MELANOSIS COLI
ITS CLINICAL SIGNIFICANCE

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Since its first description by Cruveilhier 1 in 1829 melanotic pigmentation of the large intestine had been only occasionally observed. From his autopsy observations made in 1858, Virchow 2 applied the term melanosis coli to the condition. It was 1911 before the first clinical report by Pick 3 appeared on the recognition sigmoidoscopically of this discoloration of the intestine.

While melanosis coli is not a rare condition it is practically unknown to the average physician. When observed sigmoidoscopically it usually has been passed by or referred to casually as “brown bowel” or by some other descriptive term and its clinical significance overlooked. The latter circumstance has prompted us to present this paper.

GROSS APPEARANCE THROUGH THE SIGMOIDOSCOPE

The mucosal pigmentation in melanosis coli varies widely in different persons. It is usually some shade of brown ranging from a light, almost gray, tone to a deep, dark hue almost inky black. In the earliest stages the pigmentation is demonstrable only microscopically. Most writers have described the pigmentation as being deepest in the cecum and ascending colon and becoming gradually less pronounced distally and rarely being more intense in the distal part of the intestine. The entire colon usually is not involved.

According to Lubarsch and Borchardt,4 the pigmentation is mainly in the cecum, appendix and ascending colon. Lignac 5 stated that the

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melanosis is mainly in the cecum and rectum, with occasional pigmentation in the ileum. Stewart and Hickman reported that the discoloration is almost invariably more intense in the cecum and ascending colon, although rarely the melanosis may be deeper in the distal than in the proximal portion of the large intestine. Bockus and his co-workers found that when the pigmentation is not uniformly distributed it is always more intense in the rectum and in many cases gradually diminishes in the upper portion of the descending intestine.

Other than a melanotic discoloration there are usually no changes in the mucosa of the intestine. Frequently there is accompanying mucous colitis. There are no indications of pigmentation elsewhere, although metastasis of the pigment to the mesocolic lymph nodes occurs at times. There is no evidence as yet as to the more widespread

metastasis of melanosis. The pigmented area of the portion of the intestine affected is broken up into small polyhedral shapes by a network of fine unpigmented lines which correspond, according to Pick, to the ramifications of the mucosal blood vessels. In less advanced stages the pigmented areas are smaller as well as lighter, with a brownish stippling against a normal-appearing mucosal background. The solitary follicles of the intestine, being devoid of pigment, stand out as small yellow follicles the size of a pinhead. They are marked even in the early stages of melanosis and are helpful in making macroscopic diagnosis.

The appearance of the mucosa of the intestine has been compared to that of snake skin, crocodile hide, tiger skin and a cross-section of nutmeg.

**Histologic Appearance**

The pigmentation is usually confined to the stroma of the mucous membrane, where it lies in the cytoplasm of large mononuclear cells, the exact nature of which has not been settled. The distribution in the mucosa varies. In mild cases it is usually in the mucosal villi, while in the advanced stage it is either in the depths of the mucosa close to the muscularis mucosae or scattered throughout its thickness. This distribution is probably due to the formation of melanin superficially followed by a migration downward.

The pigment is usually intracellular, but free granules are also present. The epithelium of the mucosa is usually pigment-free.

**Character and Source of the Pigmentation**

There is a great diversity of opinion with regard to the character and source of the pigmentation in melanosis coli. It is generally believed that the pigment is a true melanin or melanin-like substance. It was formerly believed that pigmentation of the intestine was due to ingestion of preparations of heavy metals, such as mercury or lead. This view, however, was not substantiated by subsequent chemical analysis of sections of pigmented melanotic intestines. From his chemical studies, Virchow suggested a hematogenous origin. This view was put forth by others, and the condition is still referred to in many places in the literature as hemochromatosis (Lynch; Gant). It is difficult to explain the limitation of the pigmentation to the colon if the source is hematogenous.

Vascular congestion with a predisposition to the formation of pigment (Solger\textsuperscript{10}), hemorrhage into the intestine with subsequent bacterial action (Lignac\textsuperscript{5}) and disturbance of the chromogenic function of the liver (Lynch\textsuperscript{8}) have been advanced as probable causes of melanosis coli.

According to Pick, the aromatic products of protein disintegration of the contents of the intestine (indole, skatole) are absorbed from the

Fig. 3.—A high power photomicrograph showing pigmentation in the stroma of the mucous membrane of the rectum.

intestinal and converted into melanin within the connective tissue cells of the mucosa by the action of a tyrosinase-like ferment produced by the connective tissue cells. This ferment is produced only in certain persons. Other theories propose the formation of a ferment in the

\textsuperscript{10} Solger, F. B.: Dickdarmmelanose, Inaug. Dissert., Greifswald, 1898; quoted by Lubarsch and Borchardt.\textsuperscript{4}
contents of the intestine and the production of melanin by that portion of the mucosa which comes in contact with the ferment (McFarland 11), the production of pigment by bacterial action (Dalldorf 12) and the derivation of melanin from foodstuffs and then phagocytosis by the cells of the mucosa of the intestine (Laidlaw 13).

The etiologic significance of the anthracene laxatives in relation to melanosis coli has recently been described by Bockus and his co-workers.7 These observers concluded that “the anthracene laxatives either contain or elaborate a pigment within the bowel which is phagocytised by the deep mucosal cells causing melanosis coli.” They obtained a history of the long use of anthracene laxatives, usually cascara sagrada, in 100 per cent of their cases of melanosis of the bowel. The association of melanosis with constipation or stasis seems to be definitely established. This probably explains the frequency of melanosis in surgically resected appendices and in malignant processes of the intestine.

**REPORT OF CASES**

In 7 of 200 persons examined by sigmoidoscope in our clinic we observed melanotic discoloration of the bowel. This was an incidence of 3.5 per cent. In five the appearance could be likened to an alligator skin. Another suggested somewhat the cross-section of nutmeg and the other presented widely separated stipplings of light brown pigmentation. The table summarizes certain clinical features of our cases.

The most striking feature was that all these seven patients took cascara sagrada. This is quite in agreement with the report of Bockus and his co-workers. It is of interest that when one of our patients was told that she had "brown bowel" she suggested that it might be due to the cascara sagrada which she had been taking for a long time, "for cascara is brown."

In all our cases constipation had been present for many years. Examination disclosed intestinal stasis in each. The headache, drowsiness, dizziness and other symptoms of which these patients complained were probably due to the colonic stasis and not to the melanosis, for similar symptoms may occur in patients with stasis of the colon without melanosis. It is our belief that melanosis coli per se produces no symptoms. We have no evidence that this pigmentation of the bowel is detrimental.

Contrary to the report of most observers that the pigmentation is deepest in the cecum and ascending colon and becomes more intense in the distal part of the intestine, we found that the pigmentation was deepest distally, in the rectum just inside the anal sphincter. Above this area of deepest pigmentation the coloration became gradually lighter as far as we could determine sigmoidoscopically.

Bockus reported that from four to twelve months is usually necessary for a complete disappearance of the pigmentation when proper treatment is instituted. We have observed that following treatment for the constipation and usually associated catarrhal colitis there was a partial clearing up of the pigmentation. Our treatment consisted of nonanthracene cathartics, enemas of tap water, and a nonputrefactive diet. We have not observed a complete disappearance. Our first patient has been under observation and treatment for one and one-half years.

**SUMMARY**

The gross and histologic characteristics of melanosis coli have been reviewed.

Melanosis coli was observed in seven of two hundred consecutive persons who underwent sigmoidoscopic examinations.

The chief apparent factors were chronic constipation and the use of anthracene laxatives over a long period of time.

The pigmentation partially disappears when the anthracene laxatives are stopped and steps are taken for the correction of the constipation.

Melanosis coli per se is not detrimental.