



Physiological Changes after Cholecystectomy

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gallstones, cholecystokinin, laparoscopy, gastroduodenitis

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Introduction-

Gallstone is a common problem in our surrounding and is commonly being treated by cholecystectomy, which is a simple surgery, most of the times performed by laparoscopy. Bile is synthesized in the liver and from liver, flows into the gallbladder, where it is stored until we take our meal. Now a hormone cholecystokinin sends a signal to the gallbladder to release a pool of bile into the intestine, where it breaks down the fat present in our food. Now this fat and bile is reabsorbed and bile is carried back to the liver for reuse. Gallbladder being an important organ of our body and definitely having some important functions, one can live without it because the gallbladder doesn't produce any substance required for digestion. But only difference is that after cholecystectomy instead of being stored in the gallbladder and released only when we take fatty meal, bile will continuously drip from the liver directly into the small intestine.

5 to 40 percent of patients, after cholecystectomy presents with some abdominal symptoms which is named postcholecystectomy syndrome. These problems may be transient or persist lifetime as chronic problem.

Postcholecystectomy syndrome is a dysfunction of the sphincter of oddi, caused by noncalculous obstructive disorder, which decreases bile passage and pancreatic juice outflow into the duodenum.

The patient may present with stomach upset, nausea, vomiting, gaseous distention, diarrhea or pain right abdomen particularly after taking fatty meal. Pain may be either due to sphincter of oddi dysfunction or due to post surgical adhesion. Patient may present with recurrent, severe and / or moderate pain lasting for more than 20 minutes accompanied by nausea and vomiting. About 50% of postcholecystectomy syndrome presents due to biliary causes like biliary microlithiasis, biliary injury, dysmotility and cholelithiasis. Rest of the 50% are due to non-biliary causes. Fatty meal in these patients may also cause diarrhea which sometimes is corrected shortly or improves over time and sometimes remain as a chronic problem and this chronic diarrhea is a type of bile acid diarrhea.

After cholecystectomy, there may be further progression of chronic pancreatitis, dysfunction of the sphincter of oddi, duodenogastric reflux and duodenogastroesophageal reflux. Duodenogastric reflux causes the development of bile reflux gastritis and bile acid dependent atrophic antral gastritis. Duodenogastroesophageal reflux causes the development of chronic oesophagitis. After cholecystectomy, about 5 - 40% patients presents with pain particularly in

the right upper abdomen, while 40 - 60% patients have dyspeptic disorders. About 70% of patients show symptoms of chronic "bland" intrahepatic cholestasis, chronic cholestatic hepatitis and compensatory bile-acid-dependent apoptosis of hepatocytes. While high risk of the colon cancer is found in the patients with high concentration of hydrophobic hepatotoxic co-carcinogenic deoxycholic bile acid in serum and in faeces.

Thus depending on the condition of the sphincter of oddi whether it is hypotonic or hypertonic the patient may present with symptoms of hepato-biliary, pancreatic, duodenal, gastric or oesophageal region.

Mechanism of development of Postcholecystectomy syndrome

Absence of the gallbladder after cholecystectomy causes the increase in passage of hepatic bile into the duodenum and the gallbladder-independent enterohepatic circulation of biliary cholesterol, bilirubin and bile acid. Increased gallbladder-independent enterohepatic circulation of bile acids causes increase in concentration of bile acids in the hepatocytes and decrease in the accumulation and excretory function of the liver. Increased gallbladder independent enterohepatic circulation of biliary cholesterol helps in increased absorption of biliary cholesterol in the small intestine than entering the hepatocytes and hypersecretion into hepatic bile. Due to all these alteration in enterohepatic circulation there is formation of the lithogenic hepatic bile and this predisposes to choledocholithiasis, that is formation of stones in bile ducts. Increase in the gallbladder independent output of biliary cholesterol and in the concentration of total bile acids in duodenal bile, causes the precipitation of cholesterol monohydrate crystals in the duodenal lumen.

In postcholecystectomy patient, due to sphincter of oddi incompetence surplus hepatic bile passage into the duodenum causes formation of duodeno-gastric reflux and development of chronic atrophic antral gastritis, often accompanied by intestinal metaplasia and gastroduodenitis. Sphincter of oddi dysfunction also obstructs passage of hepatic bile into the duodenum resulting in the functional biliary hypertension, dilatation of the common hepatic duct, pain in epigastrium or hypochondrium and chronic bland intrahepatic cholestasis and reactive hepatitis.

Treatment of a patient with post cholecystectomy syndrome

The problems that arise after cholecystectomy like pain abdomen, bloating, diarrhea etc. are usually less problematic than the gallstone attack and it can resolve on its own and

if not it can be treated.

The patient is advised to avoid spicy foods, milk or other dairy products, high fat food, caffeine or alcohol. Enzyme preparations and antispasmodics can also be given and sometimes chologogue.

If the pain is due to microlithiasis, oral ursodeoxycholic acid can be given. Antidiarrheal drug like loperamide can be given. Medications commonly used to treat high cholesterol – bile acid sequestrants such as cholestyramine can be used. It bind to bile and prevent it from being absorbed in the small intestine and returned to the liver for recycling.

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