probably would have the problem when they were not engaged in processing the ducks. Their symptoms would likely be rashes, swelling, and systemic problems, rather than localized infections.

The third possibility is that the young workers are being careless in their personal care after receiving small cuts and abrasions in the course of their work (physical cause). This seems the most likely cause, especially since most staphylococcal infections result from bacteria already present on the patient. Twelve patients were drawers, while the other five included two wax pullers, two killers, and one duck nail cutter. Some workers did not wear their gloves continually and some did not cuff their gloves to lessen the chances of duck material getting on their hands.

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Abdominal Pain with Brown Urine as a Diagnostic Problem

James F. Peggs, MD
Ann Arbor, Michigan

Case Report

L.S., a 63-year-old quality control inspector, was first seen, after much prodding from his wife, as an outpatient in the fall of 1978. He had an approximate six-month history of intermittent diarrhea, constipation, and crampy abdominal pain. He denied any blood or mucus in his stools. Pain was unrelated to the time of day, activity, or meals, but seemed exacerbated at times of tension or stress. There was no prior history of gastrointestinal disease or any other significant medical history. Physical examination revealed a slender white male whose abdominal examination including digital exploration of the rectum was totally unremarkable. The patient at the time was encouraged to try a diet free of dairy products and was scheduled to return within a week for sigmoidoscopy and barium enema.

The patient's symptoms had reportedly disappeared on the dietary restriction, and he unilaterally cancelled his work-up. However, over the next three to four months he noted a return of crampy abdominal pains and loose stools with flatus, alternating with constipation. These symptoms had become pronounced enough that he voluntarily rescheduled his sigmoidoscopy and x-ray examination.

At the time of his sigmoidoscopy, the patient was complaining of mild to moderate left lower quadrant abdominal pain. His bowel movements had been infrequent over the past week and yellow and loose in nature. He stated that he had felt nauseous without vomiting and had passed no blood except a scant amount of bright red blood three months prior. Sigmoidoscopy was performed to 17 cm and revealed no abnormal findings, although the patient was poorly prepared. The barium enema was cancelled. In view of his symptoms and prior history, a presumptive diagnosis of diverticulitis was made. The patient was afebrile, but slightly uncomfortable with the pain; a program of bedrest, clear liquid diet, and oral ampicillin (500 mg every six hours) was instituted. He was scheduled to return to the office in 24 hours to be rechecked.

Upon his return, he was found to be suffering from increasing intensity of pain which was described as "constant, aching pain" across his lower abdomen and at the time he was exhibiting a

From the Department of Family Practice, University of Michigan Medical School, Ann Arbor, Michigan. Requests for reprints should be addressed to Dr. James F. Peggs, Department of Family Practice, University of Michigan Medical School, Ann Arbor, MI 48109.

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slight amount of abdominal distension. His abdomen was still soft with no guarding or rebound tenderness; bowel sounds were present but hypoactive. He continued to be afebrile, but in view of his increasing discomfort, he was admitted to the hospital.

The patient was noted at the time of admission to be dehydrated and intravenous hydration and antibiotic therapy were instituted. Initial white blood cell count was 13,300 with a slight shift to the left in the differential. Abdominal flat-plate x-ray films revealed a suggestion of an ileus in the colon. The patient obtained relief of his pain only with intramuscular meperidine. The urine sample obtained upon admission was noted to be very dark brown (coffee-like) in color, only slightly cloudy in nature, with a specific gravity of 1.050. The patient gave a history of having passed a renal stone approximately four years earlier with bloody urine and typical renal colic at that time. Apparently the stone was not recovered for evaluation nor had he ever undergone any x-ray studies of his urinary tract. The admission urine sample was tested for occult blood and bilirubin as well as albumin, sugar, and acetone, and was negative for all. Cytology revealed only 0-2 red blood cells and 0-2 white blood cells per high powered field with no bacteria, casts, or crystals seen. Urine for urobilinogen was positive at a titer of 1:250 (normal 1:8). Tests for porphyrins and porphobilinogens were negative on two separate samples. The urine output remained normal, BUN was 20 mg/100 ml, creatinine was 0.8 mg/100 ml. The liver enzymes, amylase, and electrolytes were normal. Patient's status worsened over the next eight hours, as he became more nauseated and his abdomen more distended. He began vomiting bright red emesis which was strongly positive for blood; his hemoglobin remained stable at 16 gm/100 ml. At this point, a surgeon was consulted. A repeat supine film of the abdomen displayed nearly complete ileus; a nasogastric tube was passed. The patient's WBC count was again elevated at 11,500 with 25 percent band forms in the differential count. He remained afebrile but continued to require intramuscular injections for pain relief. The dark brown color of his urine remained unchanged with high specific gravity, yet his urine output was normal. Urine cultures and Gram smear were also negative.

Following evaluation by a surgeon, the patient underwent a "gentle" barium enema which revealed an obstructive pattern in the distal descending colon. The patient was prepared for emergency colostomy surgery and at that time the presence of a mass in the distal colon was confirmed. There appeared to be no evidence of acute or chronic diverticulitis, no abscess formation was seen, nor was any abnormality of the urinary tract visualized. Subsequent surgery entailed the complete excision of an adenocarcinoma of the colon. The patient's eventual recovery has been complete. Of particular interest is the fact that within six hours after relief of his intestinal obstruction, the patient's urine cleared completely.

Comment

The discoloration of the patient's urine was due to the presence of the substance indican, which is a breakdown product of the amino acid, tryptophan. Tryptophan is normally metabolized to the product indole, which is excreted almost entirely in the stool. However, in the presence of intestinal obstruction, the indole becomes absorbed and further degraded into indican.[1] Indican is dark brown in color and is excreted via the urinary tract. The laboratory diagnosis can be confirmed by the addition of one of several reagents, in this case a combination of hydrochloric acid, calcium hypochlorite, and chloroform (Jaffe method).[2] The addition of the reagent produces a deep blue (indigo) layer at the bottom of the urine sample. This type of discoloration of the urine has been known to occur under several circumstances, including gastric carcinoma, intestinal obstruction, high protein diets, hypochlorhydria, biliary obstruction, and phenylketonuria.[3]

One might find several helpful lessons in such a case. First, an inherent inadequacy in the office management system allowed a patient to cancel and avoid a necessary evaluation at the time of his initial presentation. Secondly, one must remain wary of the distraction caused by a "brown herring."

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