Diagnosis of Intestinal Tuberculosis

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Intestinal tuberculosis occurs as an ulcerative lesion complicating pulmonary tuberculosis, as a hypertrophic mass, and as a miliary form associated with tuberculous peritonitis. The ulcerative lesion is the most common of the three varieties and its diagnosis can be made with a high degree of accuracy. The chief method of diagnosis is roentgenological and its development has been due chiefly to the work of Pirie, Stierlin, Brown and Sampson. The taking of a careful history is of next importance. The clinician must be familiar with the natural history of the various dyspepsias to evaluate properly the symptoms present. Laboratory findings are usually of little aid in diagnosis.

Ulcerative intestinal tuberculosis is always a complication of pulmonary tuberculosis and often shows an activity parallel to that of the parent lesion. However, it frequently runs an independent course and may cause severe symptoms or death after the pulmonary lesions have become inactive. A study of over 300 cases shows a similar incidence in males and females. All ages are represented and the older patients have an incidence similar to younger patients, but the older the enteritis the less distressing are the symptoms. At Hopemont Sanitarium, the autopsy incidence of enteritis is 70 per cent. Clinically, however, it occurs in 15 to 20 per cent of the sanatorium population, where a great majority show far-advanced disease in the lungs. In a tuberculosis sanatorium the non-tuberculous dyspepsias are about four times more frequent than those due to tuberculous enteritis. In this series, only 1 per cent of the cases occurred in association with minimal pulmonary disease; 9 per cent occurred with moderately advanced disease; and 80 per cent with far-advanced disease. The exudative pulmonary lesion was associated with 25 per cent of the enteritis cases, and the chronic form with the remaining 75 per cent. Pulmonary cavitation was present in 94 per cent of the cases at the time of onset of bowel symptoms; cavitation had been present in 4 per cent prior to the onset of symptoms; no cavity was found in 2 per cent of the cases whose diagnosis was made by laparotomy or autopsy. The sputum was positive for tubercle bacilli in 97 per cent at the time of onset of the symptoms.

Intestinal tuberculosis may be present without symptoms. Ten per cent of the enteritis found at autopsy showed no digestive disturbances during life. Enteritis may be present long before symptoms appear and may be started clinically by a pulmonary spread, pleural effusion or an operation. The reason for such variability, we believe, is due mainly to a local neuromuscular irritability plus an afferent-parasympathetic efferent reflex to the involved segment. We feel that this neurogenic view best explains the clinical onset of enteritis, its severity, symptomatology, duration and response to treatment.

The onset of symptoms was sudden in 67 per cent and gradual in 33 per cent. The rapid onset often dates back to some food or laxative or surgical intervention. Although collapse therapy benefits greatly about 40 per cent of all cases of intestinal tuberculosis, yet some cases date their onset to some form of collapse therapy and other cases show an aggravation of symptoms by these procedures.

The symptoms of enteritis may be many or few, mild or severe, local or general. Systemic symptoms may include nervousness, insomnia, chills, fever and failure to gain weight. The digestive symptoms occur in the following order: Anorexia, 85 per cent; crampy pain, 80 per cent; nausea, 70 per cent; diarrhea, 65 per cent; flatulence, 50 per cent; vomiting, 45 per cent; epigastric distress or pain, 30 per cent; constipation, 20 per cent; pyrosis, 20 per cent; tender right lower quadrant, 10 per cent; acid regurgitation, 10 per cent; constipation alternating with diarrhea, 10 per cent; gross blood in stool, 6 per cent; appendicitis, 4 per cent; allergic phenomena in 2 per cent.

Diarrhea and pain occur in two-thirds of all cases and should one wait for these symptoms to appear many early cases will be missed. In this group we include also the cases
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who show only a change in the character of the stool from a normal to a soft consistency and without increase in the daily number; these form about 10 per cent of the diarrhea group. Pain and diarrhea occur in some cases that show no enteritis at autopsy; these are regarded as being due to a tuberculotoxemia. Occasionally, pelvic disease and more rarely cholecystitis will produce diarrhea for varying periods of time.

A common triad of symptoms occurring in 12 per cent of the cases includes constipation, crampy pain and pain in the right lower quadrant. Anorexia may be due to the general tuberculous toxemia or a disturbed intestinal gradient. In our studies, about 80 per cent of the anorexias occurring with enteritis are due to a disturbed gradient, and correction of the gradient often produces a good appetite and hunger sensation in the face of a hopeless pulmonary lesion with severe toxemia and high fever. Gross blood in the stool is of uncommon occurrence in enteritis and is of severe prognostic significance. Unusual symptoms frequently occur. One case had recurring attacks of chills, fever, leucocytosis. X-ray examination showed an enteritis and therapy cleared the symptoms immediately. Several cases showed allergic reactions of hives, diarrhea and abdominal distress upon ingestion of raw milk and fruits; correction of the intestinal gradient caused an immediate disappearance of these symptoms. Some cases presented typical attacks of appendicitis, but were proved to be enteritis. Two cases showed only a foul putrid odor of the stools.

Tuberculosis of the ileocecal area may show symptoms usually associated with the function of the esophagus, stomach and upper jejunum. For that reason, we have divided the symptoms into the divisions, gastric and intestinal. The gastric symptoms include acid regurgitation, pyrosis, nausea, vomiting, epigastric distress, and upper abdominal flatulence. The intestinal symptoms include crampy pain, diarrhea, and lower abdominal distension, constipation and blood in the stool. The intestinal symptoms are produced locally and the gastric are referred and due to a deranged gradient elsewhere in the digestive tract. Most cases show both groups of symptoms. However, in four per cent, only gastric symptoms were present and in eight per cent, only intestinal. In four per cent, the gastric symptoms were mild and in six per cent the intestinal symptoms were minimal. Six per cent of the cases showed normal stool studies with no previous diarrhea, constipation or both.

Physical examination of the abdomen is usually negative. Frequently some tenderness may be present in the right lower quadrant or over the course of the colon, but the signs are not pathognomonic of enteritis. In extensive cases the sigmoidoscope may be used for direct visualization of the ulcers.

Laboratory studies include chiefly the examination of the stool for blood and tubercle bacilli, gastric analyses and coprologic studies. When there is gross blood present in the stool one must rule out hemorrhoids, fissures, fistulae and hemoptysis. The benzedrine reaction is of little value in the diagnosis of enteritis. Prior to the test, the patient should be on a meat-free diet for at least three days and if positive one must rule out gingivitis, swallowing of blood-streaked sputum and postnasal discharge. During sleep these discharges, as well as bloody sputum, are often swallowed. The presence of the specific organism in the stool is of no value for it may be found in the feces of all cases of open pulmonary lesions without any enteritis. Again, many children who do not expectorate and adults with a low-grade cough reflex may show the organism in the stool with no apparent expectoration. When the acid-fast organisms are found in the stool it is further necessary to use cultures and guinea pigs to rule out other acid-fast organisms which are not pathogenic and are frequently present. Many cases of advanced pulmonary disease show abnormal gastric acidity curves and hypoacidity and improper digestion of fats and proteins, but none of these is pathognomonic for enteritis. Blood studies usually show no changes. A leucocytosis associated with pain in the right lower quadrant may mean appendiceal involvement which may be tuberculous or non-specific.

The most accurate method of diagnosis is roentgenologic. One may give barium orally or by enema. Both are frequently used, but in our hands the oral route has proved to be about seven times more diagnostic than the..
A modified Brown-Sampson technique is used and observations are made at six, nine, twelve and thirty hour intervals. The double meal may be used six hours apart. Each case is fluoroscoped at every interval and plates are made. The enema does not often give a positive diagnosis when the oral route is negative, but it should be used in puzzling cases. The reasons for this discrepancy between meal and enema are due to the fact that the enema is an abnormal load for the colon and finer grades of irritability are missed by the sheer weight of the solution; that the enema must be retained for at least 5 to 15 minutes prior to taking plates; that the meal gives a better insight to the physiologic disturbances present. We have gained no special information from the use of the double contrast air-barium enema in the study of this disease.

The roentgenology of ulcerative enteritis is predominantly that of the terminal ileum, appendix, cecum and colon. Although unusual emptying of the small gut or prolonged filling of a segment may point to enteritis of the small bowel, yet in the great number of cases the diagnosis is conjectural. However, the small bowel is almost always associated with involvement of the cecum or colon, which lend themselves easily to x-ray observation.

In a follow-up study of our cases we have begun to regard all pathological ulcerative enteritis to be productive of symptoms in 90 per cent of cases and to be asymptomatic in 10 per cent. Of the symptomatic group, about 85 per cent produce definite x-ray changes and 15 per cent do not. Thus, in a given series of pathological enteritis, one may find positive x-ray findings in 78 per cent with and without symptoms, and negative x-ray findings in 22 per cent with and without symptoms. We have found in our series that a positive x-ray diagnosis means enteritis in over 95 per cent of cases. However, a negative diagnosis in the face of highly suspicious symptoms is only 30 per cent reliable, and a suspicious x-ray diagnosis after two examinations is less than 50 per cent reliable. It is thus seen that the greatest value of the x-ray lies in the finding of positive roentgenologic signs and this result may be obtained in about 80 per cent of all cases of enteritis.

The criteria for a positive x-ray diagnosis are anatomic and physiologic. Actual demonstration of the ulcers is technically difficult and can be made in only very few cases. The physiologic features include those produced locally at the ulcer-bearing area and contribute factors. The local signs include a failure of the involved area to fill or fill well, resulting in a "spastic filling defect," increased irritability, spasm and rapid emptying. Contributory signs include a generalized hypermotility, rapid emptying of the entire bowel, ileal stasis and gastric retention.

Cases presenting suspicious symptoms over a long period of time may show only a dilated, atonic cecum, which may present a typical spastic picture at a later examination. Such cases should be regarded as quiescent rather than healed, for pneumoperitoneum may change a deformed cecum to a dilated one in the course of a few days.

Suspicious roentgenologic cases are often cleared up by a diagnostic pneumoperitoneum. One or two inflations, in a case presenting suspicious symptoms and suspicious roentgenology often makes the diagnosis. If such a case is not due to enteritis no change occurs, but if due to enteritis the symptoms are quickly minimized and the x-ray picture changes completely and assumes normal aspects.

**Differential Diagnosis:** Ulcerative enteritis may simulate peptic ulcer, gall-bladder disease, or any other disease of the digestive tract. On the other hand, pelvic disease, renal disease, the neuroses, pleural effusion, dyspepsias and postphrenicotomy dyspepsias may simulate enteritis and must be taken into consideration in the diagnosis. X-ray studies can rule out the neuroses, malignancy, mucous and spastic colitis, hyperthyroidism, allergic dyspepsias and pleural effusion. Amoebic dysentery must be ruled out by stool studies. Non-specific colitis may resemble the tuberculous form, but usually occurs in the distal colon. Whenever appendicitis is suspected in a case of pulmonary tuberculosis always rule out enteritis. Very rarely does appendicitis produce localized spastic defects in the cecum or colon and when one has tenderness in the right lower quadrant, with or without leukocytosis and with a normal roentgenologic cecum, one may diagnose appendicitis. Abdominal adhesions may sim-
ulate the tuberculous picture and their diagnosis depends usually upon a careful history and, if necessary, laparotomy.*

Summary

Ulcerative intestinal tuberculosis is always a complication of pulmonary disease occurring usually in far-advanced cases with cavity and positive sputum. Although most cases show a classical symptomatology, yet many other diseases resemble it and no one group of symptoms is pathognomonic. Laboratory aids and physical examination are of little help in diagnosis. The x-ray is, at the present time, our best means of diagnosis, and positive x-ray findings may be obtained in 80 per cent of all cases of enteritis. A positive diagnosis is correct in over 95 per cent of cases; a negative one with highly suggestive symptoms is only 30 per cent reliable, and a suspicious one is less than 50 per cent reliable. The diagnosis in suspicious x-ray cases may be cleared up with diagnostic pneumoperitoneum. A group of about 20 per cent of enteritis cases presents normal x-ray findings and in these a careful history and therapeutic pneumoperitoneum will usually clear up the diagnosis.

References

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3 Salkin, David: "Pneumoperitoneum in Intestinal Tuberculosis," American Review of Tuberculosis, XXXIII, 4, April, 1936.

* Editor's note: Abdominal adhesions may very often be demonstrated by x-ray after pneumoperitoneum is established.