FCPD may present without diarrhea, steatorrhea or ketonuria. It also shows that serum amylase, a marker of pancreatic inflammation, may be normal. The latter suggests chronic fibrotic ‘burnt-out’ pancreatic disease. Several possible mechanisms of causation have been suggested to explain development of TCP/FCPD; they include initial stasis due to prolonged lack of proteinaceous foods; and blockage of the pancreatic ducts by laminated secretions or inspissated mucus plugs, which enlarge with repeated infection and ultimately calcify [Figures 1-3]. Other mechanisms include cassava toxicity (cyanogens), genetic factors and oxidative stress.

However, these mechanisms have not been consistently confirmed. Opinion is divided on whether FCPD is a part of the TCP spectrum or a different disease entity.

DM is usually quite severe and insulin-requiring type, though ketosis resistant. Patients require large insulin doses, but macrovascular complications are less common than in type 1 DM. Both endocrine and exocrine functions may be impaired, and pancreatic enzyme supplements are used for relief of abdominal pains and steatorrhea. Surgery may be required; ductotomy, stone clearance and drainage may give good results.

Causes of deaths include diabetic nephropathy and pancreatic adenocarcinoma.

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Vitamin C and the treatment of tetanus

Sir,
Chukwubike et al.\textsuperscript{[1]} estimated that the case fatality rate in their hospital in Nigeria was 43% implying that there is basis for searching new or additional
I collected literature on animal studies that tested the effect of vitamin C against infections and purified bacterial toxins. Dozens of studies reported that vitamin C protected against diverse viral and bacterial infections in guinea pigs, mice, rats and a few other species. Furthermore, 13 study comparisons reported that vitamin C significantly protected against endotoxin and diphtheria toxin administration in guinea pigs and rats.

As to vitamin C and tetanus, the study published by Dey in 1966 is directly relevant. Dey administered tetanus toxin to five control rats, all of which died. When vitamin C was administered after the tetanus toxin to 15 rats, all survived. Although no great weight should be put on an old study as a single report, the context of 13 other comparisons, which found benefit against endotoxin and diphtheria toxin, supports the notion that vitamin C may protect against certain bacterial toxins, including the tetanus toxin.

To my knowledge, only one controlled trial, carried out in Bangladesh in the 1980s, has tested the effect of vitamin C on tetanus patients. Jahan et al. administered 1 g/day of vitamin C intravenously to children aged 1 to 12 years and none of them died (0/31), whereas 74% (23/31) of those in the control group died; both groups received conventional tetanus treatment so that vitamin C operated over and above the normal medication. In patients aged 13 to 30 years, 37% (10/27) of the vitamin C group died compared with 68% (19/28) of the control group. Jahan et al. trial has methodological shortcomings, but the findings cannot be disregarded on the basis of potential biases.

Although vitamin C has effects on the immune system, there is no evident mechanism explaining the effect against tetanus. Nevertheless, the particularly low plasma vitamin C levels in tetanus patients compared with healthy controls does give one way to rationalize the benefit of vitamin C administration to tetanus patients. In any case, proponents of evidence-based medicine emphasize that, when evaluating medical interventions, the focus should not be on biological rationalization, but on controlled trials with clinically relevant outcomes, which means the type of information provided by the Jahan trial. Evidently, more research is needed into the effect of vitamin C on tetanus patients, because the case fatality rate is high with the treatments currently in use.

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