that, too, was again negative. After the patient's thrombosis and the viral infection that precipitated it had resolved, in the absence of another prothrombotic factor, a decision was made to stop her anticoagulation therapy. Subsequently, the patient remained asymptomatic, with no indicators of thrombotic relapse 6 months after her anticoagulation therapy was suspended.

Splenoportal venous thrombosis (SPVT) usually occurs in the context of an underlying cause which should be investigated and treated. The main causes of thrombosis are divided into systemic causes, such as hereditary thrombophilia, myeloproliferative syndromes, nocturnal paroxystic haemoglobinuria, systemic infections or hormone therapy. Among local causes, tumours and cysts that compress the vein tract may give rise to thrombosis.[3] When SPVT is diagnosed, it is important to search for other more common thrombotic risk factors, since in many cases the cause is multifactorial. However, acute CMV infection itself can cause thrombosis. Although they are uncommon, cases of immunocompetent patients with portal and/or mesenteric vein thrombosis have been reported, in a context of acute CMV infection with no other predisposing cause, as occurred in our case.[2]

In general, viral infections have been linked to a higher risk of thrombosis, since they generate a systemic inflammatory response that activates coagulation through cytokine production. In particular, for CMV, it has been reported that 6.4% of hospitalised patients with acute CMV infection present venous thrombosis, the most common forms being splenic vein thrombosis and splenic infarct, though cases with pulmonary thromboembolism and deep vein thrombosis have also been observed.[4] This predisposition to thrombosis is shared by other viruses belonging to the *Herpesviridae* family (herpes types 1 and 2) since, apart from systemic inflammation, they have an intrinsic procoagulant effect. Their viral structure consists of a covering with expression of procoagulant phospholipids capable of assembling coagulation factors Xa and Va into prothrombinase, so they are able to produce thrombin. In addition, they are expressed on the surface of tissue factor which results in activation of factor X.[5] Therefore, CMV per se acts as an activator of the coagulation cascade, thereby creating a predisposition to formation of thrombi at any level. It has also been observed that acute CMV infection appears to cause a transient elevation in antiphospholipid antibodies. Once the infection resolves, their levels decrease or become negative, thus also creating in this way a predisposition to a prothrombotic state during the infection.[2,4]

In conclusion, we highlight the relationship between CMV and thrombotic events, which in many cases goes unnoticed, and the significance of acute CMV infection in the differential diagnosis of SPVT.

References

1. Zuhair M, Smit GSA, Wallis G, Jabbar F, Smith C, Devleesschauwer B, et al. Estimation of the worldwide seroprevalence of cytomegalovirus: a systematic review and meta-analysis. Rev Med Virol. 2019;29:e2034.
2. Bertoni M, Squizzato A, Foretic M, Zanieri S, Di Natale ME. Cytomegalovirus-associated splanchnic vein thrombosis in immunocompetent patients: a systematic review. Thromb Res. 2018;168:104–13.
3. European Association for the Study of the Liver. EASL clinical practice guidelines: vascular diseases of the liver. J Hepatol. 2016;64:179–202.
4. Justo D, Finn T, Atzmony L, Guy N, Steinvil A. Thrombosis associated with acute cytomegalovirus infection: a meta-analysis. Eur J Intern Med. 2011;22:195–9.
5. Sutherland MR, Raynor CM, Leenknegt H, Wright JF, Pryzdial EL. Coagulation initiated on herpesviruses. Proc Natl Acad Sci U S A. 1997;94:13510–4.

María del Carmen García Gavilán[a,*], Roque Miguel Gálvez Fernández[a], Alfonso del Arco Jiménez[b]

[a] *Servicio de Aparato Digestivo, Hospital Costa del Sol, Marbella, Málaga, Spain*
[b] *Servicio de Medicina Interna, Grupo Enfermedades Infecciosas, Hospital Costa del Sol, Marbella, Málaga, Spain*

* Corresponding author.
E-mail address: marigarciagavilan@hotmail.es (M.d.C. García Gavilán).

Symptomatic giant liver cysts: When and how to treat☆

Cuándo y cómo tratar los quistes hepáticos gigantes sintomáticos

Liver cysts comprise a mixed group of diseases with different aetiologies and incidences but similar clinical signs.

We report four cases of symptomatic giant liver cysts treated by means of laparoscopic deroofing with excellent outcomes.

Case 1. A 53-year-old woman visited the emergency department for pain in her right hypochondrium for a week, initially thought to be nephritic colitis. Ultrasound and magnetic resonance imaging showed a simple cyst measuring 20 cm in liver segment 6. The cyst was hypointense in T1 and showed no wall enhancement following contrast administration.

Case 2. A 77-year-old man sought treatment for bilious vomiting and pain in his right hypochondrium for some months. An abdominal CT scan showed a cyst measuring 14 cm between liver segments 7 and 8, which, given

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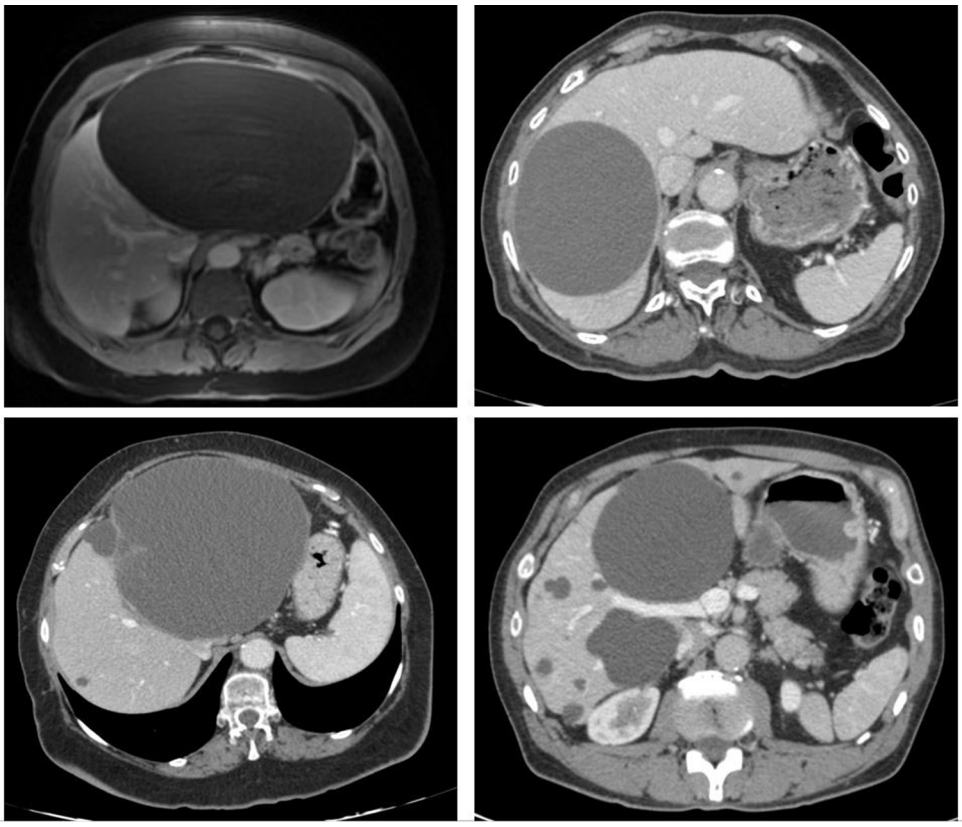

Figure 1 Case 1. Abdominal MRI: liver cyst measuring 20 cm in liver segment 6. It shows no wall enhancement following contrast administration (upper left). Case 2. Abdominal CT: cyst measuring 14 cm between liver segments 7 and 8 (upper right). Case 3. Abdominal CT: cyst measuring 18 cm. The upper portion features septations that raise suspicion of a hydatid cyst (lower left). Case 4. Abdominal CT: multiple liver cysts. The largest has a diameter of 12 cm (lower right).

the density of its contents, raised a differential diagnosis between a simple cyst, cystadenoma and/or biloma. A decision was made to perform exploratory laparoscopy. After the contents of the cyst were aspirated and it was confirmed that they were not of a biliary nature, deroofing was done.

Case 3. A 75-year-old man visited the emergency department due to a feeling of a mass in the epigastrium. An abdominal CT scan revealed a cyst measuring 18 cm that appeared to depend on the left liver lobe, occupying its entirety, with septations in its upper portion that raised suspicion that it might be a hydatid cyst. Serology tests ruled out this possibility and during the intervention it was observed that the cyst contained transformed blood.

Case 4. A 68-year-old man with a diagnosis of asymptomatic polycystic liver disease in follow-up for the past 5 years sought treatment for abdominal pain. A CT scan showed multiple cysts, the largest of which measured 12 cm and was causing displacement of the left portal branch (Fig. 1).

The mean postoperative stay was 3 days. None of the patients experienced complications, and after a 5-year follow-up period none of them showed any recurrence of symptoms.

Both solitary and polycystic lesions grow slowly and are relatively asymptomatic. When they exceed 5 cm, they can cause symptoms due to compression of the hepatic veins, vena cava or bile ducts, or they can cause acute abdominal pain in the event of complications such as rupture, torsion or intracystic haemorrhage.[1]

Imaging tests (CT and/or magnetic resonance imaging) must be done to confirm a diagnosis of a simple cyst. Cystadenoma and cystadenocarcinoma should be ruled out, since laparoscopic fenestration would not be indicated. CT may aid in showing thick and irregular walls, septa, papillary inclusions and loculations. In endemic areas for hydatid disease, such as in our area, hydatid disease should be ruled out by means of serology (90–95% sensitivity), ultrasound and CT (95% specificity).[2]

Simple liver cysts generally require neither treatment nor follow-up, unless they are giant (10—20 cm) and cause symptoms and/or complications. Various techniques have been reported for the treatment of such cysts, depending on their number, location, relationship to other structures and contents: aspiration with or without injection of sclerosing agents[3], argon plasma coagulation in the cyst cavity wall (contraindicated if it is in contact with the biliary tract)[4], deroofing and connection with the peritoneal cavity, cystojejunostomy, complete cyst excision, partial hepatectomy, liver lobectomy (in cases of suspected cyst malignancy) and even liver transplantation (in cases of liver dysfunction due to advanced polycystosis).

At present, laparoscopic deroofing is the technique of choice, as reported by a recent systematic review of 62 studies with 1314 patients[5], with a relapse rate similar to open surgery (25%), but with all the advantages a laparoscopic approach offers.

Laparoscopic deroofing is the technique of choice in symptomatic and/or complicated simple giant liver cysts.

References

1. Antunes H, Braga-Tavares H, Cunha I, Mendes V, Cadilhe A, Matos-Cruz J, et al. [Congenital simple hepatic cyst: a diagnostic and management challenge] Spanish. Gastroenterol Hepatol. 2009;32:92–6, http://dx.doi.org/10.1016/j.gastrohep.2008.09.014.
2. Clancy C, Gibbons C, Ridgway PF. Surgical management of a giant hepatic cyst with suspicious radiological features. Ann R Coll Surg Engl. 2019;101:e76–8, http://dx.doi.org/10.1308/rcsann.2018.0206.
3. Wijnands TF, Görtjes AP, Gevers TJ, Jenniskens SF, Kool LJ, Potthoff A, et al. Efficacy and safety of aspiration sclerotherapy of simple hepatic cysts: a systematic review. AJR Am J Roentgenol. 2017;208:201–7, http://dx.doi.org/10.2214/AJR.16.16130.
4. Manner H. Argon plasma coagulation therapy. Curr Opin Gastroenterol. 2008;24:612–6, http://dx.doi.org/10.1097/MOG.0b013e32830bf825.
5. Bernts LHP, Echternach SG, Kievit W, Rosman C, Drenth JPH. Clinical response after laparoscopic fenestration of symptomatic hepatic cysts: a systematic review and meta-analysis. Surg Endosc. 2019;33:691–704, http://dx.doi.org/10.1007/s00464-018-6490-8.

Marta Allue*, Pilar Palacios, Alfredo Jimenez

Hospital Clínico Universitario Lozano Blesa, Zaragoza, Spain

* Corresponding author.
E-mail address: martitaallue@hotmail.com (M. Allue).

Intestinal obstruction secondary to jejunal intussusception in an adult☆

Obstrucción intestinal secundaria a invaginación yeyunal en el adulto

Intestinal intussusception is an uncommon disease in adults, with an incidence of 1–5%.[1,2] The mean age of onset is 50, and there is a similar distribution between the sexes.[1] Exceptionally, it presents as a bowel obstruction; such instances account for 1% of cases.[1] In 90% of cases, it is linked to adherences or a Meckel's diverticulum, whereas in 10% its aetiology is tumoural. In 50–75% of cases, tumours causing intestinal intussusception are benign. Notable among these, in order of frequency, are lymphoid hyperplasia, lipomas, leiomyomas, haemangiomas and polyps. The most common malignant tumour is a metastatic tumour.[3] The management of such tumours is debated, given the non-specific nature of the signs and symptoms and the discrepancy among complementary tests. Hence, diagnosis tends to be intraoperative.[1,3]

The following is a case report that illustrates the importance of a proper medical history and clinical suspicion in the management of a patient with a bowel obstruction caused by jejunal intestinal intussusception.

A 23-year-old woman with no prior history of note came in with abdominal pain, vomiting for four hours and gradual weight loss in the past year. She had experienced intermittent episodes leading her to seek emergency care on up to six different occasions. On those occasions, her signs and symptoms resolved with observation and treatment with non-steroidal anti-inflammatory drugs (NSAIDs). Examination found her to be in good general condition, with no fever, and haemodynamically stable. Her abdomen was slightly distended, with pain in all quadrants and without any peritonism or guarding. No mass or organomegaly was palpated. Laboratory testing showed leukocytosis (18,000/mm^3) with neutrophilia and elevated PCR (190 mg/dl). A computed tomography (CT) scan revealed a tumour measuring 3 cm × 2 cm × 2 cm in the jejunum and signs of intestinal intussusception (Fig. 1). An emergency laparotomy showed a bowel obstruction secondary to intussusception of jejunal loops, mild signs of loop strangulation and an intraluminal lesion with a lipomatous appearance infiltrating the wall and protruding through the serosa. The intussusception was reduced, and after lukewarm normal saline was administered, the appearance and peristalsis of the bowel loops were restored. A jejunal resection was performed that included the tumour with clear margins and its mesentery, as well as an antiperistaltic mechanical laterolateral anastomosis for bowel transit reconstruction. The patient was discharged on the sixth day after surgery with no associated complications. The pathological workup confirmed the presence of an intraluminal lipoma in the resected segment measuring 4 cm × 3 cm × 3 cm, with no signs of malignancy and no lymphadenopathy. At present, the patient is asymptomatic.

The non-specific clinical presentation of intestinal intussusception in adults represents a diagnostic challenge, as few cases in the literature begin as a bowel obstruction.[1,3] Signs and symptoms include intermittent and recurrent abdominal pain with weight loss and melaena at times, associated with normal complementary test results, which makes a definitive diagnosis difficult and also makes these patients high users of emergency departments.[1] A differential diagnosis is made with acute appendicitis, since the most common location is enteroenteric (34%)[4,5] and the pain

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