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A Case Report of Lamblial Cholecystitis

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A case of lamblial cholecystitis in 31-year-old male patient was studied, and the following findings were obtained: 1. The patient developed no intestinal symptom, whereas the main complaint was fever and right hypochondralgia. 2. He was a hard muscular laborer and a heavy drinker of raw alcoholic beverage. 3. There was found hepatomegaly, a tumor in cystic region with tender-ness. Lamblia intestinalis was discovered in B-and C-gall. 4. Gastric juice, urine and feces were examined for lamblia. No lamblia was identified by smear method, but culture in Dobell and Waidaw's media proved the presence of lamblia in feces. 5. Hematological study revealed no eosinophilia but relative lymphocytosis. 6. Hypersensitivity of the autonomous nervous system causing Oddi's muscle contraction was not demonstrated. 7. As for treatment, chloroquine diphosphate was used with good response.

Lamblia intestinalis, a lamblia of protozoa, is known to have parasitic life in the upper part of the small intestine, duodenum, ductus choledocus or gall bladder. Except rare instance of asymptomatic cases, it often causes various symptoms of lambliasis.

Originally this protozoa was discovered by LAMB in 1859 in the mucous stool of a child who had diarrhea. Since his classical treatise of its relation to diarrhea, various opinions on the pathogenesis have appeared without reaching to any final conclusion.

We are going to report here an interesting case of cholecystitis which was caused by lamblia intestinalis.

CASE REPORT

Name of the patient: T. K., a 31-year-old male mason used to drink every day a considerable amount of raw whisky of poor quality since August 1960.

Present History: In December 1960 he noticed slight right hypochondralgia without other symptoms. In January 1961, however, the hypochondralgia increased its intensity. Owing to the feeling of fullness of stomach after eating small amount of food, he could not eat any more in spite of his good appetite, gradually resulting in fatigue and general
malaise, to which he paid little attention. He did not notice any gastrointestinal disorder such as nausea, vomiting or diarrhea, although the right hypochondralgia bothered him intermittently once a week or every ten days and disappeared after bed rest for a few days.

On February 8, this year, however, febrile feeling accompanied hypochondralgia, general malaise, fullness of abdomen, and anorexia. Fever and pain increased their severity so rapidly that he visited our clinic and was admitted on February 18.

Chief Complaint: right hypochondralgia.

Clinical Findings at Admission: A man of medium height and structure looked emaciated and pale, although he was mentally alert with normal temperature and pulse rate. No jaundice was noted on the skin, conjunctiva bulbi and soft palate. Cervical and other lymphnodes were not enlarged. Tongue, throat and mouth were essentially normal. Pupils were round and equally responsive to light. Chest was negative without any murmur or rale. Abdomen was slightly distended but fluctuation was not demonstrated. The hepatopulmonary border was at the lower end of the 6th rib on the right mamillary line. The lower edge of the liver was felt three finger breadth below the costal arch on the right mamillary line. The consistence was normally soft but slightly tender. There was palpable an egg-size mass suggestive of the gall bladder four finger breadth below the lower edge of the liver on the mid-clavicular line. The surface of the mass was flat and smooth. The consistence was elastic soft, and its tenderness was as mild as the liver. There was no abnormality in extremities with normal deep reflex and negative pathological reflexes.

Laboratory Findings: Blood; erythrocytes 4,400,000, leukocytes 9,800, hemoglobin 85% (SAHLI). Differential cell count of peripheral blood shows segmented neutrophile leukocytes 42%, stab 5%, lymphocytes 45%, monocytes 5%, eosinophile leukocytes 2%. Urine reveals no albumin, glucose, or urobilinogen. Lamblia was negative both on smear and culture. Gastric content showed low acidity. Smear and culture proved no lamblia.

Liver Function Tests: BSP 2.5% after 30 minutes. LUGOL test negative. Takada reaction was positive in only one tube. Icterus index of the serum was 5.

Cholecystogram revealed normal position and filling, but contraction of the gall bladder was poor, as shown in Figure 1.

Gall: It lacked A-gall. Twenty cc of B-and C-gall showed clear normal color without urobilinogen. Sediment of B-and C-gall contained many bodies of protozoa and some leukocytes. No pyogenic bacteria, E. colli or erythrocytes were demonstrated.

Pancreas Function Tests: Serum amylase was 8 units, and urine amylase was 4 units.
Seroreaction for syphilis was negative. Blood sedimentation rate was 2 mm/1 h, and 7 mm/2 h. Blood pressure was 125/80 mm Hg.

Chest X-ray revealed no abnormality.

Treatment and Course: After admission right hypochondralgia was complained once a day accompanied by temperature of 37.0 C., general malaise, abdominal distention, anorexia. The liver and the mass suggestive of the gall bladder were palpable with tenderness, whereas liver function test was normal without any visible jaundice or abnormal finding of urine. The patient was treated symptomatically for the first week during which time clinical and laboratory examinations were carried out.

Lamblia intestinalis was identified in B-and C-gall first time on February 27, and other microorganism was excluded. Chloroquine (Resochin-Beyer) was administered orally after each meal three times a day, totaling 0.75 g per day under the diagnosis of amblial cholecystitis. Clinical symptoms disappeared one month after admission. After end of the first month of hospitalization all clinical symptoms disappeared together with tenderness of the liver which was palpated only 1½ cm. below the costal arch, and the tumor around the gall bladder was palpable only as a small limited mass. Lambliae in the gall have decreased after 10 days of Resoquine administration and disappeared completely from B-and C-gall three weeks later on smear.

DISCUSSION

There are many theories on parasitic location of lamblia in human body, the upper small intestine, bile duct and gall bladder, temporary or permanently. However, clinical classification into gastrointestinal and cholecystic types has been accepted by many scholars. Among numerous reports on the bile duct and gall bladder, only a few, WESTPHAL u. GEORGI, CHARIOT and NOGI, were able to demonstrate lambliae in these organs. Araki proved the absence of lamblia in the removed gall bladder of a patient with choledocho-cholecystic type lambliasis. He also proved histologically the absence of inflammatory infiltration, edema, or vascular change which should be expected at the site of parasitic habitation. From these facts he doubts the possibility of lamblia habitation in the bile duct and gall bladder and considers it premature to suspect bile duct or gall bladder habitation on the basis of the presence of lambliae in the fluid collected through gastric tube in the duodenum.

As to pathogenesis of lamblia some believe in toxicity of lamblia, some in bacterial infection, and others in spasm of the Oddi's muscle. No one has based on animal experimentation but clinical observation on individual cases, because pure culture of lamblia has been extremely
difficult. **Araki**, however, succeeded in pure culture, and he tested effects of various drugs, coming to the conclusion that lambliae may obtain pathogenesis as soon as they reach to a certain concentration.

We attempted culture of lamblia in order to study its rasitic habit. Discovering microscopically many lamblia bodies (productive form) in sediments of B-and C-gall, we assumed the location of habitation as the gall bladder or bile duct. Similar examination on feces, urine, gastric fluid revealed no presence of lamblia body or spore. Suspecting the possible excretion of invisible spores into feces from the bile in which we discovered many lambliae, culture of feces was repeated on Dobell and Waidaw's media, which finally exhibited lambliae swimming after 24 hours of incubation in 37.0°C. Their identity was confirmed by microscopical examination. The same method was applied on urine and gastric juice without proving amblia. These findings indicate that parasitic location in this case must be below the gall bladder. It is unlikely that reproduction of amblia took place in the intestinal canal because no intestinal symptoms were noted clinically.

On the other hand, there are clinical findings supportive of pathogeneity of amblia. First, lymphocytosis was common whereas eosinophilia was absent. Second, many lambliae and a few leukocytes, instead of pyogenic cocci, colli or erythrocytes, were found in the gall. Third, hypersensitivity of the autonomous nervous system to cause muscular spasm of the Oddi's muscle was not noted. These three facts are against both infection theory and Oddi's muscle contraction theory. Repeated episodes of pain and fever can be ascribed to non-bacterial chronic inflammation caused by reproduction of ambliae.

Above-mentioned facts lead us to the following unprecedented opinion concerning the etiology of this disease. Fatigue due to muscular labor and habitual intake of raw alcoholic beverage might have worked as a stress providing opportunities for inactive ambliae in the small intestine to enter into the bile duct and gall bladder causing symptoms.

For treatment of this disease, Atebrin, Carbamigen and Choromy-cetin have been used by many authors and their effects were satisfactory. In our experience, symptoms as well as lambliae in the gall disappeared after twenty days of administration of Chloroquine phosphate (Resoschin-Beyer), a new anti-malaria drug which is replacing quinine and Atebrine in recent years.

**SUMMARY**

According to the statistical reports, incidence of amblia infection among Japanese is around 10 per cent. It is by no means a rare parasite, whereas ambliasis has been regarded as a rare disease which actually escaped the
attention of most clinicians. We found a 31-year-old male who suffered of ambiasis, and the clinical picture and review of this disease were presented in this paper.

REFERENCE

Fig. 1. Cholecyst gram. The left: One hour after injection of 20cc of 50% Biligraphin. No filling defect, unusual filling or position are demonstrated. The right: One hour after eating an egg (2 hours after Biligraphin injection). Insufficient contraction is noted.

Fig. 2. Lambriae in B-bile. Arrow indicates numerous productive forms of lambriae. High magnification (Field's method)

Fig. 3. Lambriae in C-bile, not as many as in Fig. 2. High magnification (Field's method)