BOWEL X-RAY ALTERATIONS IN ACUTE HUMAN SCHISTOSOMIASIS

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A radiological study of the small intestine of 17 untreated patients in the acute phase of schistosomiasis was performed. Twelve patients (70% of total) had alterations: nine had clear-cut thickening of the duodenal and jejunal folds, one flocculation, one fragmentation and one thickening of mucosae, flocculation and fragmentation of the barium column.

There was no correlation of the gastrointestinal symptomatology (vomiting, diarrhoea, dysentery, hepatomegaly) neither with the parasitological load nor with the x-ray alterations.

Key words: Schistosoma mansoni, Acute toxemic schistosomiasis, Bowel x-ray alterations.

The post-postural phase of schistosomiasis mansoni has extremely protean clinical manifestations. Usually the acute phase is oligosymptomatic. However, some cases present severe symptoms with peculiar clinical and pathological manifestations, defined as the toxemic acute form of the disease.

This paper reports the clinical and radiological aspects of the small intestine within two months after infection in patients with the toxemic acute form of schistosomiasis mansoni.

MATERIAL AND METHODS

Seventeen patients, infected for the first time, presenting with the toxemic acute form of schistosomiasis mansoni were studied before treatment.

Diagnosis was made based on prior negative coproscopic examination that became positive for S. mansoni eggs from 30 to 60 days after the infecting bath, and absence of other parasites in 6 consecutive coproscopic examinations. Also at least one of two clinic-epidemiological criteria namely: absence of prior contact with natural waters contaminated by Schistosoma mansoni cercariae; emergence of a toxic-infectious clinical picture post-contact with infected water consisting of diarrhoeal and/or dysenteric syndrome, cough, fever and hepatosplenomegaly; or hematologic: blood eosinophilia above 1,000 cells/mm³.

The parasitological stool examination was performed by the Kato’s method modified by Katz et al. The patients were divided in 3 groups, according to the average number of eggs of S. mansoni per gram of feces: high load = more than 400 eggs/gram of feces (epg), medium load = between 100 and 400 epg, low load = less than 100 epg.

After previous consent a conventional radiological barium contrast study of the small bowel was performed in all patients. The small intestine was studied radiographically following the administration of a mixture of 8 ounces by volume of barium sulphate and water to make 16 fluid ounces. The initial film was made at 15 min. and a second after another 15 min. interval. Further film depends on the rate of passage of the barium meal, which usually enables an examination every 30-60 min. Neither saline nor ice water were administered because these preparations in themselves disturb the pattern of the small intestine. In the interpretation of diffuse lesions of the small intestine, larger quantities of barium were preferable so that several intestinal loops of bowel were visualized in continuity at the same time. Small amounts of barium may not reflect the morbid anatomy of the small intestine due to incomplete filling. Frequently, incomplete distention may simulate an abnormality. The larger quantity of barium has the following advantages: 1. fewer films are needed, 2. increased motility is obtained; and, 3. relationship of one intestinal segment to another can be determined (thickness of wall can be evaluated).

Clinically all patients were well nourished with normal blood protein levels.

The x-rays were analysed blind by three investigators and alterations were considered when at least two of the observations agreed.

The chi-square (X²) test was used. The significance level of 5% was accepted for all tests.
RESULTS

Twelve (70%) out of 17 patients showed some alteration in the x-ray of the small intestine ranging from flocculation/fragmentation of the barium column to clear-cut mucosal thickening in the duodenal loop and proximal jejunum (Table 1 and Fig. 1).

Among the 12 patients with intestinal radiological abnormalities, nine had also clinical manifestations (diarrhoea, dysentery, nausea, vomiting). On the other hand out of the five patients without x-ray alterations, two had gastrointestinal manifestations (Table 2).

There was no correlation of the x-ray alterations, neither with the gastrointestinal symptomatology nor with the parasite load (Table 3).

DISCUSSION

On reaching their maturity in the portal system, the adult worms of *S. mansoni*, alone or coupled, migrate against the blood stream in direction to the mesenteric venous branches, specially to the inferior mesenteric ones. Once there, the female begins to deposit her eggs. The onset of the oviposition is variable, ranging between the 27th and the 49th days after the infection in humans.\(^5\) \(^6\) \(^21\) \(^22\).

The manifestations of the toxemic form of *S. mansoni* infection represent an exacerbation of the pre-postural clinical features\(^18\) and may be confused with typhoid fever and several other diseases. Based on the clinical picture presented by the patients and the total and differential leukocyte counts the diagnosis of enteritis associated with *Staphylococcus*, *Salmonella*, *Shigella*, ECHO virus, Poliovirus, Coxsackievirus and Legionnaire's disease are less likely. The coproscopic examinations failed to find evidence of *Giardia*, *Entamoeba*, *Balantidium*, *Taenia*, *Enterobius*, *Ascaris*, *Ancylostoma*, *Necator*, and *Strongyloides*.

In pathological terms toxemic acute schistosomiasis mansoni infection can be characterised by the following events: 1) intense, massive, miliar dissemination of eggs with formation of schistosomal granulomas in the liver, large and small bowel, visceral peritoneum, abdominal and mediastinal lymph nodes, lungs, pleurae and pancreas; 2) all the granulomas are in the necrotic-exsudative phase (hyperergic); 3) the

Table 1 – Alterations observed by the radiological study of the small intestine in 17 patients with the toxemic form of schistosomiasis mansoni.

<table>
<thead>
<tr>
<th>Radiological picture</th>
<th>Number of Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Flocculation of contrast medium</td>
<td>1</td>
</tr>
<tr>
<td>Fragmentation of contrast medium</td>
<td>1</td>
</tr>
<tr>
<td>Mucosal thickening of duodenal loop</td>
<td>3</td>
</tr>
<tr>
<td>Mucosal thickening of proximal jejunum</td>
<td>1</td>
</tr>
<tr>
<td>Mucosal thickening of duodenal loop and proximal jejunum</td>
<td>5</td>
</tr>
<tr>
<td>Mucosal thickening of duodenal loop and proximal jejunum with flocculation and fragmentation</td>
<td>1</td>
</tr>
<tr>
<td>Normal</td>
<td>5</td>
</tr>
<tr>
<td>Total</td>
<td>17</td>
</tr>
</tbody>
</table>
granulomas are numerous at unusual places, mainly the visceral peritoneum, pancreas and lungs; the hepatic distribution is irregular; 4) in the majority of cases, a superficial disseminated, acute ulcerative enterocolitis coexists with the former events; sometimes it can be slightly haemorrhagic. Usually the intestinal ulcers are small (1.0 – 1.5 mm in diameter), with plain and regular edges. Occasionally these lesions can be larger, with irregular edges, leading to perforation as reported in two autopsy cases. The acute catarrhal enterocolitis is characterized by congestion, edema and epithelial scaling, followed by inflammatory infiltration of the lamina propria and submucosa, with neutrophils, eosinophils, lymphocytes and plasma cells, diffuse or focal, almost always intense, and independent of the granulomatous lesion.

The intestinal symptomatology may be related to alterations in the large bowel and not to the small bowel radiological findings here described. In other words, the pathological alterations are more intense in the large bowel and this dominates the clinical picture. The lack of relation between the x-ray alterations and the parasitological load suggests that a factor other than the mechanical one (obstruction by the presence of eggs) may be responsible for the small intestine involvement.

The deposition of immune complexes in the genesis of these alterations can not be excluded since they have been demonstrated in high concentrations in
the initial phase of the disease\textsuperscript{10,24} and they may deposit in various organs such as kidney, lung, etc.\textsuperscript{1,2,3,4,7,20}

Thus in the acute form of schistosomiasis mansoni the small intestine lesions may not be directly related to the presence of eggs and/or worms, but may be secondary to a hypersensitivity phenomenon\textsuperscript{3,7,18}.

**REFERENCES**