Complications of the hydatid disease of the liver

Xu Ming-qian

Department of Surgery, The People's General Hospital of Xinjiang Uighur Autonomous Region

Six hundred and fifty patients with hydatid disease of the liver had been treated by surgical operation in our hospital between 1953 and 1982, of these 220 developed various complications (38.8%). In this paper, a preliminary analysis is presented of the forms of the manifestations.

1. Hydatid cyst infection

One hundred and twelve cases had hydatid cyst infection. The frequency of developing cyst infection was higher in hepatic echinococcosis than in pulmonary echinococcosis, and it was more often encountered in adults than in children. Biliary fistula was the leading cause of infection, other factors such as too numerous daughter cysts, decreased cystic fluid, regressive cystic degeneration due to malnutrition, too long a disease history, old and feeble cysts, reduced biologic activity, inflammatory infiltration from adjacent areas and blood-disseminated infection were also causative of this complication.

Hepatopostema was the patient's first presenting symptom seen after hydatid cyst infection, followed by systematic inflammatory responses, chilly feeling and fever and accompanied often by jaundice, toxic anemia, wasting and failure. The local signs were significant: hepatomagaly, persistent dull pain, tenderness and knock pain might be found. Chronic consumption with long period of infection led to wasting changes. A small proportion of the patients had no significant clinical inflammatory responses probably owing to the hypertrophied wall of ectocysts which restricted the infiltration of inflammation into hepatic tissues. Roentgenoscopy showed raised diaphragm and decreased movement of diaphragm with respiration; in severe cases inflammatory pleural effusion occurred with disappearance of costophrenic and cardiophrenic angles and an obscure shadow between the right lower chest and the liver area.

2. Hydatid cyst rupture

There were 53 cases (8.10%) with hydatid cyst rupture. The incidence of rupture was lower in hepatic echinococcosis than in pulmonary or other visceral echinococcosis, and higher in children than in adults. The forcible pressure from the outside might be the leading cause of rupture, followed in turn by local shaking, perforation due to infection

Present address: Department of Surgery, The People's General Hospital of Xinjiang Uighur Autonomous Region, 91, Heaven Lake Road, Urumqi, Xinjiang, China
and spontaneous rupture.

(1) Rupture into biliary tract

Twenty-four cases were of rupture into biliary tract. The extensive growth of the cysts in the liver gave rise to pressure on liver tissues, which caused the hepatic ductules to be displaced and distorted and the bile drain to be not free, thus being liable to develop biliary effusion or minor cholerrhagia, and it was seen on surgical operation that the endocyst wall stained yellow and that the cystic fluid remained as clear as water. Because the ectocyst wall is the products of proliferation of hepatic tissues, among which is hepatic duct, which became atrophic because of long-term pressure, being a membrane-like residual end, the residual end of hepatic duct ruptured with the loss of supporting of endocyst in the presence of the impulse of internal pressure within the hepatic duct or the distortion of hydatid cyst due to being shaken and opened to the inner wall of ectocyst, forming biliary fistula. With larger fistula, endocyst rupture might be brought about and the cystic fluid might flow into hepatic duct, provoking acute cholecystalgia and chills and fever, often accompanied secondary bacterial infection. Obstructive jaundice was seen in the situation where the biliary tract was blocked by daughter cysts or endocyst fragments, often producing acute obstructive and pyogenic cholangitis. It was observed on operation that bile had leaked out from the ectocyst wall, this being the principal cause of infection of effusion in residual loculi (Fig. 1).

Two cases of hepatic echinococcosis with cholecystofistula were seen in our study of this series of cases. Cystic fluid was found to have escaped into gall bladder and bile to have deposited in hydatid cysts, leaving the cysts to become necrotic and to form calculus after long period of time. Clay-like calculus of bile pigments with collapsed and necrotic daughter cysts and endocyst fragments in it was seen in hydatid cysts on surgical removal (Fig. 2).
Cholangiography revealed chains of small spherical defects in multitude and hepatic duct was seen to connect with residual loculi of ectocyst by Tube T method.

(2) Rupture into abdominal cavity

It was seen in 17 cases and more frequent in children than in adults. After rupture the cystic fluid rapidly escaped into abdominal cavity, immediately causing acute diffuse peritonitis and anaphylactic reaction. Of the 17 cases, 9 had anaphylactic shock and 1 had abrupt onset of sharp abdomen pain and anaphylactic shock owing to the pressure of the thigh on the abdomen when bowing his trunk to put on his boots while staying in the hospital. He was immediately operated on, and the hydatid cyst was found to have ruptured, with the cystic fluid escaped into peritoneal cavity. Measures to clean the endocyst and to wash abdominal cavity repeatedly were undertaken to tide the patient over the anaphylactic episode. But six years later another 13 hydatid cysts were removed from the patient’s abdominal cavity. Three other cases were observed in which ruptured ectocysts of hydatid cysts of the liver permitted the entrance of entire endocysts into peritoneal cavity, where adhesion of the endocysts to epiploon, mesentery and intestinal tract formed new ectocysts wherein they resided. Suggestion was stimulated from this case that removal of intact endocysts be adopted in situation where hydatid cysts project over the surface of the liver.

(3) Rupture into lungs

It occurred in 8 cases. The hydatid cysts at the top of the liver, being subjected to the resistance of the hepatic tissues, grew upwards towards the diaphragm and gradually projected over the surface of the liver; in addition, that the pressure of the chest was lower than that of abdominal cavity facilitated further the extension of hydatid cysts into thoracic cavity; and when the ectocysts adhered to the diaphragm and the bottom of the lungs following complicating secondary bacterial infection, rupture into the lungs followed owing to the penetration of the diaphragm by the inflammatory infiltration, resulting in pulmonary abscess and bronchial fistula, with daughter cysts and cyst fragments in the purulent sputum coughed out. In the presence of purulent sputum staining yellow, it might be regarded as a sign of producing hepatic duct-hydatid cysts-bronchial fistula.

Roentgenoscopy showed inflammatory mass images of pulmonary abscess to drain for the hilus pulmonis. With gas entering the hydatid cyst, there appeared an air-fluid balance in the

Fig. 3 Hydatid cyst in the right lobe of the liver, complicated with rupture into the lung and bronchial fistula.
It shows air-fluid balance in the liver and inflammatory shadows of the lower field of the right lung.
liver, and the endocyst floated on the fluid, showing "lotus-on-water" sign. If there were lots of daughter cysts to float, a "soap bubble" sign could be observed (Fig. 3).

(4) Rupture into thoracic cavity

Two cases had rupture into thoracic cavity. Huge hydatid cysts at the top of the liver are to adhere, at length, to the diaphragm, which remains detached from the bottom of the lungs in the absence of inflammation. When trauma leads to the sudden increase of intra-abdominal pressure, the hydatid cysts at the top of the liver may be ruptured directly into thoracic cavity, causing acute pleural effusion, chest pain, stifling chest, high fever, rapid breathing and anaphylactic reaction. One of the two patients who had rupture into thoracic cavity developed anaphylactic shock. Roentgenography showed pleural effusion and pulmonary atrophy.

(5) Rupture into pericardium

Two cases developed rupture into pericardium. It was observed that it was the huge hydatid cysts of the liver that penetrated the diaphragm, the bottom of the lungs and the lower pulmonary ligaments through the inflammatory infiltration following complicating secondary bacterial infection and ruptured into pericardium, causing acute pericarditis and symptoms of pericardial plugging. Roentgenoscopy showed high position of right diaphragm, extensive pericardial effusion with disappearance of fluctuation, and mass inflammatory shadows of cardiacophrenic angle, lung bottom and diaphragm.

On performing pericardiotomy, it was found that the pericardium was full of purulent fluid and daughter cysts. The pericardium was cleaned and the penetrated sinus and its adjacent inflammatory pulmonary and diaphragmatic tissues were removed. Both of the patients were cured of their hydatid disease by removal of liver cysts through diaphragm.

(6) Rupture out of abdominal wall

It was seen in 1 patient who had hydatid cyst at the lower part of the liver, which projected into abdominal cavity and adhered to the abdominal wall following infection. With long time of inflammatory infiltration, the perforation of ectocyst led to rupture of the hydatid cyst into muscularis of abdominal wall, forming abscess, which gradually ulcerated and drained off, leaving a collapsed endocyst. Having formed auto-"marsupialization", the patient recovered by free drainage (Fig. 4).

3. Anaphylactic shock

It occurred in 10 patients, 9 of whom were of cyst rupture into abdominal and 1 into thoracic cavity, but in none of the 34 cases with rupture into biliary tract, lungs, pericardium,
or abdominal wall. There were 4 patients in this series who developed anaphylactic shock during operative phase. Clinical observations demonstrated that anaphylactic shock appeared often in the presence of contamination of serous cavity by cystic fluid and that rupture into tract or duct did not result in anaphylactic reaction.

4. Disseminated implantation

As a result of hydatid cyst rupture into abdominal cavity, the cystic fluid flows into the cavity, with scolices swarming into the abdominal cavity. If not phagocytized by immunized cells, the scolices adhere to the serous membrane of abdominal viscera, and develop into new secondary hydatid cysts (if scolices are ingested by dog, they develop into *Echinococcus granulosus* in the dog's intestine).

Multiple cysts may occur following ingesting many of the tapeworm eggs at a time. One case in this series had 27 cysts, as large as table tennis, it might be due to infection at a time. Some patients with hepatic echinococcosis developed abdominal cysts several years later, which might be derived from superinfection. Of the 17 cases with rupture into abdominal cavity, 14 had secondary abdominal echinococcosis. Four patients with liver cysts developed secondary abdominal cysts after surgical operation.

It was observed that secondary disseminated hydatid cysts caused by rupture of liver cysts, as small as grains of rice or as large as a fist, numbered from several to hundreds, much like abdominal miliary tuberculosis. Adhesive intestinal obstruction and complications such as infection and rupture were generally encountered. Multiple surgical operations by areas involved and by stages were indicated over a period of years. Because of the multitude of large and small hydatid cysts, it was not easy to clean them completely and the adjuvant treatment with praziquantel was found to be necessary for control.

No secondary hydatid cysts were found in the case of rupture of liver cysts into the lungs, which might be due to the fact that the liver cysts had been so infected as to become purulent and that the scolices had decayed and could not regenerate; nor were secondary hydatid cysts seen in the case of rupture into biliary tract.

5. Blood-disseminated implantation

Two-three years after removal of endocysts by puncturing in this series of patients with liver cysts, 3 cases were found to have lung cysts. Although it might be due to superinfection, the possibility existed that the scolices entered the liver tissues and then the blood circulation, producing regeneration through blood-disseminated metastasis because of the pressure on liver cysts on performing operation, the contamination of liver tissues by cystic fluid on incising ectocysts on the surface of the liver, and the liver damage caused by over force exerted on wiping out the ectocyst wall.

6. Jaundice

Thirty-nine cases had increased icteric index. Jaundice usually appeared as hydatid
cyst infection infiltrated hepatic cells. Obstructive jaundice occurred in the presence of cyst rupture into hepatic duct or when biliary tract was blocked by daughter cysts or endocyst fragments. Of the 24 cases with rupture into biliary tract seen in this series, 6 were completely obstructive jaundice. Larger hydatid cysts at the site of porta hepatitis caused compression obstructive jaundice. After cyst rupture into hepatic duct, secondary acute pyogenic and obstructive cholangitis was more often observed, cholecystalgia, fever and jaundice being more prominent.

After removal of endocyst the adjacent liver tissues began to move towards the cyst cavity owing to the collapsed ectocyst, thus leaving the hepatic duct to be displaced and distorted and the bile drain to be not free. And the inflammatory infiltration of the residual loculus wall facilitated the rupture of residual end of hepatic duct, which had become atrophique, into the residual loculi of ectocysts. Large amounts of yellow slimy bile and purulent exudate drained from residual loculi after operation generally lasted 1~3 weeks and then gradually decreased.

7. Portal hypertension

Twenty cases had portal hypertension. All had huge liver cysts and a long disease history. Clinical manifestations were mainly enlarged liver and spleen, ascites and varices of abdominal wall.

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References


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