Editorial

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Editorial Note on Dysfunction of Gall Bladder

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Editorial

Under normal physiologic conditions, gallbladder contraction often occurs throughout the day. Between meals, the gallbladder retains hepatic bile (with a fasting rate of 25 to 30 ml in healthy subjects). After a meal, depending on the degree of neurohormonal reaction, the gallbladder releases a variable amount of bile. Studies using a combination of choclescintigraphy with the US have found that after a meal, the gallbladder sheds rapidly and replenishes itself. In contrast, an increase in fasting gallbladder volume, as well as incomplete excretion and high volume of residual gallbladder, are often seen in patients with cholesterol gallstones, whether they have small or large stones or lithogenic bile. In patients with cholesterol gallstones and abnormal gallbladder motility, inflammation of the gallbladder wall is usually mild and cannot account for gallbladder fluctuations. In addition, poor interdigestive gallbladder supplementation is associated with the delivery of a large percentage of lithogenic bile from the liver directly into the small intestine, leading to increased enterohepatic effects of increased digestion and bile salt hydrophobicity. This observation shows that the removal and filling of the gallbladder is affected in patients with gallbladder hypomotility. Clinical research has confirmed that hypomotility of the gallbladder is primarily associated with the formation of cholesterol gallstones, although low levels of gallbladder dysmotility, in the absence of an enlarged gallbladder in a fasting state and any gallbladder inflammation, are also found in patients with pigmented gallstones. . In patients with cholesterol gallstones, impaired gallbladder motility persists in the stagnant gallbladder following effective treatment of extracorporeal shock-wave lithotripsy and oral bile acid dissolution therapy. The rate of gallbladder dysfunction has been found to increase with the content of cholesterol in the bile duct, even in healthy subjects without gallstones. These findings suggest that excess cholesterol molecules on the gallbladder wall may act as myotoxic agents.

In vitro studies have found that compared with those in control studies, the function of the gallbladder in patients with cholesterol gallstones shows an abnormality in binding agonists such as CCK to plasma CCK-1 receptors (CCK-1R), changes in fluid retention. different. muscle cells, with reduced contraction of distinct muscles and all gallbladder arrangements. In particular, signal transmission in response to binding agonists is impaired. Shortness of breath associated with cholesterol gallstones is repaired early and is mainly due to the accumulation of excessive biliary cholesterol in the lining of smooth muscle cells in the gallbladder. This mechanism seems to explain why the removal of the gallbladder is damaged before the formation of gallstones in animal models at a time when bile is full of cholesterol. In addition, intracellular pathways for smooth muscle contraction appear in the human gallbladder muscle cells from patients with cholesterol gallstones. These findings support the view that increased cholesterol absorption in the lumen of the gallbladder is associated with dysfunction of the gallbladder muscles. This mutation may cause sarcoplasmic membrane inflammation following increased cholesterol content in the membrane. As a result, when CCK binds its receptor to the smooth muscle cells of the lithogenic gallbladder, the G-proteins are not activated and the gallbladder movement is impaired.

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Conflict of Interest

None