revealed edematous enlarged pancreas with peripancreatic fluid and no gall bladder disease. All patients recovered with conservative management consisting of intravenous fluids and omega-3 esters.

Another 27-year-old man of Asian Indian descent developed acute pancreatitis with lactic acid serum and a triglyceride level of 3780 mg/dL (42.7 mmol/L) and a normal serum amylase level. He had complications, required ICU admission and succumbed to his illness.

Acute pancreatitis is a well-recognized complication of elevated triglyceride levels. Although the exact mechanism is unknown, there is the view that an elevated cholesterol level alone may not lead to pancreatitis. When serum triglycerides are elevated above 1000 mg/dL (11.3 mmol/L), there is invariably chylomicronemia, which may impair circulation in the capillary beds, exposing the chylomicrons to pancreatic lipase, thus damaging the pancreatic acini and microvasculature.

Although elevated triglyceride levels may predispose to acute pancreatitis, there is usually a precipitating factor including (but not limited to) uncontrolled diabetes, alcohol and medications. Other causes of pancreatitis such as pregnancy, endocrine abnormalities such as hypothyroidism and/or hypopituitarism and estrogen replacement therapy, were also ruled out in our women patients.

There is overall agreement that serum amylase may often be normal or only minimally elevated in these patients even when imaging studies show severe pancreatitis. This is due to the presence of an amylase inhibitor in these cases. Our patients also had normal or slightly elevated amylase levels. Hence serum amylase level should not be relied upon as a diagnostic parameter in hyperlipidemic pancreatitis. Only one patient in our case series had recurrent attacks of acute pancreatitis, but there has been none with chronic pancreatitis. Chronic pancreatitis following hypertriglyceridemia-induced pancreatitis is rare, but has been previously recognized.

There has been a suggestion that Asian Indian migrants to the West have an increased predisposition to coronary heart disease due to elevated “lipoprotein-a” levels. The dietary and environmental factors may also play a role in dyslipidemia. Hence, large-scale genetic and epidemiological studies are required to elucidate the predisposition of migrant Asian Indians to the development of hypertriglyceridemia-induced pancreatitis. Moreover, if any patient of Asian Indian ancestry presents with upper abdominal symptoms suggestive of peptic ulcer, gall bladder disease, GERD, etc., hypertriglyceridemia-induced pancreatitis should be considered, investigated and appropriate treatment instituted, with the recognition that serum amylase may be normal in these patients.

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References

Colonic explosion with use of argon plasma coagulation for radiation proctitis

Argon plasma coagulation (APC) is a non-contact electrocoagulation device that uses high-frequency monopolar current conducted to target tissues through ionized argon gas. Reported complication rates range from 0% to 24% and
include pain at treatment site, gaseous distention, pneumatosis intestinalis, pneumoperitoneum, pneumomediastinum, chronic ulceration, stricture, bleeding, transmural burn syndrome, perforation, and death. A few cases of colonic explosion during APC have been published so far.

A 42-year-old woman, a known case of radiation proctitis, reported for second session of argon plasma coagulation (APC). The colon was prepared with oral saline laxative containing monosodium and disodium phosphate. Colonoscopy showed liquid stool in rectum and features of radiation proctitis. The sigmoid colon was normal. APC was performed using the ERBE 300 system at argon flow rate of 2 liters per minute and power setting of 60W. There was a loud explosion in the colon with the first pulse of APC and the patient complained of a severe burning session in the rectum. Colonoscopy revealed extensive superficial bleeding ulcers in rectum and sigmoid colon. She was hospitalized for observation and managed with IV fluids and tramadol for pain relief. There was no clinical or radiological evidence of perforation and she was discharged after 24 hours.

Methane and hydrogen gas are produced by degradation of alcohol sugars present in bowel cleansing enemas by colonic bacteria. Hydrogen concentration of 4% and methane of 5% can lead to colonic explosion. Accordingly, the use of colonic preparation agents containing mannitol and sorbitol has been discontinued. However, presence of stool in poorly prepared cases may also lead to high concentration of these explosive gases. The use of antibiotics prior to APC to reduce colonic bacterial load has also been considered but is not a viable or cost effective option. Presence of oxygen in the colon has also been implicated as a cause of explosion due to APC leading to suggestion for use of carbon dioxide instead of air for insufflation. There are six reports published so far on colonic explosion due to APC. All these patients except one had colonic preparation with enema, thus highlighting the importance of oral preparation. In all patients except one, the explosion resulted in colonic perforation necessitating surgery. Despite all precautions, however, the risk of colonic explosion cannot be excluded and should be a part of informed consent. Ours is the second report with oral preparation and highlights the importance of deferring a procedure for want of adequate bowel cleansing rather than embarking upon APC with inadequate colonic preparation.

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References