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ERRATUM

In the article entitled "Prolonged ketosis and glycosuria secondary to SGLT2 inhibitor therapy" which was previously published in Volume 9 Issue 11 of clinical case reports, the abstract was wrongly published as the first paragraph in the introduction section. The correct abstract and introduction should be as follows.

We sincerely apologize for this error.

Abstract

Current guidelines do not recognize the potential for the effects of sodium-glucose cotransporter 2 (SGLT2) inhibitors to persist beyond five half-lives of elimination. The objective of this report is to describe a case of prolonged SGLT2 inhibitor effects and to provide a review of similar cases and outline possible explanatory mechanisms.

Introduction

Sodium-glucose cotransporter 2 (SGLT2) inhibitors, such as canagliflozin, reduce the reabsorption of filtered glucose at the proximal renal tubules and lower the renal threshold for glucose excretion.[1] This inhibition promotes glycosuria, resulting in decreased plasma glucose and increased risks of volume depletion and mycotic urinary tract infections.[2] SGLT2 inhibitors may also directly act on pancreatic α-cells to increase plasma glucagon levels.[1] These metabolic changes produce a lower insulin-to-glucagon ratio, which can predispose patients to ketoacidosis while maintaining normoglycemia.[1] The objective of this report is to describe a case of SGLT2 inhibitor-associated euglycemic diabetic ketoacidosis (DKA) with prolonged ketosis and glycosuria and discuss the potential for SGLT2 inhibitor effects to persist well beyond five half-lives of drug elimination.

REFERENCE

1. Bobrowski D, Kumar R, Wu PE, Lapointe-Shaw L. Prolonged ketosis and glycosuria secondary to SGLT2 inhibitor therapy. *Clin Case Rep*. 2021;9:e05057. doi:10.1002/ccr3.5057