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Sofosbuvir Causing Diabetes Mellitus: Is there a Link?

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Sir,

Chronic hepatitis C infection affects 170 million individuals worldwide. Since the introduction of new oral direct acting antiviral (DAA) drugs, the treatment of chronic hepatitis C infection has been revolutionised. Although metabolic derangements along with insulin resistance are widely recognised with HCV infection, there are few case reports of how treatment with the newer oral antiviral agents leads to diabetes in previously non-diabetic chronic hepatitis C infected patients, after successful eradication of the infection itself.

Here, we describe a case of a 50-year female with a body mass index (BMI) of 32.8 kg/m$^2$ with chronic hepatitis C virus infection, mixed genotype 3 and 6, diagnosed and started on three drug regimens with sofosbuvir, 400 mg QD, Daclatasvir Dihydrochloride, 60 mg QD, and Ribavarin 400 mg TID. She achieved end-of-treatment response (ETR) at 6 months but thereafter presented with history of progressive leg pains. On testing, her random blood sugar was 494 mg/dl with an anion gap of 12 and no urinary ketones. Prior to starting antiviral agents, her HbA1c was 5.50% and three months post-treatment, her HbA1c was 11.40%. Her HbA1c pre- and post-treatment were done by our laboratory by immunoturbidty method and data from this laboratory is traceable to NGSP and is DCCT certified.

There was no history of gestational diabetes mellitus (DM) or pre-diabetes, except that her father was a known diabetic. Other etiologies for her raised blood sugars were excluded, including exposure to certain drugs, pancreatitis or any ongoing acute infection.

In recent past, there has accumulated conflicting data on effects of oral DAAs with few studies favouring good glycemic control following treatment; whereas, others showing no effects on HbA1c levels pre- and post-treatment. Our patient did not have any comorbidities or a pre-diabetic range HbA1c level prior to treatment with DAAs and it was only after completion of treatment and achieving ETR that there was a sudden rise in HbA1c levels. Therefore, we could not completely rule out the possibility that the DAAs might have led to overt hyperglycemia in our patient or this could simply be a coincidence. It is not possible to pinpoint the precipitating factor on the basis of just one case presented here. However, it provides food for thought for future studies on this subject.

REFERENCES


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