prophylactic subcutaneous unfractionated heparin 5,000 U every 12 h for 5 days was administered to prevent venous thromboembolism (VTE). The patient had been on long-term oral atorvastatin 20 mg daily prior to admission. Upon presentation, fenofibrate 200 mg once daily was added for intensified triglyceride lowering, while the existing atorvastatin 20 mg daily was continued, and a strict low-fat diet was maintained. As plasma glucose and triglyceride levels gradually declined, the insulin infusion rate was correspondingly reduced. On day 7, insulin therapy was transitioned to oral metformin 850 mg twice daily plus sitagliptin 100 mg once daily (quaque die [QD]), with atorvastatin 20 mg daily and fenofibrate 200 mg once daily maintained. A low-sugar, low-fat diet was strictly followed throughout hospitalization. At discharge, laboratory values had improved substantially, with triglycerides remaining severely elevated (9.77 mmol/L); fasting glucose was 5.70 mmol/L and total cholesterol was 5.14 mmol/L. Concurrently, the papular lesions progressively involuted. After discharge, the patient was maintained on atorvastatin 20 mg daily plus fenofibrate 200 mg once daily as combination lipid-lowering therapy. Omega-3 fatty acids were not initiated or planned, as the combination of statin plus fibrate was considered sufficient for this patient's triglyceride management at that time. The long-term management strategy focused on strict glycemic control, a low-sugar and low-fat diet, complete alcohol abstinence, and continued dual therapy with statin plus fibrate. Close outpatient follow-up was planned to monitor triglyceride response and adjust therapy accordingly. At the 3-month follow-up, metabolic parameters had markedly improved: fasting glucose 7.00 mmol/L, triglycerides 2.40 mmol/L, total cholesterol 4.58 mmol/L, HDL-C 1.08 mmol/L, LDL-C 1.41 mmol/L, HbA1c 7.1%, and Lp(a) 168 mg/L. The papular lesions began to involute within 3 weeks of initiating therapy, and near complete resolution of all cutaneous lesions was documented (Fig. 1C).

Discussion

Lipid disorders rank among the most prevalent risk factors for cardiovascular disease globally. Xanthomas serve as early cutaneous manifestations of lipid disorders,

with a predilection for severe hypertriglyceridemia; the overall incidence is estimated at approximately 18 cases per 100,000 individuals[4]. The diagnosis of EX relies primarily on characteristic clinical presentation, dermoscopic findings, and laboratory abnormalities. Histopathology remains the gold standard for confirming the presence of dermal foam cells when the diagnosis remains equivocal.

The development of EX in this patient was not attributable to a single factor but resulted from the interplay of metabolic, genetic, and behavioral factors, representing the classic pathophysiological pattern of multifactorial chylomicronemia syndrome (MFCS)[5,6]. Long-standing uncontrolled T2DM led to relative insulin deficiency, significantly reducing the activity of lipoprotein lipase (LPL), the key enzyme responsible for hydrolyzing triglycerides (TG) in chylomicrons and very low-density lipoprotein (VLDL). Simultaneously, insulin resistance increased adipose tissue lipolysis, flooding the liver with FFAs and stimulating excessive ApoB-100 synthesis and massive VLDL secretion. This metabolic derangement explained the dissociation observed in this case—elevated ApoB (1.23 g/L) with relatively LDL-C (1.43 mmol/L)—wherein the elevated ApoB was carried primarily by VLDL and chylomicrons rather than low-density lipoprotein (LDL). Heavy alcohol consumption further amplified hypertriglyceridemia by inhibiting LPL activity and increasing hepatic VLDL synthesis; on the foundation of preexisting insulin resistance and potential genetic susceptibility, alcohol served as the precipitating factor that pushed moderate hypertriglyceridemia into extreme chylomicronemia (TG >11.30 mmol/L). When serum TG exceeds approximately 11.30 mmol/L, chylomicron particles are too large to traverse normal vascular endothelium; however, under conditions of increased capillary permeability induced by hyperglycemia-related endothelial injury and inflammatory cytokine release, lipoproteins extravasate into the dermis, are phagocytosed by tissue macrophages forming foam cells, and aggregate into xanthoma nodules[7]. The distribution of lesions on extensor surfaces of the extremities and buttocks in this case was consistent with the classic predilection sites of EX and was also related to increased local capillary permeability due to pressure and friction, suggesting that the mechanical irritation may have favored local lipid extravasation.

Although the diagnosis of EX was established based on typical clinical presentation, severe hypertriglyceridemia, and histopathological findings, several differential diagnoses were considered and excluded. Disseminated granuloma annulare typically presents as skin-colored or erythematous annular papules or plaques with palisading granulomas and mucin deposition on histopathology[8], whereas the lesions in this case were yellow papules rather than annular plaques, and histopathology showed foam cells and lipid deposition rather than mucin. Generalized eruptive histiocytoma is characterized by firm, reddish-brown or brown papules appearing in crops on the face, trunk, and proximal extremities, with mononuclear histiocytic infiltration rather than foam cells on pathology[9]; the prominent yellow hue of the lesions, the presence of typical foam cells on histopathology, and the explainable metabolic background allowed exclusion of this entity. Langerhans cell histiocytosis (LCH) is more common in children and presents with seborrheic dermatitis-like or purpuric papules, with Langerhans cells visible on pathology[10]; this patient was an adult, and histopathology showed foam cells rather than Langerhans cells, which helped exclude LCH.

Notably, xanthomas may also be associated with hematologic malignancies. In patients presenting with EX in the absence of hypertriglyceridemia, a paraneoplastic syndrome should be entertained; thus, the emergence of xanthomatous lesions may prompt further investigation for underlying hematologic neoplasms[11].

Recognizing xanthomas as early biomarkers of lipid disorders is imperative for timely diagnosis and intervention. Control of underlying hypertriglyceridemia not only facilitates resolution of cutaneous lesions but also mitigates the risk of life-threatening complications. This case underscores that systematic evaluation of multiple factors influencing lipid metabolism—including age, family history, and lipoprotein metabolic status—is critical to achieving prompt diagnosis in patients with EX. The absence of a family history of hyperlipidemia does not exclude underlying genetic susceptibility; rather, the family history of diabetes supports insulin resistance

as the core secondary factor, consistent with the MFCS paradigm. Synthesis of these data enables elucidation of etiology and formulation of optimal therapeutic strategies.

Conclusion

This case demonstrates that EX can serve as a striking cutaneous sentinel of profound hypertriglyceridemia and uncontrolled T2DM. Prompt recognition of the characteristic papular eruption, combined with histopathological confirmation and aggressive metabolic management—including glycemic control, lipid-lowering therapy, and dietary modification—can lead to complete resolution of skin lesions and substantial improvement in systemic parameters. Clinicians should maintain a high index of suspicion for EX in patients with poorly controlled diabetes and severe hypertriglyceridemia, as early diagnosis not only resolves cutaneous manifestations but also mitigates the risk of life-threatening complications such as acute pancreatitis and cardiovascular disease.

Author contributions

Zining Xu conceived the study, drafted the manuscript, and supervised the work; Lingbo Bi and Ziyi Wang conceived the study; Yujun Sheng and Yong Cui provided resources. All authors reviewed, edited, and approved the final version of the manuscript and agreed to its publication.

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Ethics statement

In accordance with the institutional review board (IRB) guidelines of China-Japan Friendship Hospital, ethical approval was waived for this single case presentation, as it involved no experimental interventions and used only de-identified patient data. This study was conducted in accordance with the Declaration of Helsinki.

All figures are either original to this work or properly attributed. No original images have been previously published, and all images comply with research integrity standards without inappropriate manipulation or misrepresentation.

Patient consent statement

Written informed consent was obtained from the patient for publication of this case presentation and accompanying images.

Data availability statement

The data that support the findings of this study are available from the corresponding author upon reasonable request, but are not publicly available due to privacy or ethical restrictions.

AI statement

The authors used ChatGPT (GPT-4, online version) as a language-editing tool to improve grammar, clarity, and readability. The AI tool was not used for data analysis, data generation, image processing, or scientific content creation. All outputs were carefully reviewed and verified by the authors, who take full responsibility for the final content.

Conflicts of interest

Yong Cui serves as one of the Editors-in-Chief of this journal. He was not involved in the editorial review or decision-making process for this manuscript. All editorial decisions were made independently by other members of the Editorial Board who have no conflicts of interest.

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