underwent endoscopic transsphenoidal surgery 7 years later, which was complicated by panhypopituitarism and cranial diabetes insipidus. Postoperatively, CD was cured, with the resolution of her metabolic comorbidities.

CONCLUSION

Despite the increasing prevalence of T2D in adolescents, clinicians must recognize the diagnostic challenge of CD in this age group. Atypical manifestations in a presumed T2D patient should prompt consideration of Cushing's syndrome.

EP_A058

INSULIN-RECALCITRANT HYPERGLYCEMIA FOLLOWING CAPIVASERTIB THERAPY: A NOVEL CHALLENGE IN A NON-DIABETIC PATIENT

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INTRODUCTION

Capivasertib is a selective oral AKT inhibitor approved for hormone receptor-positive, HER2-negative advanced breast cancer with AKT pathway mutations. As AKT is integral to insulin signaling, its inhibition predisposes patients to hyperglycemia. We report a case of severe capivasertib-induced hyperglycemia with profound insulin resistance in a non-diabetic patient.

CASE

A 56-year-old female with metastatic breast cancer (ER/PR-positive, HER2-negative, AKT1- and ESR1-mutated, TMB-high) presented with severe hyperglycemia following capivasertib initiation. She had no prior diabetes history, with a baseline hemoglobin A1c of 5.6%. Her first cycle (200 mg twice daily, 7 April 2025) was uncomplicated metabolically. The second cycle was escalated to 400 mg twice daily on 21 April 2025. By Day 3, blood glucose rose significantly. Premixed insulin 20 units failed to achieve glycemic control, necessitating intravenous insulin infusion. Despite escalation to 30 units per hour, euglycemia could not be achieved. Notably, there was no biochemical evidence of diabetic ketoacidosis or hyperosmolar hyperglycemic state, and the patient remained hemodynamically stable. No corticosteroids or other contributing medications were administered. Blood glucose normalized spontaneously approximately 36 hours after the last capivasertib dose, and all insulin was weaned and discontinued by 26 April 2025. She remained euglycemic without antidiabetic therapy thereafter. The patient and family opted for palliative care, and she passed away on 6 May 2025.

CONCLUSION

Capivasertib can precipitate severe, insulin-resistant hyperglycemia even in non-diabetic patients, without progression to overt hyperglycemic crisis. Spontaneous resolution upon drug cessation supports a direct drug-mediated mechanism via AKT-disrupted insulin signaling. Clinicians should monitor glucose closely during therapy and consider drug cessation in refractory cases. Further studies are warranted to guide optimal glycemic management.

EP_A059

THE VARIABLE NATURE OF BIOPSY-PROVEN VILDAGLIPTIN-INDUCED BULLOUS PEMPHIGOID IN AN ELDERLY PATIENT: A CASE SERIES

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INTRODUCTION

Bullous pemphigoid (BP) is the most common autoimmune blistering disease. Drug-induced BP has been increasingly reported, with dipeptidyl peptidase-4 (DPP-4) inhibitors, particularly vildagliptin, emerging as a notable cause. The pathogenesis is thought to involve the disruption of immune tolerance and epitope spreading, leading to a broader autoimmune response against basement membrane antigens beyond the classic NC16A domain of BP180. This case series highlights the variable clinical spectrum and therapeutic challenges in vildagliptin-induced BP.

CASES

A 74-year-old male with type 2 diabetes mellitus (T2DM), hypertension, dyslipidemia, and a history of stroke presented with a 2-month history of recurrent, pruritic, tense bullae. He had been on vildagliptin for 36 months. Histopathology confirmed subepidermal blistering, and direct immunofluorescence demonstrated linear IgG/C3 deposition at the dermo-epidermal junction, with positive anti-BP180 antibodies, diagnosing BP. Discontinuation of vildagliptin and initiation of systemic corticosteroids resulted in significant clinical improvement, allowing a rapid prednisolone taper to 5 mg daily without new bullae formation.

A 62-year-old female with T2DM and dyslipidemia presented with bullous eruptions 3 months after initiating vildagliptin. Skin biopsy confirmed BP, revealing subepidermal blistering with eosinophils and linear C3/IgG deposition at the basement membrane zone. Vildagliptin

was discontinued. Despite initial management with systemic corticosteroids, the disease course was refractory. The patient experienced a flare upon steroid taper and had persistent blistering on prednisolone 20 mg daily, necessitating the addition of azathioprine as a steroid-sparing agent.

CONCLUSION

This case series illustrates the variable clinical course of vildagliptin-induced BP in which the therapeutic trajectories diverged significantly. Recognition of BP as a potential adverse effect of DPP-4 inhibitors is critical, and management should be individualized.

EP_A060

FLUCONAZOLE-INDUCED REFRACTORY HYPOGLYCEMIA IN GLICLAZIDE THERAPY: A PREVENTABLE INTERACTION

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INTRODUCTION

Clinically important drug–drug interactions are a frequently overlooked cause of severe hypoglycemia in patients with type 2 diabetes mellitus (T2DM). Gliclazide is primarily metabolized via cytochrome P450 2C9 (CYP2C9), while fluconazole is a potent inhibitor of this enzyme.1,2 Concomitant administrations markedly increase gliclazide plasma concentration and prolong its hypoglycemic effect, substantially increasing the risk of severe and prolonged hypoglycemia. In some cases of refractory sulfonylurea-induced hypoglycemia, octreotide may need to be considered early.

CASE

A 63-year-old Malay female with a 15-year history of T2DM on maximum-dose gliclazide (320 mg/day) presented with syncope and recurrent severe hypoglycemia 5 days after receiving a single oral dose of fluconazole 150 mg for a superficial fungal skin infection. Her other anti-diabetic medications included metformin 1 g twice daily and vildagliptin 50 mg once daily. Her blood pressure on presentation was 151/74 mmHg, and her glucose level was 2.8 mmol/L. Her presentation was compounded by community-acquired pneumonia and acute kidney injury (estimated glomerular filtration rate 52 mL/min/1.73 m^2). Biochemical evaluation excluded adrenal insufficiency, with normal serum cortisol (1,098 µg/dL), serum sodium (141 mmol/L), and serum potassium (4.6 mmol/L). Thyroid and liver function tests were normal. Despite repeated boluses of 50% dextrose and continuous infusion of 20% dextrose, recurrent hypoglycemia persisted for up to 48 hours after discontinuation of gliclazide, with glucose ranging from 1.7 to 5.1 mmol/L, reflecting prolonged sulfonylurea activity.

CONCLUSION

This case highlights a clinically significant yet preventable interaction between fluconazole and gliclazide, mediated through CYP2C9 inhibition, resulting in sustained hyper-insulinemic hypoglycemia. Gliclazide should be reduced or withheld prior to initiating fluconazole, and medication review should be performed at every consultation.

EP_A061

THE HIDDEN RISK OF A FIRST-LINE THERAPY: RENAL ABSCESS WITH SGLT2 INHIBITOR USE

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INTRODUCTION

Sodium-glucose cotransporter-2 (SGLT2) inhibitors are cornerstone therapies for heart failure and diabetes, offering proven cardiorenal benefits. However, expanded use necessitates vigilance regarding adverse effects, particularly genitourinary infections. While mild cystitis is common, serious upper urinary tract infections remain rare and potentially life-threatening. We describe a case of renal abscess presenting as recurrent urinary tract infections (UTI) following SGLT2 inhibitor initiation, emphasizing the need for clinical vigilance.

CASE

A 69-year-old male with a significant cardiovascular history, including heart failure with reduced ejection fraction (HFrEF), hypertrophic cardiomyopathy with an implantable cardioverter-defibrillator for non-sustained ventricular tachycardia, diabetes mellitus, hypertension, and hyperlipidemia, presented with a 1-week history of right flank pain, dysuria, urinary frequency, and fever. Initial labs confirmed infection: leukocytosis (22.6 × 10^3/ µL), markedly elevated CRP (200 mg/L), and bacteriuria. He was diagnosed with a UTI and started on IV Cefuroxime. This marked his third UTI admission in 5 months, following discharge just 3 weeks prior for septic shock secondary to UTI, establishing a relapsing pattern. Subsequent urine and blood cultures were unremarkable. Medication review revealed Dapagliflozin had been initiated for HFrEF 8 months ago. Following clinical improvement from