Dear Editor,

Chronic pancreatitis is a condition characterized by irreversible destruction and fibrosis of the pancreatic parenchyma, leading to exocrine insufficiency and progressive endocrine failure leading to diabetes. Fibrocalculous Pancreatic Diabetes (FCPD) refers to the diabetes secondary to fibrocalculous pancreatitis.[1] Due to chronic exocrine insufficiency, there is diminished entry of nutrients due to malabsorption which can eventually lead to absent or blunted incretin response to meals and reduce postprandial insulin secretion.[2] However, the pancreatic enzyme replacement therapy (PERT) with mixed meals to evaluate changes in glycemic state have yielded variable results.[3]

The objective documentation of insufficiency of the exocrine pancreas is difficult and controversial. Examination of stool collected over 72 h following the intake of 100 g of oral fat daily is gold standard but the procedure is cumbersome, time-consuming, and may be unacceptable with duration of diabetes (\( r = -0.582, P < 0.002 \)) and type 1 diabetes was 37.9% (severe deficiency in 16.7%). As compared to subjects with FCPD (with 100% deficiency), the FEC deficiency with significantly lower in type 2 (37.5%, \( P < 0.001 \)) and type 1 diabetics (37.9%, \( P < 0.001 \)). In FCPD group there was no significant correlation between fecal elastase with HbA1c, duration of diabetes. On the other hand, in subjects with type 2 diabetes fecal elastase was negatively correlated with duration of diabetes (\( r = -0.519, P = 0.002 \)) and HbA1c (\( r = -0.519, P = 0.009 \)).

The deficiency of fecal elastase in FCPD is not surprising. Exocrine insufficiency in FCPD is already manifest by the time the patients present with diabetes. The documentation of exocrine insufficiency may also indicate that PERT may have some therapeutic effect in better glycemic management of FCPD subjects. In subjects with T2DM, fecal elastase was negatively correlated with duration of the disease and HbA1c.[4] This is also in line of the hypothesis that suggests a significant endocrine-exocrine association of the pancreas in the pathogenesis of T2DM. Exocrine pancreatic deficiency is already documented in Type 1 DM.[5]

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