(Refer to page 89)

Answer: Melanosísis coli

Black pigmentation of the colon was first described by Cruveilhier in 1829 but it was Virchow who coined the term ‘melanosísis coli’ to describe this disorder.

Melanosísis coli is associated with chronic use of laxatives, commonly anthraquinone-containing such as senna. In our local setting, such compounds can be found in over the counter remedies that are used as slimming medications or medications to help bowel movements. The anthranoid laxatives pass through the gastrointestinal tract unabsorbed until they reach the large intestine, where they are changed into their active forms. The resulting active compounds cause damage to the cells in the lining of the intestine and lead to apoptosis (a form of controlled cell death). The damaged (apoptotic) cells appear as darkly pigmented bodies that may be taken up by macrophages. The dark pigmentation is due to lipofuscin, a cellular pigment that forms when cells are destroyed, often called “wear and tear” pigment. When enough cells have been damaged, the characteristic pigmentation of the bowel wall develops. It can develop after a few months of laxative use and disappear when discontinued. Other causes apart from chronic laxatives or bowel stimulants used, that results in increased colonic epithelial cells apoptosis have also implicated.

On histology melanosis coli shows characteristic pigment-laden macrophages within the mucosa on Positive–Acid Schiff (PAS) staining. Melanosísis coli is a misnomer as lipofuscin is different from melanin and hence melanosis coli is sometimes referred to as pseudomelanosísis coli.

The diagnosis can only be made after colonoscopy and this typically shows blackish discolouration or pigmentation of the colonic mucosa distributed in a lace like pattern that resembles snake skin. The degree of pigmentation can range from very mild to very severe such as our case. It appears to be most intense and most readily detected in the caecum and ascending colon. This may suggest differences in the luminal concentrations of possible offending agents (i.e. higher in the proximal colon), such as laxatives or their by-products. Alternatively, there may be differential regional rates of mucosal absorption within the colon. Finally, there may be colonic regional differences in the topographic distribution of macrophages within the colonic lamina propria.

No adverse effects or consequences of melanosis coli have been identified but patients are usually advised to stop using these medications.

REFERENCES