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Editorial

Clinical Significance of Biliary Sludge

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Biliary sludge and microlithiasis appear to be stages of the same metabolic disorder, diagnosed differently; sludge by its typical features on ultrasonography and microlithiasis by microscopic examination of bile collected from the duodenum or common bile duct. Biliary sludge is typically echogenic without any acoustic shadowing; seen as low-level echoes that shift and layer in the dependent portion of the gallbladder; whereas microlithiasis is diagnosed by polarizing light microscopy; under which these crystals are found to have rounded contours and black centres due to scattering/absorption of light.

The pathogenesis of both biliary sludge and microlithiasis appears to be similar – biliary sludge progressing to microliths and then in about 20% to cholesterol gallstones in patients with deranged gallbladder motility and/or the cholesterol supersaturation of bile. This sequence may actually represent progressive stages of cholesterol gallstone formation. Some clinical conditions appear to predispose to the formation of biliary sludge and/or microlithiasis, such as pregnancy, rapid weight loss, organ transplantation, bariatric/ gastric surgery, drugs such as ceftriaxone, atazanavir, somatostatin analogues, and some critical illness involving low or absent oral intake with or without prolonged total parenteral nutrition.

The chemical composition of biliary sludge varies; in pregnant women the sludge consists mainly of cholesterol monohydrate crystals, whereas calcium bilirubinate predominates in those on prolonged TPN, which predisposes gallbladder stasis leading to precipitation of solid cholesterol crystals from bile. On the other hand biliary sludge is composed mostly of calcium-ceftriaxone complexes in individuals receiving prolonged or high-dose ceftriaxone therapy. Although, it is not known if the natural history of these types sludge differs in any way in the clinical setting.

Whereas biliary sludge may remain asymptomatic and get picked up on routine ultrasonograpy in patients with various acute or chronic illnesses, but in a small number the sludge may lead to epigastric and right upper quadrant abdominal pain and mimic biliary colic and be the cause of acute acalculus cholecystitis, acute cholangitis and idiopathic acute pancreatitis.

Administration of ursodeoxycholic acid has been seen to cause a resolution of biliary sludge in a significant number of patients. If it persists laparoscopic cholecystectomy should be offered as definitive therapy. There are few patients with history of with recurrent severe biliary pain, who on ultrasonography have gall stones and whose liver enzymes are found to be elevated; but on magnetic resonance cholangio-pancreatograpghy (MRCP) do not have any

filling defect or dilatation of the CBD. Another sub-set, post cholecystectomy patients continue to have pain and due to recurrent mild acute pancreatitis, most probably due to biliary sludge or microlithiasis. If ursodeoxycholic acid does not resolve their symptoms and reduce levels of liver enzymes; microscopic examination after aspiration of bile from the duodenum or common bile duct has been found to be both diagnostic and endoscopic sphincterotomy may resolve the symptoms and prevent pancreatitis.