BULLOSIS DIABETICORUM COMPLICATED BY SECONDARY INFECTION AND NECROSIS IN A 29-YEAR-OLD MALE WITH NEWLY DIAGNOSED DIABETES MELLITUS: A CASE REPORT

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Abstract
Bullosis diabeticorum (BD) is a rare cutaneous manifestation of diabetes mellitus characterized by spontaneous bullous eruptions, predominantly on the extremities. This report describes a 29-year-old male car driver from Dubai who presented with pruritus on the right foot, progressing to vesicles, papules, pustules, bullae, and eventual necrosis despite multiple interventions. The condition was complicated by secondary infection and swelling, with laboratory findings revealing mild leukocytosis, eosinophilia, and elevated HbA1c, confirming newly diagnosed diabetes mellitus. Multidisciplinary management involving antimicrobial therapy, antifungal agents, wound care, and lifestyle modifications led to clinical improvement and glycemic stabilization. This case underscores the potential for BD to progress to necrosis in poorly controlled diabetes and highlights the importance of early diagnosis and comprehensive management.

Keywords: Bullosis diabeticorum, diabetic foot, bullous lesions, necrosis, diabetes mellitus

ДИАБЕТИЧЕСКИЙ БУЛЛЕЗ, ОСЛОЖНЕННЫЙ ВТОРИЧНОЙ ИНФЕКЦИЕЙ И НЕКРОЗОМ, У 29-ЛЕТНЕГО МУЖЧИНЫ С ВПЕРВЫЕ ДИАГНОСТИРОВАННЫМ САХАРНЫМ ДИАБЕТОМ: КЛИНИЧЕСКИЙ СЛУЧАЙ

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Аннотация
Диабетический буллез (ДБ) — редкое кожное проявление сахарного диабета, характеризующееся спонтанными буллезными высыпаниями, преимущественно на конечностях. В данном сообщении описывается 29-летний водитель автомобиля из Дубая, у которого был зуд на правой стопе, прогрессирующий до образования везикул, папул, пустул, булл и, в конечном итоге, некроза, несмотря на многочисленные вмешательства. Состояние осложнилось вторичной инфекцией и отёком. Лабораторные данные выявили лёгкий лейкоцитоз, эозинофилию и повышенный уровень HbA1c, что подтвердило впервые выявленный сахарный диабет. Многопрофильное лечение, включающее антимикробную терапию, противогрибковые препараты, уход за раной и изменение образа жизни, привело к клиническому улучшению и стабилизации гликемии. Данный случай подчёркивает вероятность прогрессирования БД до некроза при плохо контролируемом диабете и подчеркивает важность ранней диагностики и комплексного лечения.

Ключевые слова: Bullosis diabeticorum, диабетическая стопа, буллезные поражения, некроз, сахарный диабет

Introduction

Bullosis diabeticorum (BD), or diabetic bullae, is an uncommon dermatologic complication of diabetes mellitus, manifesting as tense, fluid-filled blisters, primarily on the lower extremities [1, 2]. Although its etiology is poorly understood, associations with poor glycemic control, neuropathy, and microangiopathy have been proposed [3]. Typically self-limiting, BD can become complicated by secondary bacterial or fungal infections, leading to ulceration, necrosis, and significant morbidity [4]. Risk factors include longstanding diabetes, male gender, and peripheral neuropathy, with lesions often arising spontaneously or following minor trauma. Histologically, BD shows intraepidermal or subepidermal cleavage without significant inflammation, distinguishing it from autoimmune bullous disorders. This case report details a rare presentation of BD in a young male with newly diagnosed diabetes, complicated by secondary infection and necrosis, emphasizing the critical role of prompt diagnosis, glycemic optimization, and integrated care to prevent severe outcomes such as amputation.

Case Presentation

A 29-year-old male car driver from Dubai, with an 8-hour daily work schedule involving prolonged sitting and potential exposure to heat and friction, presented with a one-week history of pruritus on the dorsal right foot, rapidly progressing to vesicles and papules (Figure 1). Vital signs were unremarkable (blood pressure 120/80 mmHg, heart rate 78 bpm, temperature 36.8°C), and the patient reported no systemic symptoms such as fever, malaise, or weight loss. He had no prior history of diabetes, skin disorders, allergies, or immunosuppressive conditions, and denied recent trauma, insect bites, or exposure to irritants.

Initial management with topical aciclovir 5% cream (suspecting a viral etiology like herpes simplex) and oral ebastine (10 mg daily for antihistaminic relief) provided no improvement after 5 days. The lesions spread distally, forming intertarsal pustules (Figure 2), prompting hospital evaluation. Pustule drainage revealed seropurulent fluid, and sterile bandaging was applied. However, new lesions emerged on the plantar surface within a week, accompanied by diffuse erythema, edema, and warmth suggestive of cellulitis (Figures 3, 4). The patient reported increasing pain (VAS score 6/10) and difficulty bearing weight.

Oral ampicillin-cloxacillin (500 mg twice daily for 7 days) was initiated under medical supervision, but symptoms worsened with progressive swelling, erythema, and induration. Subsequent treatment included itraconazole (100 mg daily for 7 days, suspecting fungal superinfection), serratiopeptidase (10 mg thrice daily for anti-inflammatory and fibrinolytic effects), and fusidic acid cream applied topically twice daily (Table 5). Two tense, fluid-filled bullae (approximately 2-3 cm in diameter) developed on the lateral foot margin (Figure 7), requiring aseptic drainage and daily wound care with saline irrigation. Despite these interventions, the plantar surface became necrotic and eschar-formed within two days (Figure 6), followed by dorsal necrosis the next day. Sensory examination revealed intact pressure and fine touch sensation, with no evidence of peripheral neuropathy on monofilament testing.

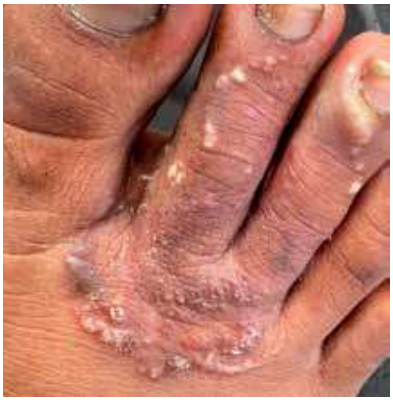
Treatment was escalated to mupirocin 2% ointment (applied twice daily for broad-spectrum bacterial coverage), terbinafine (250 mg daily for antifungal therapy), levofloxacin (500 mg daily for gram-negative coverage), and metronidazole (500 mg thrice daily for anaerobic organisms). Initial healing was noted with reduced exudate and eschar sloughing over several

days, but three days later, multiple dorsal bullae recurred (Figure 8), necessitating repeated drainage and debridement. Clinical examination at this stage revealed hyperpigmented, necrotic skin on the toes and dorsum with bullous lesions, crusting, malodorous exudate, and surrounding maceration (Figures 1–10). No signs of deep tissue involvement, such as crepitus or probing to bone, were observed, and radiographs ruled out osteomyelitis.

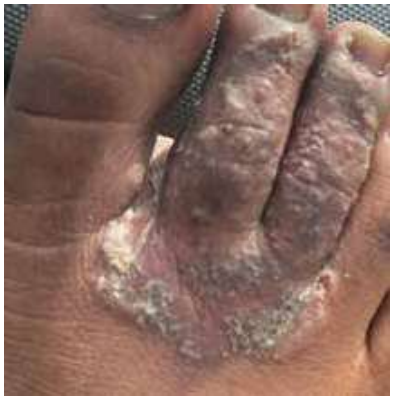
Laboratory findings on admission included mild leukocytosis (WBC 11.0 × 10^3/μL, reference 4–10), neutrophilia (64.31%), eosinophilia (8.63%, reference 1–6), lymphopenia (19.11%, reference 20–40), and elevated red cell distribution width (RDW-CV 14.3%, reference 11.6–14.0), suggesting inflammatory response and possible nutritional deficiency. Repeat hematology confirmed persistent leukocytosis (WBC 10.60 × 10^3/μL) and neutrophilia (71%). Pus culture yielded no bacterial growth, possibly due to prior antibiotic use. Random blood glucose was within normal limits (116 mg/dL), but HbA1c was elevated at 6.00% (reference <5.7%), confirming pre-diabetes transitioning to overt diabetes (Tables 11–14).

A comprehensive wellness panel four weeks post-infection revealed borderline dyslipidemia (HDL cholesterol 39.50 mg/dL, reference 40–60, indicating increased cardiovascular risk; LDL cholesterol 132.00 mg/dL, reference <100 optimal, suggesting early atherogenesis), vitamin D deficiency (25-OH vitamin D 10.93 ng/mL, reference <20, potentially impairing immune function and wound healing), and mild iron deficiency (serum iron 63.00 μg/dL, reference 65–175, linked to chronic inflammation). Persistent leukocytosis (WBC 11.70 × 10^3/μL) and eosinophilia (9.10%, absolute 1.10 × 10^3/μL, reference 0.02–0.5) indicated ongoing subclinical inflammation, possibly exacerbated by medications or occupational exposures such as prolonged driving in a hot climate (Tables 15–17). Thyroid function, renal profile, and hormone assays were unremarkable.

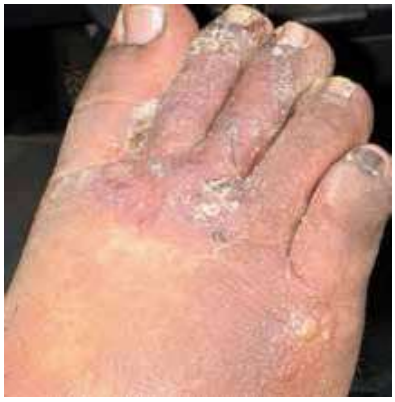
Following diabetes diagnosis, lifestyle interventions were implemented, including a low-glycemic diet (emphasizing whole grains, vegetables, and lean proteins), moderate exercise (30 minutes of walking daily, adjusted for foot healing), vitamin D and iron supplementation, and metformin initiation (500 mg twice daily). These measures normalized glucose (average 110 mg/dL) and lipid profiles within two months, correlating with lesion improvement. Wound care continued with regular debridement, hydrocolloid dressings, and offloading using orthopedic footwear, leading to gradual epithelialization without scarring or amputation (Figure 18).



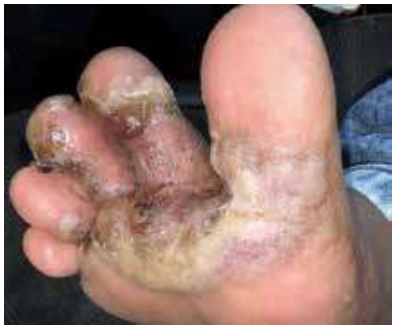
• *Figure 1: Initial dorsal foot with vesicles, papules and early bullae showing erythema and swelling*



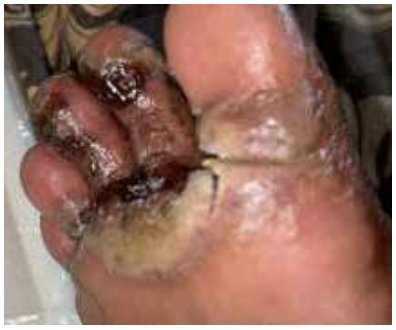
• *Figure 2: Spreading of infection in toes*



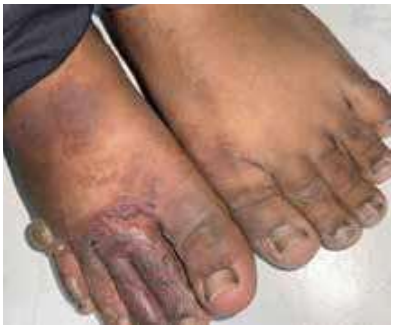
• *Figure 3: Swelling and redness after cleaning the wound in second week of infection look like cellulitis*



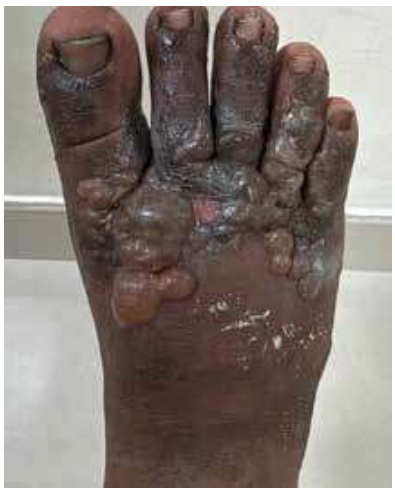
• *Figure 4 : Extension of infection to planter surface*



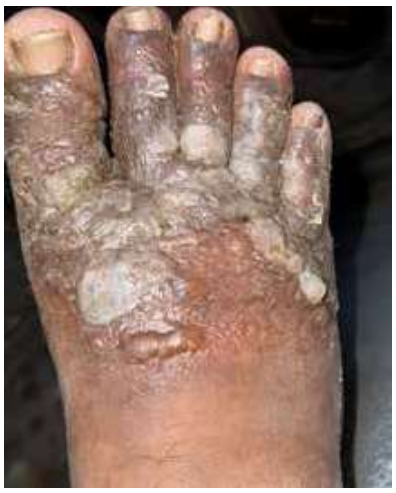
• *Figure 6: Foot with crusting and healing lesions*



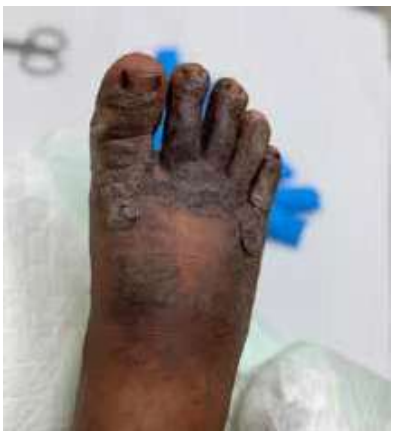
• *Figure 7: Comparing the healthy and infected foot after recurrence of bullae after 3 days of cleaning*



• *Figure 8 : progression of bullous formation after debridement*



• *Figure 9: Dorsal foot in later stage with residual necrosis and bullae After drainage*



• *Figure 10: Result after cleaning and bandages with saline solution, povidone-iodine , hydrogen peroxide and triple antibiotic ointment*

• *Table 5: Prescription started after two week of infection*

Medicine Name	*Strength*	*Dosage*	*Frequency*	*Duration*	*Qty*	*Remarks*
Itrazol 100Mg 4 Tab	100 Mg	1	Every 12 Hours	7D	14	Use 1 Capsules Every 12 Hours For A Duration Of 7 Days. After Food
Amoclan Forte 625Mg Tab 15S	125 Mg/ 500 Mg	1	Every 12 Hours	3D	6	Use 1 Tablets Every 12 Hours For A Duration Of 3 Days. After Food
Fucidin Cream 30Gm	2%	1	Every 12 Hours	7D	1	Use 1 Gm Every 12 Hours For A Duration Of 7 Days

• *Table 11: Hematology report leukocytosis & eosinophilia*

Test Name	*Result*	*Units*	*Ref. Range*	*Method*
WBC Count	11.03*	10^3/µL	4-10	El. Impedance
Neutrophils (%)	64.31 L	%	40-80	El. Impedance
Lymphocytes (%)	19.11 L	%	20-40	El. Impedance
Monocytes (%)	7.80	%	2-10	El. Impedance
Eosinophils (%)	8.63 H	%	1-6	El. Impedance
Basophils (%)	0.15	%	0-1	El. Impedance
RBC Count	5.38	10^6/µL	4.5-5.9	El. Impedance
HEMOGLOBIN (Hb)	16.31	g/dL	13-17	El. Impedance
HEMATOCRIT (HCT/PCV)	48.7	%	40-50	El. Impedance
MCV (Mean Cell Volume)	90.6	fL	78-100	Calculation
MCH (Mean Cell Hemoglobin)	29.9	pg	27-32	Calculation
MCHC (Mean Cell Hemoglobin Conc)	29.9	g/dL	31.5-34.5	Calculation
RDW CV	13.1	%	11.6-14.0	El. Impedance
RDW SD	45.5	fL	36.5-46.0	El. Impedance
Platelet Count	354.9	10^3/µL	150-400	El. Impedance

• *Table 12: Additional hematology confirming infection markers*

Test Name	*Result*	*Ref. Range*	*Units*	*Method*
RBC (CBC sample)	4.80	4.5-5.9	10^6/µL	Hydrodynamically focused DC
Haemoglobin	14.30	13.5-18	g/dL	RBC pulse height
Hematocrit	43.30	40-58	%	Cell count computation
MCV	90.3*	80-101	fL	Cell count computation
MCH	29.6	27-35	pg	Cell count computation
Red Cell Distribution Width	10.4 H	11-16	%	Cytometry Flow
Total WBC Count	10.4 H	3.4-10.4	10^3/µL	Cytometry Flow
Neutrophils (%)	71.60	40-75	%	Fluorescence Flow
Lymphocytes (%)	18.6 L	20-40	%	Fluorescence Flow
Eosinophils (%)	4.7	1-6	%	Cytometry Flow
Monocytes (%)	3.7	2-10	%	Cytometry Flow
Basophils (%)	0.3	0-2	%	Cytometry Flow
Absolute Neutrophil Count	7.53 H	2-7	10^3/µL	Cytometry Flow
Absolute Lymphocyte Count	1.76	1-3	10^3/µL	Fluorescence Flow
Absolute Eosinophil Count	0.50	0.02-0.5	10^3/µL	Cytometry Flow

• *Table 13: Report of specimen culture of pus*

Section	*Detail*	*Result*
Investigation	MICROBIOLOGY	-
Specimen	Pus	-
Culture	Pus	No Pathogen Grown after 36 hours of Aerobic Incubation
Note	-	*** Kindly correlate with Clinical History

• *Table 14: Biochemistry report with normal random blood*

Test Name	*Result*	*Biological Interval Reference*	*Units*	*Specimen*	*Test Method*
Random Blood Sugar	116	Diabetic: >200 mg/dL (ADA Guidelines)	mg/dL	Fluoride Plasma	Enzymatic Hexokinase

Notes: Factors such as type and time of food intake, infection, physical or psychological stress, exercise and drugs can influence the blood glucose level.

• *Table 15: Hematology report after 4 weeks of infection*

Test Name	*Value*	*Units*	*Bio. Ref. Range*	*Methodology*
*Total Leucocytes Count (Wbc)	11.70	$X10^3$/Ml	4.0-11.0	Coulter Principle
*Hemoglobin	16.20	G/Dl	13-17	Photometric Measurement
*Platelet Count	372.00	$X10^3$/Ml	150-410	Coulter Principle
*Total Rbc	5.38	$X10^6$/Ml	4.5-5.5	Coulter Principle
*Hematocrit (Pcv)	48.70	%	40-50	Calculated Rbc
Mean Corpuscular Volume (Mcv)	90.60	Fl	78-101	Derived Rbc Histogram
Mean Corpuscular Hemoglobin (Mch)	30.20	Pg	27-32	Calculated
Mean Corpuscular Hemoglobin Conc (Mchc)	33.40	G/Dl	31.5-34.5	Calculated
Red Cell Distribution Width - Rdw-Sd	43.80	Fl	37.1-48.3	Derived Rbc Histogram
Rdw-Cv Distribution Width	13.70	%	11.6-14	Derived Rbc Histogram
(Rdw-Cv) Neutrophils	58.10	%	40-80	Histogram/Impedance
*Lymphocyte Percentage	23.40	%	20-40	Optical/Impedance
*Monocytes	8.90	%	2-10	Optical/Impedance
*Eosinophils	9.10	%	1-6	Optical/Impedance
*Basophils	0.50	%	<1-2	Optical/Impedance
*Neutrophils - Absolute Count	6.80	$X10^3$/Ml	2.0-7.0	Calculated
*Lymphocytes - Absolute Count	2.70	$X10^3$/Ml	1.0-3.0	Calculated
Monocytes - Absolute Count	1.00	$X10^3$/Ml	0.2-1.0	Calculated
*Eosinophils - Absolute Count	1.10	$X10^3$/Ml	0.02-0.5	Calculated
*Basophils - Absolute Count	0.10	$X10^3$/Ml	0.02-0.1	Calculated
*Mean Platelet Volume (Mpv)	8.80	Fl	7.5-11.2	Derived Plt Histogram

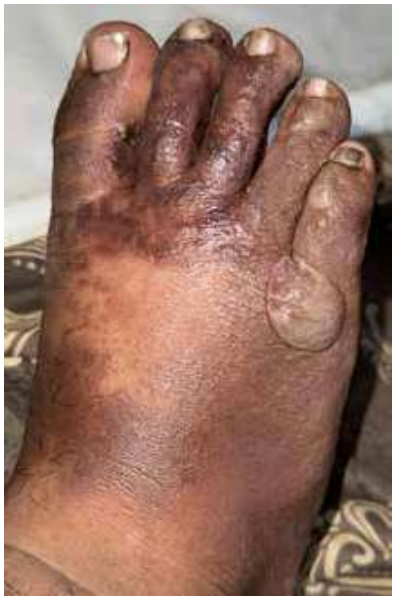
• *Table 16: Lipid Profile, Liver function test after 4 weeks of infection*

Test Name	*Value*	*Units*	*Bio. Ref. Range*	*Methodology*
LIPID PROFILE				
*Total Cholesterol	186.00	mg/dL	Desirable <200 mg/dL, Borderline high 200-239 mg/dL, High ⩾240 mg/dL	Enzymatic Assay
*HDL Cholesterol - Direct	39.50	mg/dL	40-60 mg/dL, High >60 mg/dL	Elimination/ Catalase
*LDL Cholesterol - Direct	132.00	mg/dL	Optimal <100, Near optimal 100-129, Borderline high 130-159, High ⩾160-189, Very high ⩾190	Enzymatic/ Colorimetric Method
*Triglycerides	133.00	mg/dL	<150 Normal, 150-199 Borderline high, 200-499 High, ⩾500 Very high	Enzymatic Assay
VLDL Cholesterol	26.60	mg/dL	2-30	Calculated
Non-HDL Cholesterol	146.50	mg/dL	<160, 160-189	Calculated
TC/HDL Cholesterol Ratio	4.71	Ratio	3.5-5.0	Calculated
LIVER FUNCTION TEST				
LDL/HDL Ratio	3.34	Ratio	<3.5	Calculated
*Bilirubin Total Test	0.39	mg/dL	0.3-1.2	Vanadate Oxidation
*Bilirubin - Direct	0.14	mg/dL	⩽0.3	Vanadate Oxidation
Bilirubin (Indirect)	0.25	mg/dL	0-0.9	Calculated
*Aspartate Aminotransferase (SGOT)	22.00	U/L	<34	IFCC (without pyridoxal phosphate)
*Alanine Transaminase (SGPT)	37.00	U/L	10-49	IFCC (without pyridoxal phosphate)
*Alkaline Phosphatase	97.00	U/L	46-116	IFCC Standardization
*Gamma Glutamyl Transferase (GGT)	52.00	U/L	<73	Modified IFCC Method
*Protein - Total	7.48	g/dL	5.7-8.2	Biuret Method
*Albumin - Serum	4.47	g/dL	3.2-4.8	Dye Binding: Bromocresol Green
Serum Globulin	3.01	g/dL	2.2-4.0	Calculated
Serum ALB/Globulin Ratio	1.49	Ratio	>1	Calculated

• *Table 17: Renal function test, Diabetic Profile, Thyroid Function test, Hormone Assay, Vitamin and Iron Profile after 4 weeks of infection*

Test Name	*Value*	*Units*	*Bio. Ref. Range*	*Methodology*
Renal Function Test				
*Blood Urea Nitrogen (Bun)	11.57	Mg/Dl	9-23	Gldh Kinetic Assay
Bun/S.creatinine Ratio	14.65	Ratio	9:1-23:1	Modified Jaffe Kinetic Calculated
Est. Glomerular Filtration Rate (Egfr)	121.00	Ml/ Min/1.73M²	>90	Photometry
*Uric Acid	5.74	Mg/Dl	3.7-9.2	Uricase/Peroxidase
*Calcium	8.90	Mg/Dl	8.3-10.6	Enzymatic Colorimetric Method
Diabetic Profile				
*Hba1c	6.00	%	Normal <5.7, Pre Diabetes 5.7-6.4, Diabetes >6.5	H.p.l.c
Average Blood Glucose (Abg)	125.50	Mg/Dl	90-120	Calculated
Thyroid Function Test				
*Thyroid Stimulating Hormone (Tsh)	1.93	Miu/Ml	Adult 0.55-4.78, 1St Trimester 0.48-2.50, 2Nd 0.20-3.00, 3Rd 0.20-3.0, Newborn >20	Two Site Sandwich Immunoassay
*Free Thyroxine (Ft4)	1.40	Ng/Dl	0.89-1.76	Clia
*Free Triiodothyronine (Ft3)	3.21	Pg/Ml	2.3-4.2	Clia
Hormone Assay				
*Testosterone	479.70	Ng/Dl	260-1000	Clia
Vitamin				
*25-Oh Vitamin D (Total)	10.93	Ng/Ml	Deficiency <20 Ng/Ml, Insufficiency 20-30 Ng/Ml, Sufficiency 30-100 Ng/Ml	Clia
*Vitamin B-12	354.00	Pg/Ml	211-911	Clia
Iron Profile				
*Iron	63.00	Mg/Dl	65-175	Ferrozine Sequential Release & Uptake Of Iron
*Total Iron Binding Capacity (Tibc)	281.00	Mg/Dl	240-450	Immunoturbidimetry
% Transferrin Saturation	22.42	%	16-50%	Calculated

Discussion



• *Figure 18: Image after cleaning, drainage of reoccurred bullous and recovery phase*

This case presents a rare and severe manifestation of bullosis diabeticorum (BD) in a 29-year-old male with newly diagnosed diabetes mellitus, characterized by rapid progression from pruritus to bullae, secondary infection, and necrosis [5]. Unlike typical BD, which manifests as painless blisters in patients with longstanding diabetes, this case was exacerbated by occupational factors, including prolonged sitting and potential heat or friction exposure as a car driver, alongside undiagnosed hyperglycemia, which likely intensified microvascular and immune dysfunction [1]. The initial eosinophilia (8.63%) suggests a possible allergic or environmental trigger, such as footwear irritation or heat exposure in Dubai's climate, though parasitic infection was not confirmed via stool analysis or serology. Differential diagnoses included bullous impetigo, excluded by negative pus culture; necrotizing fasciitis, ruled out due to the absence of systemic toxicity or crepitus; pyoderma gangrenosum [6]; and drug-induced bullous pemphigoid [7]. The strong temporal association with new-onset diabetes and the response to broad-spectrum antimicrobials and glycemic control confirmed BD with superinfection. Necrosis, a rare complication of BD, highlights the risks of uncontrolled diabetes, where impaired immunity and tissue perfusion can lead to tissue death, consistent with prior reports requiring surgical intervention [8].

In 2009, Lopez et al. reported a 54-year-old male with type 2 diabetes and neuropathy who developed painless bullae on the lower legs and feet without clear triggers. Managed conservatively with aspiration and topical antiseptics, the lesions resolved within three weeks without infection or necrosis [10]. In contrast, our patient's case was complicated by rapid infectious progression and necrosis, likely due to undiagnosed diabetes and occupational stressors, highlighting the role of early glycemic control in preventing complications. In 2012, Bello et al. described two cases of BD triggered by long-distance bus journeys in patients with poorly controlled type 2 diabetes. The first, a 59-year-old male, developed bilateral foot bullae with secondary staphylococcal infection, resolving in four weeks with antibiotics and glycemic management. The second, a 47-year-old female, progressed to purulent discharge and dry gangrene, necessitating toe disarticulation [11]. Similar to our case, necrosis occurred, but our patient avoided amputation through aggressive antimicrobial therapy and timely diabetes diagnosis, suggesting that early intervention can mitigate severe outcomes.

In 2013, Zhang et al. documented a 56-year-old male with longstanding type 2 diabetes and neuropathy presenting with haemoserous plantar blisters. Conservative management with aspiration, antiseptic washes, and pressure offloading led to resolution in 3–6 weeks without complications [12]. Unlike our case, the absence of infection or necrosis may reflect established diabetes management and lack of weight-bearing trauma, underscoring the impact of undiagnosed diabetes in our patient's severe presentation. In 2014, Gupta et al. reported a 27-year-old male with uncontrolled type 1 diabetes developing painless elbow blisters following minor trauma (sleeping on a hard surface). Biopsy-confirmed BD resolved in four weeks with hydrotherapy and elbow protection, without infection or necrosis [13]. The milder course and upper extremity involvement contrast with our case's lower extremity

severity, likely exacerbated by weight-bearing stress and occupational factors. These comparisons demonstrate that BD's clinical course varies with glycemic control, anatomical site, and external triggers. Our patient's young age, undiagnosed diabetes, and occupational exposures (prolonged sitting, heat, and friction) likely amplified the risk of infection and necrosis, distinguishing this case from milder presentations [10,12,13]. The progression to necrosis aligns with the severe case reported by Bello et al. [11], though our patient's favorable outcome, avoiding amputation, underscores the efficacy of escalated antimicrobial therapy and early glycemic intervention. Initial treatment resistance necessitated broad-spectrum antimicrobials targeting polymicrobial infection, including anaerobes, reflecting the complexity of superinfected BD. Early HbA1c screening was pivotal, as glycemic optimization facilitated healing [3]. Addressing comorbidities, such as dyslipidemia, vitamin D deficiency, and iron insufficiency, through lifestyle modifications and supplementation further supported recovery and reduced recurrence risk. This case advocates for a multidisciplinary approach, integrating dermatology, endocrinology, infectious disease expertise, and podiatry, potentially incorporating advanced therapies like negative pressure wound therapy for refractory cases.

Conclusion

Bullosis diabeticorum can manifest aggressively in young patients with undiagnosed diabetes, progressing to necrosis when complicated by secondary infections. Early diagnosis, optimized glycemic control, and targeted antimicrobial therapy are critical to mitigating morbidity. This case underscores the importance of screening for diabetes in patients presenting with unexplained bullous foot lesions and highlights occupational factors as potential precipitants.

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