

HYDATID DISEASE OF THE SPLEEN AND PORTAL HYPERTENSION: IS IT A CAUSAL RELATIONSHIP, COMPLICATION OR A COINCIDENTAL FINDING. EVALUATION OF THE MANAGEMENT OPTIONS.

By

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Although, Invasion of the spleen is rare in hydatid disease, the spleen is the third common organ involved by hydatid cysts. Despite there is not a consensus about the way of treatment, it should be surgical to avoid complications. Splenectomy has been the treatment of choice however; attempting to preserve as much splenic tissue has been possible. Segmental portal hypertension due to splenic hydatid disease has been mentioned in the literature in a few case reports. An occasional association between hypersplenism and splenic hydatidosis has been also, reported. The authors hypothesize that there is a possibility of altered portal venous hemodynamics as a cause of lodgment of the Echinococcus embryo into the spleen leading to hydatid cyst formation. Aim of the study: Evaluation of the surgical treatment options for hydatid disease of the spleen. A special emphasis is done on the possible relationship between splenic hydatidosis and portal hypertension and /or hypersplenism.

Patients & methods: *Between 1998-2003 fourteen patients with diagnosed splenic hydatidosis were treated. Patients were fully evaluated by clinical examination, detailed abdominal sonography, and routine laboratory studies. Assessment for portal hemodynamics was done by Doppler ultrasonography and direct measurement of splenic and portal venous pressures via simple manometer at operation. Testing for hypersplenism was done by full blood picture and bone marrow biopsy if needed. Liver histopathological study was performed to exclude or prove co-existing liver cirrhosis. All patients included in the study underwent surgical treatment and percutaneous treatment was the initial option in five of them.*

Results: *The evidence of portal hypertension was found in only three patients (21.4%) (one with hepatosplenic hydatid cysts and two with hydatid cyst of the spleen only). Those three patients had bilharzial liver fibrosis on histopathological study. None of the patients have had hypersplenism. Eight of the patients underwent splenectomy including the three patients with associated bilharzial portal fibrosis. Endocystectomy and omentopexy was performed for five patients and a partial splenectomy was done for another patient. There was no mortality and no major postoperative complications occurred.*

Conclusion: *Hydatid cysts of the spleen do not cause portal hypertension per se and so far, portal hypertension can not explain splenic localization of hydatid disease. Hypersplenism was not found in those patients with splenic hydatid cysts. Splenectomy is the standard treatment of splenic hydatid cysts; however, spleen- preserving surgery is feasible and safe in selected cases. Percutaneous drainage of abscessed splenic lesions must be avoided particularly when hydatid disease is suspected.*

Key words: Spleen hydatidosis, Treatment.

INTRODUCTION

Hydatid disease is a highly endemic parasitic disease caused by the tapeworm *Tenia Echinococcus*. It may affect several organs in the human body and thus represents a

major challenge for the general surgeon. The most commonly involved organ is the liver (75%), followed by the lung (15.4%) and the spleen (5.1%)^(1,2). Uncommon sites reported in the literature include the pancreas, the gallbladder, the kidney, the thyroid gland, the breast, the

pericardium, the supraclavicular region and the thigh (3). Hydatid disease should be included in the differential diagnosis of cystic masses in solid organs or other anatomic sites, especially in endemic countries (1). There is not a consensus about the way of treatment of splenic hydatid cyst. Since there is no effective medical treatment, it should be treated surgically due to the high risk of rupture (4). Surgery remains the treatment of choice, offering a good clinical result and an acceptable recurrence rate (5). The ideal procedure in adulthood is standard splenectomy (4). Although splenectomy is the conventional treatment, there is other spleen preserving treatment options as well, such as partial splenectomy, endo-cystectomy and omentopexy (6-9) or the percutaneous treatment (10).

Portal hypertension as a complication of hepatic hydatidosis is well known in literature (11-13). Therefore hydatidosis should be remembered amongst the causes of portal hypertension in countries where the disease is endemic (14,15). However Segmental portal hypertension due to a splenic Echinococcus cyst has been mentioned in the literature in a few case reports as isolated splenic vein obstruction that may lead to bleeding from gastric varices (16,17). The condition is rare, the varices may be difficult to demonstrate, and therefore the diagnosis often is delayed. Also, There are few case reports in literature regarding the association between splenic hydatid cyst and hypersplenism and its disappearance after splenectomy (18-20). It is hypothesized by the authors that there is a possibility of altered hemodynamics due to portal hypertension of any etiology as a cause of lodgment of the Echinococcus embryo into the spleen leading to hydatid cyst formation. The significance of this whether it is due to hydatid pathology or mere co-incidence needs to be verified as the management strategy for those patients with hydatid disease affecting the spleen may be completely altered and a splenectomy would be a mandatory option.

Aim of the work

The aim of this study was the evaluation of the surgical treatment options performed for hydatid disease of the spleen. A special emphasis is done on the possible relationship between splenic hydatidosis and portal hypertension and /or hypersplenism.

PATIENTS AND METHODS

During the last five years 1998-2003 fourteen patients with diagnosed splenic hydatidosis were treated. Patients were fully evaluated by clinical examination, detailed abdominal sonography, and routine laboratory studies. Pre-treatment diagnosis was accomplished with plain X-ray abdominal films, abdominal ultrasonography, computerized tomography, and serological tests involving Casoni's test and ELISA technique. The last ten patients were assessed preoperatively for portal hypertension and

portal hemodynamics by clinical examination, upper gastro-intestinal endoscopy, and Doppler ultrasonography and intraoperatively by direct measurement of splenic and portal venous pressures via simple manometer at operation. Liver histopathological study was performed by percutaneous needle or operative wedge biopsy to exclude or prove co-existing liver cirrhosis or fibrosis. Assessment for hypersplenism was done by a full blood picture and bone marrow biopsy if needed. All patients included in the study underwent surgical treatment and percutaneous treatment was the initial option in five of them. Pathological study of the splenic tissue removed at surgery was done. Clinical, laboratory, endoscopy, imaging studies, intraoperative findings, and post-treatment follow up data of patients were meticulously analyzed.

Endocystectomy: It involved isolation of the operative field from the rest of the peritoneal cavity. Mobilization of the spleen was not done to avoid cyst rupture. Careful paracentesis of the cyst was done followed by meticulous washing of the cyst cavity using a sterile 10% saline then by povidone- iodine. Removal of the inner germinal layer by a gauze swab was performed. After careful hemostasis of the cyst edges, the omentum was sutured to those edges by 2/0 chromic catgut. Two tube drains were placed one into the residual cavity and one into the left paracolic gutter.

Percutaneous treatment: It included the puncture and free drainage of the cyst fluid under sonographic guidance. After drainage has stopped, alcohol 96% was used as sclerosing agents. The patients were followed up with periodic sonographic examinations.

Duplex Doppler examination: All subjects were studied in the morning after an overnight fasting. All Doppler studies were performed with Hitachi EUB-515 ultrasound scanner with 3.5MHz convex probe and a low - value high-pass filter. Doppler study included qualitative and quantitative assessment. By qualitative assessment we mean assessment of the flow in the portal vein whether it is normal hepatopetal or reversed i.e. hepatofugal. The quantitative assessment included the following hemodynamic parameters:

1-The Mean blood velocity of the portal vein (PMV cm/sec.) was measured according to the standard method (41) in its mid portion on oblique subcostal scan. The axial size of the sample volume was adjusted to encompass the portal vein lumen. The angle between the Doppler beam and the long axis of the vessel was made to be less than 60°.

2-The diameter of the portal vein (in cm) was measured in longitudinal section and cross sectional area (CSA) was measured (in cm²).

3-The congestion index (CI) of the portal vein was

measured from the equation: $CI = CSA \text{ (cm}^2\text{)} / PMV \text{ (cm/sec)}$ expressed in cm/sec.

4-The mean blood velocity in the splenic vein was measured (SMV cm/sec.) at the splenic hilum where the probe was positioned below the left costal margin or in the left intercostal spaces.

The roles applied for the portal vein were also applied for the splenic vein.

1-The CSA of the splenic vein was measured (cm²) in longitudinal section.

2-The splenic venous flow volume (SVF) expressed in ml./min was calculated from the equation: $SVF = SMV \text{ (cm/sec)} \times CSA \text{ (cm}^2\text{)} \times 60$.

To be noticed that each result was a measure of 3-5 measures in all of the previously mentioned parameters.

Statistical analysis:

Results were expressed as mean \pm SD. Differences among groups were evaluated by analysis of variance (ANOVA) using the least significant difference (LSD). The Student's t-test for quantitative parameters.

RESULTS

Fourteen patients with hydatid disease of the spleen were included in the study. Their mean age was 41.6 years (range 16-52 years). They were eight males and six females.

It was the only location of the disease in ten of the patients. It was accompanied by hydatid cysts of the liver in four patients. Most of the patients presented with abdominal discomfort with and without palpable swelling in the left hypochondrium. The diagnosis was incidental finding in two patients (14.2%) during abdominal sonographic examination for symptoms unrelated to the spleen. The splenic hydatid cyst in thirteen patients was unilocular on ultrasonography and it was a multilocular cyst in one patient. The largest diameter of those cysts size varied from 6.5 cm to 15 cm (mean 9.4 cm). The serology findings were positive in nine patients by Casoni's test (64.2%) and in ten out of the 14 patients (71.4%) by ELISA technique. Abdominal sonography and CT were highly suggestive of hydatid cyst of the spleen although the serology was negative in one patient. So, preoperative diagnosis was made in 11 (78.6 %). The pre-operative diagnosis was not conclusive in three of the patients (two with infected cysts and in one patient with serology negative tests and a presumed heterogeneous focal splenic lesion on ultrasonography. Ten patients have had no associated medical or surgical illness. Three patients have had co-existing Bilharzial liver fibrosis and silent second degree esophageal varices and one patient has had gallstones. Two of the three patients with a coincident bilharzial liver have had hydatid cyst of the spleen only and one has had hepatosplenic hydatid disease. The presenting characteristics of the patients are presented in (Table 1). None of the patients have had a clinical or laboratory evidences of hypersplenism neither those with hydatid cyst of the spleen only, hepato-splenic hydatid cysts nor those with associated portal hypertension.

Table (1) pre-treatment data of the patients.

	<i>Patients data</i>	<i>Number of Patients</i>
Age (years)	<20	2
	20-40	7
	>40	5
Sex	Males	8
	Females	6
Clinical presentation	Abdominal discomfort	11
	Upper left quadrant abdominal pain	2
	Mass left. Hypochondrium	4
	Abdominal tenderness & toxemia	2
	Shock	-
X-Ray Chest	Elevated left copula of diaphragm.	2
	Cyst wall calcification	1
Abdominal Ultrasonography & CT	Cyst size:	
	6.5-10cm	10
	>10cm	4
Positive Serology	Associated :	
	Hepatic hydatid	4
	Casoni's	9
	ELISA technique	10

Table (2): Parameters of the Portal Vein.

Parameters.	Normal liver N = 11	Periportal fibrosis N=3
PMV (cm/sec)	18.9 ± 4.28*	13.33 ± 3.02*
CI	0.05 ± .01*	0.14± .01*

* Normal : periportal fibrosis $p < 0.05$.

Table (3) :Parameters of the splenic vein.

Parameters	Normal liver N = 11	Periportal fibrosis N = 3
SMV (cm/sec)	19.59 ± 5.4*	18.81 ± 6.2*
SVF	412 ± 156.6*	1099 ± 772*

*Normal : periportal fibrosis. $P < 0.001$

Table (4): Operative data of patients.

	No. of patients
Site of the cyst at the spleen :	
Lower pole	2
Involving middle part	8
The whole organ	4
Infected cyst	2
Excessive adhesions around the spleen	5
Involvement of diaphragm	2
Inflammation of the tail of pancreas	1
Involvement of Other organs:	
Liver hydatid cyst	4
Cirrhotic liver	3
Gallstones	1

Table (5): Patients management data:

	No. of cases
Preoperative Percutaneous Treatment	5
Type Of Surgery :	
-Elective	12
-urgent	2
The Surgical Procedure	5
-Splenectomy.	1
-Partial splenectomy.	1
-Liver pericystectomy and splenectomy	1
-Splenectomy and cholecystectomy.	1
-Liver endocystectomy and splenectomy	3
-Splenic Endocystectomy and omentopexy.	2
-Hepato-splenic endocystectomy and omentopexy.	

Table (6): Site of the splenic hydatid, associated pathology, and previous PC treatment relative to the surgical procedure performed.

The Surgical procedure	Site of the cyst				Associated pathology				Previous PC treatment
	Lower Pole	Middle zone	Whole organ	Liver cyst	Bilharzial liver	Gall stones	Infection	None	
Splenectomy N=5	1	2	2	-	2	-	2	1	3
Partial Splenectomy N=1	1	-	-	-	-	-	-	1	-
Splenectomy & cholecystectomy N=1	-	-	1	-	-	1	-	-	-
Splenectomy & hepatic pericystectomy N=1	-	-	1	1	-	-	-	-	-
Splenectomy & hepatic endocystectomy N=1	-	1	-	1	1	-	-	-	-
Hepatosplenic endocystectomy and omentopexy N=2	-	2	-	2	-	-	-	-	-
Splenic endocystectomy & omentopexy N=3	-	3	-	-	-	-	-	3	2
Total	2	8	4	4	3	1	2	5	5



Fig (1): Abdominal CT scan cut showing a case with huge hydatid cyst of the spleen [left] and another one with a large multiloculated cyst [right].

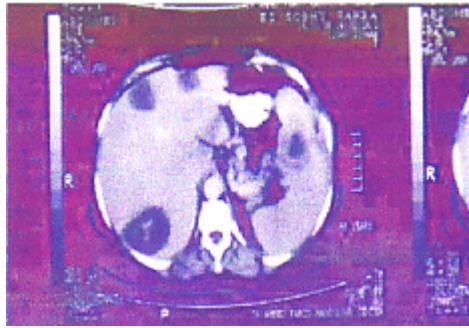


Fig.(2): Abdominal CT scan showing hydatid cyst of the spleen and multiple liver hydatid cysts in one of the cases.

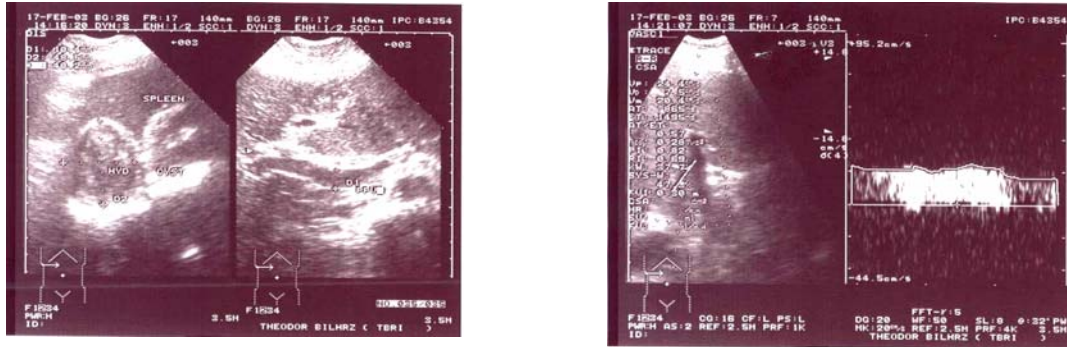


Fig. (3): [Rt.] Abdominal sonography of a case of hydatid cyst of the spleen [Lt.] Doppler sonography of the splenic vein of that case.

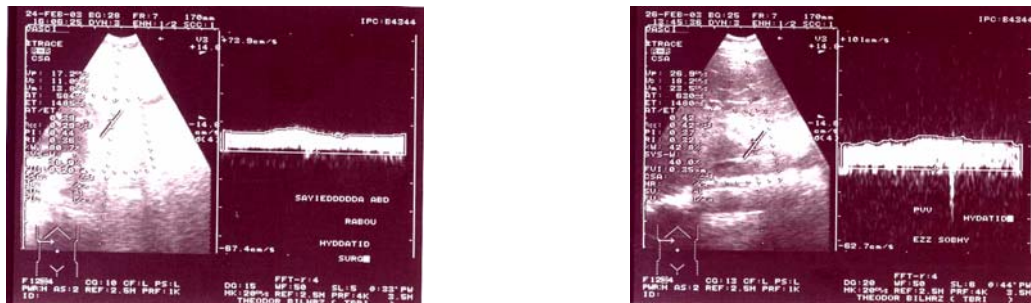


Fig.(4): Doppler sonography of the portal vein of a case with splenic hydatid without portal hypertension [left] and another case with associated bilharzial portal hypertension.

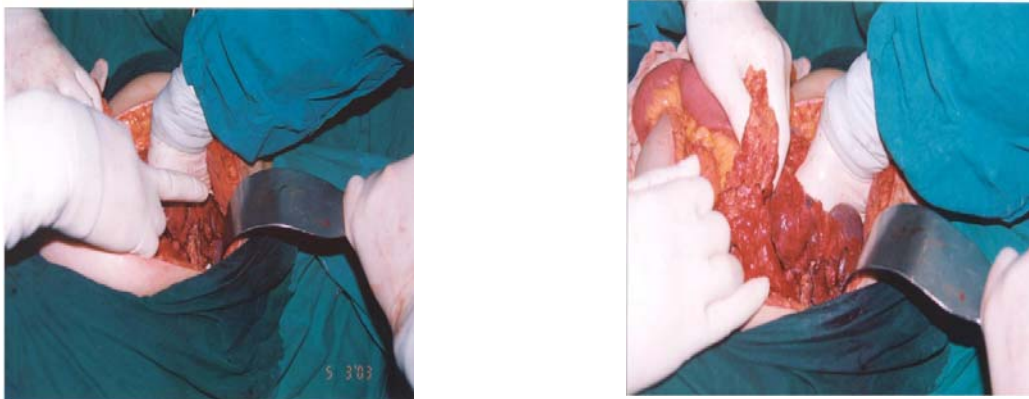


Fig. (5): Intra-operative photography of one case that was presented following the application of percutaneous treatment with infected hydatid cyst showing site of the previous PC puncture [left] and perisplenic collection [right].

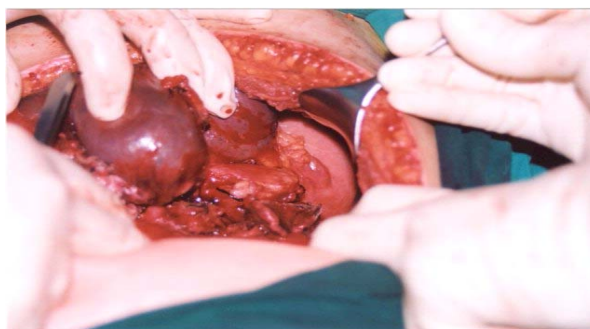


Fig.(6) : Intra-operative photography of the previous case showing inflammation of the pancreatic tail and the site of splenic cyst puncture.

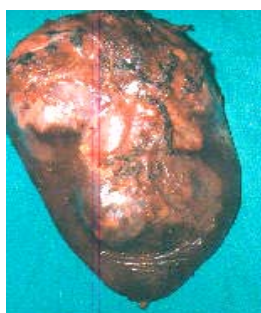


Fig. (7) : post-splenectomy operative specimen of hydatid cyst of the spleen [left] and the opened suppurated hydatid cyst of the spleen [right].

The clinical evidence of portal hypertension in the form of congestive splenomegaly, endoscopically found esophageal varices, ultrasonographically demonstrated patent portosystemic collaterals were found in only three patients (one with hepatosplenic hydatid cysts and two patients with hydatid cyst of the spleen). All those three patients have had coincidental bilharzial liver fibrosis on histopathological study of their liver biopsy. None of the patients with sole hydatid cyst of the spleen or with hepatosplenic hydatid disease without bilharzial liver fibrosis revealed a clinical evidence of portal hypertension. Abdominal Doppler sonographic evaluation detected the presence of spontaneous portosystemic shunts in those patients with bilharzial periportal fibrosis as splenorenal shunts, patent umbilical veins and gastric collaterals. The presence of shunts was associated with that of esophageal varices and splenomegaly. The direction of portal venous flow was normal (hepatopetal) in all patients. The clinical evidence of portal hypertension in those three patients with bilharzial liver and hydatid disease was supported by Doppler sonography findings. (Table 2) shows significant difference between patients with periportal fibrosis and patients with normal liver, as the PMV (=Mean blood velocity of the portal vein) was significantly lower in patients with periportal fibrosis and cirrhosis. The CI

(=congestion index) in the portal vein was significantly higher in patients with periportal fibrosis.

(Table 3) shows the splenic vein hemodynamic changes with significant increase in SVF (=splenic venous flow volume) in patients with periportal fibrosis than patients with normal liver.

Twelve of the patients were operated on electively. Two cases were operated on urgent basis because of a suspected infected hydatid cyst of the spleen in one case and a perisplenic collection in another case. Both cases were presented to surgery following PC treatment. Operative Findings are summarized in (Table 4). The hydatid cysts located at the lower pole of the spleen in two cases otherwise, it occupied the middle part or the whole organ. Encroachment on the diaphragm was present in two cases. Adhesions were to the stomach, left colon, the tail of the pancreas, the diaphragm, and the anterior abdominal wall. Massive adhesions were particularly present in cases that underwent percutaneous treatment before referral to surgery and infected cysts. (Five cases).

Intraoperative direct measurement of portal and splenic venous pressures by simple manometer was

performed in ten cases. It revealed an elevated both portal and splenic venous pressures in the three patients with co-existent bilharzial liver fibrosis (a nearly double fold increment in two and approximately threefold increment in one) but, it was within normal limits in the other seven patients (three with hepatosplenic and four with sole splenic hydatid cysts).

Eight of the patients underwent splenectomy including the three patients with bilharzial portal fibrosis. Endocystectomy and omentopexy was performed for five of the patients. Partial splenectomy was intended for in two patients with the hydatid cyst at the lower pole of the spleen but in one of them who initially had partial splenectomy; the procedure ended up by a splenectomy because of intraoperative hemorrhage from the splenic remnant. Management of associated hydatid cysts of the liver in four of the cases was performed by endocystectomy in three and by pericystectomy in one case. Treatment options performed for the patients are summarized in (Table 5).

Splenectomy indications were associated bilharzial liver with splenomegaly (three), infected cysts (two), whole organ involvement (two) and intra-operative bleeding during attempted partial splenectomy in one of the patients. Cases selected for spleen preserving surgery were without associated pathology other than uncomplicated hydatid disease. Five of the cases were treated by endocystectomy and omentopexy. They have had the cyst less than 10 cm and it was occupying almost the middle zone of the spleen. One case with a small hydatid cyst (6.5 cm) that was located at the lower pole of the spleen has been removed by a partial splenectomy. (Table 6)

Two patients with a suppurated cyst underwent splenectomy that was very difficult in one of those patients due to excessive adhesions. He had iatrogenic diaphragmatic tear that was repaired and the pleural cavity was drained by a chest tube. There was no mortality and no major postoperative complications occurred. Two of the patients developed mild left pleural effusion following splenectomy that responded to conservative treatment. The surgically treated patients were followed up by periodic clinical and sonographic examinations. The median follow-up period was 21 months (range: 3 - 36 months). No recurrence occurred during this follow-up period.

In five of the patients, the percutaneous (PC) treatment was the initial choice of treatment but, surgery was performed eventually (splenectomy three and endocystectomy with omentopexy in two of the patients). The median follow-up of patients treated by the percutaneous approach was four months (range: 6- 10 months). The median diameter of the cysts decreased from 63.0 mm to 33.3 mm. The entire cyst cavity filled with a solid echo pattern in three cysts and partial filling of the cyst

cavity that was shown as a pseudotumor pattern in two cysts. Apart from an urticarial reaction in case, there were no major complications during the follow-up period. Two cases presented late after percutaneous treatment with an infection of the residual cavity. In one case this was accompanied with a perisplenic collection which was persistent seven weeks after a trial of its percutaneous drainage under ultrasonographic guidance. In another case; inflammation of the pancreatic tail was found intraoperatively with massive adhesions around the spleen. Those patients had undergone splenectomy and peritoneal drainage. Another patient with persistent left upper quadrant pain eleven months after PC treatment was operated upon by splenectomy. Another two cases with persistent partial resolution of the hydatid cysts of the spleen (6 and 8 months respectively) after percutaneous treatment were operated upon by endocystectomy and omentopexy. The last approach was chosen in one patient due to technical difficulty to do splenectomy because of the spleen was firmly adhered to its bed, and it was preferred in another patient to preserve the splenic tissue because of his age was sixteen years old.

DISCUSSION

In the medical literature, reports about isolated splenic hydatidosis are quite rare. The spleen is infrequently involved in hydatid disease even in endemic countries. The reported incidence was 2.5 to 5.1% of all cases with hydatid disease ^(1,21,23,28). However, the spleen is the third most commonly involved organ after the liver and the lung ^(1,24). Gomez, et al. 1992 ⁽²⁵⁾ found that among 417 patients with surgically treated abdominal hydatid cyst disease, twenty-eight patients had extrahepatic abdominal cyst disease (6.7%). Most frequent sites were the peritoneum (52.7%), then the spleen (22%) and pelvis (11%). Among a total of 49 patients suffering from hydatid cysts located in various organs other than the liver, there were, 25 had the parasitic cyst in the peritoneal cavity and 10 in the spleen ⁽³⁾. The authors experience with splenic hydatidosis is presented as fourteen cases over a time period of five years; seven (50%) of them were diagnosed in the last year.

The infestation of the spleen usually takes place by arterial route after the parasite has passed through the two other filters, hepatic and pulmonary. The retrograde venous route, which avoids the liver and lung, is also considered. As reported the spleen was either the only location of hydatid disease or there was concomitant liver; or disseminated intraabdominal hydatid disease ^(1, 3). Cystic lesions of the spleen include parasitic and nonparasitic cysts. Parasitic cysts are due almost exclusively to Echinococcal disease representing 50 to 80 per cent of splenic cysts. Nonparasitic cysts are classified as primary or true cysts and pseudocysts. True cysts of the spleen are very rare. Pseudocysts are much more common and may be post-

traumatic, degenerative, or inflammatory. Such conditions can be occasionally observed or detected in emergency in case of splenic rupture (26). Splenic involvement is a rare manifestation of hydatid disease but should be looked for in a systematic way in patients with this diagnosis. The symptoms are not very evident, being characterized by left upper quadrant fullness or discomfort due to splenomegaly. The complications arising from splenic hydatid cysts were infection, rupture in the abdominal cavity and fistulization to the colon or the stomach (21). Splenic hydatid cyst perforating into the diaphragm or left pleural space has been reported (27). There is also, a rare case reports of acute abdomen caused by the spontaneous rupture of a splenic hydatid cyst into the abdominal cavity, causing a massive hemoperitoneum due to accompanying rupture of the spleen which required splenectomy (22). Differential diagnosis is more challenging than usual, but imaging devices combined with immunobiological tests could solve the diagnostic problem. Ultrasonography and axial computed tomography (axial CT) were the most useful diagnostic tools. Plain films well show calcification of the cyst wall. US most clearly demonstrate the hydatid sands in anechoic purely cystic lesions, as well as floating membranes, daughter cysts, and vesicles. . On CT scans, all lesions were of lower attenuation than the surrounding spleen. None of the lesions enhanced after administration of IV contrast material. CT is best for detecting calcification and revealing the internal cystic structure posterior to calcification. Nevertheless, familiarity with imaging findings, especially in patients living in endemic regions, is advantageous in this context (29,30).

There are few case reports of the association between hydatid disease of the spleen and hypersplenism (18-20). However, this seems to be a coincidental finding. None of the study cases showed an evidence of hypersplenism even, the three cases with portal hypertension and bilharzial liver.

Currently splenectomy is the conventional treatment of choice because it demonstrates low morbidity and mortality rates (5, 23). By removing the spleen cysts the risk factor that may be the source for further seeding of parasite hexacanth embryo is eliminated. Gollackner B, et al 2000 (31) recommended radical surgical therapy of abdominal cystic hydatid disease because the rate of recurrence was significantly lower than that after conservative surgery. However, experience suggests that partial cystectomy and omentopexy would be the procedure of choice for the treatment of splenic hydatidosis. The strive for salvage of the spleen whenever possible is fully justified based on updated knowledge of the role it plays in promoting protection against infection. (32). The authors show the technical feasibility and safety of endocystectomy for splenic hydatid cyst and recommend it as the treatment of choice in selected cases. The anatomical conditions must be taken in consideration. Organ preserving operations are indicated in

subcortical localization of the hydatid in the liver, in one of the poles of the spleen and in the kidney when the fibrous capsule is well preserved. Splenectomy or nephrectomy must be carried out in large and giant hydatids of the spleen and kidney localized in the organ or in its hilum and in irreversible derangement of the organ (33). The conservative surgical treatment in attempting to preserve as much splenic tissue is impossible, when there massive involvement of the organ (28) and complicated cysts. The technical feasibility of Conservative surgery for splenic hydatid cyst recommends it as the treatment of choice particularly in children.

Splenic hydatid cyst abscess formation was seen in cases that underwent percutaneous treatment in our study. We presented a surgically proved two cases of abscessed hydatid cyst of the spleen, although their appearance on sonography and computed tomography (CT) imaging methods didn't confirm a diagnosis of abscessed splenic hydatidosis. Although they are not valuable examinations to support the diagnosis, these imaging could define the extent of the disease. Percutaneous drainage of splenic abscessed lesions must be avoided when hydatid disease is suspected (34).

There was no mortality in our patients, and the morbidity rate was acceptable. By using an ultrasonic surgical aspirator it was reported that blood loss was limited that is useful in cases in which it is possible to avoid splenectomy in order to reduce the risk of uncontrolled postoperative infections (35). Also, laparoscopic treatment of hydatid cysts of the spleen was shown to be feasible and safe, offering the advantages of laparoscopic surgery (36-39). Despite these techniques were not attempted yet in our patients, it would be valuable adjutant towards a conservative surgery for splenic hydatid disease.

Long-term results indicate that percutaneous treatment modality of splenic hydatidosis can be effective and safe method and causes no major complications (10). However, the duration of treatment follow-up is long and there is uncertainty about the possible recurrence rate. Simple and small cysts must be selected by combined opinion of the surgeon and the interventionist. Cases in which were subjected to surgery following the percutaneous treatment were more technically difficult than fresh cases.

Splenic vein obstruction due to a solitary Echinococcal splenic cyst which is lodged in a critical location at the hilar region of the spleen with collateral blood flow via the short gastric veins resulting in bleeding gastric fundus varices has been mentioned in literature as a case reports. After splenectomy bleeding did not recur (16, 17). Involvement of the splenic venous outflow tract by pathological lesion at the region of the hilum of the spleen and pancreatic tail has been reported in literature from hemodynamic points of view as a cause localized splenic venous hypertension and

esophageal varices⁽⁴⁰⁾. Thus, clinical features of this entity must be carefully assessed according to the nature of the underlying disease. Splenectomy absolutely eliminates risk from re-bleeding. The flow pattern in the splenic vein has been previously reported in patients with portal hypertension^(41,42) but with no reference to the flow within the intrasplenic venous radicals by using Doppler ultrasound. In this series, a normal flow pattern was maintained in all of the patients. None have had either reversed or dual venous drainage patterns resulting in trans-splenic portosystemic shunts. These abnormal patterns were reported as Doppler signs of portal hypertension. It is recommended that the intrasplenic venous flow pattern should be assessed before surgical intervention involving the spleen⁽⁴³⁾.

CONCLUSION

Hydatid cysts of the spleen do not cause portal hypertension per se, and so far portal hypertension can not explain splenic localization of hydatid disease. Hypersplenism was not found in those patients with splenic hydatid cysts. Splenectomy is the standard treatment of splenic hydatid cysts; however, spleen-preserving surgery is feasible and safe in selected cases. Percutaneous drainage of splenic abscessed lesions must be avoided particularly when hydatid disease is suspected.

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