Comments on "Clinical Gallbladder Disease in NIDDM Subjects"

The study by Steven M. Haffner (1) gives a very surprising result indicating that fasting glucose concentration is inversely associated with gallbladder disease.

This result should not be surprising, however, because the gallbladder disease status was determined by a home interview and was considered to be present if the participant responded "yes" to the question, "Have you ever had your gallbladder removed?" Gallbladder disease was also considered to be present if participants had a previous history of stones on X-ray or ultrasound. I would guess that many patients in the study already had a cholecystectomy. Diabetic patients often have enlargement of their gallbladder (2–5) and decreased gallbladder emptying (6), which most likely could dispose them to chronic gallbladder infection.

In my experience, it is not rare for a patient's hyperglycemia to improve markedly after removal of a chronically infected gallbladder. It is my clinical impression that many diabetic patients have a mild, chronic gallbladder infection complicating the management of their diabetes, and many of those patients do not necessarily have gallstones. Further studies are needed to establish the importance of chronic gallbladder disease in diabetes.

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Macrovascular Disease Is Not That Uncommon in Fibrocalculous Pancreatic Diabetes

Fibrocalculous pancreatic diabetes (FCPD) is now a widely recognized subset of malnutrition-related diabetes mellitus, the other being protein-deficient diabetes mellitus (PDDM). FCPD is associated with a chronic, often calculous, pancreatitis of uncertain etiology and is prevalent mainly in certain zones of some tropical developing countries including some of the more southern states of India. We were the first to document its prevalence and characteristics in the eastern Indian state of West Bengal (1). The 28 FCPD patients we came across were consistently young (<30 years of age) with female preponderance. Though they required moderately high doses of insulin from the beginning, they did not develop acetonuria or ketosis despite long interruptions of 8–12 months in insulin therapy. All had suggestive pancreatic ultrasonographic abnormalities, and most had pancreatic calculi in abdominal radiographs and steatorrhoea with normal D-xylose excretion, which was subsequently reduced by pancreatic lipase supplementation. Subsequent to this report, we affirmed the pancreatic involvement with endoscopic retrograde cholangiopancreatography in those without radiologically detectable pancreatic calculi. Later we reported frequent human leukocyte antigen (HLA) DR3 and infrequent HLA DR2 associations in our FCPD patients and raised the possibility of their comprising a subset of chronic calcific pancreatitis of tropics with genetic predisposition to type I diabetes (2).

During these studies we were struck by the absence of macrovascular disease in our FCPD subjects, as assessed clinically and by resting ECG, despite their having long durations of diabetes (8–12 years).

This appeared to conform to the general impression that macrovascular disease is rare in FCPD except coronary heart disease in the occasional elderly patient (3). However, hyperglycemia has been implicated as an independent coronary risk factor (4), and we decided to determine with more certainty the macrovascular status of our patients with treadmill testing, Doppler echocardiography, and Doppler ultrasonographic studies of limb vessels particularly as no such data appeared to be available. We have been able to muster reevaluated data on 24 of our 28 subjects whose other characteristics have already been mentioned. Among the four patients not reevaluated, two died and two were lost to follow-up. The reassessed patients were young (14–33 years of age) with fairly long durations of diabetes (7–12 years). Their serum lipid profiles were quite acceptable and body mass index was low (12.8–19.2 kg/m^2). None of them smoked or were hypertensive, and they appeared to lack any atherosclerotic risk factor besides diabetes.

Despite this we found evidence of subclinical peripheral vascular disease