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