SCIENTIFIC ARTICLE

Hypercholesterolaemia in patients with symptomatic gallstones

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Abstract

Objective

To determine the incidence of hypercholesterolaemia in patients with symptomatic gallstones

Methods

This is a descriptive observational study. Preoperative serum cholesterol level of patients admitted for surgical removal of the gall bladder to a single surgical unit was studied.

Results

There were 33 patients with a median (range) age of 47 (32-74) y e a r s. 6(18.18%) p a t i e n t s were known to have hyperlipidaemia and 12(36%) patients were newly found to have high serum cholesterol levels: 5(15%) were less than 40 years old. The lowest recorded cholesterol level was 118mg/dl (normal <200 mg/dl).

Conclusions

More than 50% of patients with symptomatic gallstones have hypercholesterolaemia.

Introduction

Gallstone disease is common in Sri Lanka. Although there are anecdotal reports of the high incidence of hypercholesterolaemia among patients with symptomatic gallstones, little published data is available on this subject. Cholesterol is exclusively excreted in bile and it is also known that cholesterol is a

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constituent of gallstones [1, 2]. Therefore, it seems logical to assume that patients with hypercholesterolaemia may form gallstones more frequently than those with normal serum cholesterol, and this would be reflected in a higher incidence of hypercholesterolaemia among patients with symptomatic gallstones. In this study, we analyzed the levels of serum cholesterol in patients with symptomatic gallstones, awaiting elective surgery.

Material and Methods

Patients with symptomatic gallstones, who were admitted to a single surgical unit for elective surgery from November 2010 through January 2012, were included in this study. Patients with haemolytic disorders, common bile duct stones, common hepatic duct stones and obstructive jaundice were excluded from the study. Blood samples were obtained following a prescribed fast before operation. Analysis of serum cholesterol level was performed at the biochemistry laboratory of the Colombo South Teaching Hospital.

Results

The total number of patients included in this study was 33 and the mean age was 48.75 years. The median (range) of the serum total cholesterol was 220 mg/dl (118.2 mg/dl - 284.1 mg/dl). Of 33 patients, 18 (54.5%) had a higher than normal serum cholesterol level, 6(18%) were known to have hyperlipidaemia and 12(36%) were newly found to have high cholesterol levels. 5(15%) of 18 patients were less than 40 years old.

Discussion

Cholesterol is exclusively excreted via bile. Cholesterol concentration in bile is considered to be the main pathological determinant of stone formation. [1,2].

Epidemiological studies done in Taiwan described no relationship between hypercholesterolaemia and gallstones, whereas some other studies showed that hyper-tryglyceridaemia decreased sensitivity of the gallbladder to cholecystokinin (CCK) and increased the risk of stone formation. [3,4].

In this study, just over one half of patients had high serum cholesterol levels compared with 45% of patients with symptomatic gallstones in whom serum cholesterol was within the normal range. It would therefore appear that patients with normal serum cholesterol levels too are at risk of developing gallstones. The safe maximum serum cholesterol level associated with a zero risk of developing gallstones has not been defined thus far, although in this small observational study all patients had serum cholesterol levels higher than 118.2mg/dl. It seems that a large multi-centric study including ultrasound examination of an asymptomatic control population would be necessary to define a serum cholesterol level above which gall stones are likely to occur.

In this study we found over one half of patients who had high serum cholesterol levels, just over one third had a diagnosis of hypercholesterolaemia newly found and one sixth were young patients less than 40 years of age. It is reasonable to suggest that all patients with gallstones other than known causes, such as haemolytic disorders, have their serum cholesterol levels estimated. This would help to find and treat their hyperlipidaemia, which if undetected, may have other cardiovascular health consequences.

There are several mechanical and molecular factors that influence the excretion of cholesterol. Experiments on mice suggest that transport factors murine protein kinase, apolipoprotein E, fibrates, and dietary factor capsacin have some influence on cholesterol excretion in bile and an effect on stone formation(4,5,6). Biliary tract obstruction is the commonest mechanical factor, and that was excluded in this study. However, exclusion of abnormalities of molecular factors such as transport proteins murine protein kinase, apolipoprotein E were not feasible given the limited resources available to us.

In addition, statins and fibrates too are known to

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influence cholesterol excretion – statins, in particular, decrease cholesterol saturation in bile (7). Despite regular use of statins, a small proportion of patients in this study were found to have gallstones.

Conclusion

More than one half of patients with symptomatic gallstones had higher than normal serum cholesterol levels in this observational study of a small cohort of patients. Serum cholesterol estimation in patients with symptomatic gallstones may help find new cases of hypercholesterolaemia. The level of serum cholesterol beyond which individuals risk development of gallstones is yet to be determined, although we understand there may be multiple confounding aetiological factors. A large scale study with ultrasound examination of asymptomatic patients and biochemical analysis of retrieved gallstones is likely to provide more information.

References

- Dikkers A, Tietge UJF. Biliary cholesterol secretion: More than a simple ABC. World J Gastroenterology 2010; 16(47): 5936–5945.
- Venneman NG, van Erpecum KJ. Pathogenesis of gallstones. Gastroenterology Clinics of North America. 2010; 39(2):171-83.
- 3. Chen CH, Huang MH, Yang JC, et al. Prevalence and risk factors of gallstone disease in an adult population of Taiwan: an epidemiological survey. Gastroenterol Hepatol. 2006; 21(11):1737-43
- 4. Smelt AH. Triglycerides and gallstone formation. International journal of clinical chemistry 2010; 411(21-22):1625-31.
- 5. Huang W, Bansode RR, Xie Y, et al. Disruption of the murine protein kinase Cbeta gene promotes gallstone formation and alters biliary lipid and hepatic cholesterol metabolism. Journal of Biological Chemistry 2011; 286(26):22795-805.
- Shubha MC, Reddy RR, Srinivasan K. Antilithogenic influence of dietary capsaicin and curcumin during experimental induction of cholesterol gallstone in mice. Applied Physiology Nutrition and Metabolism 2011; 36(2):201-9.
- 7. Smith JL, Nathanson LK, Riottot M. Effects of statins on Billiary lipids and cholesterol gallstones. Journal of cardiology 2002; 9(7-8), 295-298