Patient was started on antituberculosis therapy. Symptoms gradually improved over the next weeks. After completing the two-month intensive phase of therapy with four drugs, a follow-up endoscopic assessment showed complete remission of the lesion, whereas a significant reduction in lymph nodes sizes was observed in the CT scan (Fig. 1c). The two-drug continuation phase (isoniazid and rifampicin) was uneventfully maintained for four further months, with complete symptom resolution.

Tuberculosis is a rare cause of dysphagia that should be considered in patients from endemic regions with uncertain esophageal lesions.[2,3] In addition, dysphagia may also result from the extrinsic compression of the esophagus by mediastinal or neck lymph nodes (as occurred in the present case) or due to the development of tracheoesophageal fistula. Esophageal ulcer is the most common endoscopic finding, whose appearance is often suggestive of malignancy.[4] However, as mentioned above, other endoscopic findings reported in patients with esophageal tuberculosis include esophageal stenosis, tracheoesophageal fistula or exophytic mass, widening the differential. Therefore, histological and microbiological examination plays a crucial role. Culture positivity for *M. tuberculosis* on tissue samples is uncommon, and the diagnosis is only established by demonstrating clinical, radiological and endoscopic response to anti-tuberculosis treatment, as exemplified by our experience.[2] In the absence of culture confirmation, endoscopic ultrasound-guided fine needle aspiration of the lymph node represents a valuable diagnostic tool, particularly taking into account the need of ruling out alternative Follow-up endoscopic assessment is mandatory in order to confirm endoscopic healing of lesions, since malignancy and esophageal tuberculosis may coexist.[4] Of note, a case of esophageal tuberculosis diagnosed after an esophagectomy performed due to esophageal stricture with histologic features of high-grade dysplasia has been described, stressing the need of considering tuberculosis in the differential diagnosis.[5] In conclusion, the possibility of esophageal ulcer caused by paraesophageal tuberculous lymphadenitis with mucosal involvement should be kept in mind in patients from high-prevalence countries and evidence of esophageal granulomas, even if *M. tuberculosis* is not isolated in tissue cultures. Anti-tuberculosis therapy is usually curative in this uncommon condition.

Conflict of interest

The authors declare that there are no conflicts of interest in relation to this work.

References

1. Sutton FM, Graham DY, Goodgame RW. Infectious esophagitis. Gastrointest Endosc Clin N Am. 1994;4:713–29.
2. Marshal JB. Tuberculosis of the gastrointestinal tract and peritoneum. Am J Gastroenterol. 1993;88:989–99.
3. Perdomo JA, Naomoto Y, Haisa M, Yamatsuji T, Kamikawa Y, Tanaka N. Tuberculosis of the esophagus. Dis Esophagus. 2017;11:72–4.
4. Jain SK, Jain S, Jain M, Yaduvanshi A. Esophageal tuberculosis: is it so rare? Report of 12 cases and review of the literature. Am J Gastroenterol. 2002;97:287–91.
5. Mbiine R, Kabuye R, Lekuya HM, Manyillirah W. Tuberculosis as primary cause for oesophageal stricture: a case report. J Cardiothorac Surg. 2018:743–4.

Santiago de Cossío[a,*], Blas Labrador[b], Ramón Yarza[c], Laura Corbella[d], Mario Fernández-Ruiz[d]

[a] *Servicio de Medicina Interna, Hospital Universitario ''12 de Octubre'', Madrid, Spain*
[b] *Servicio de Medicina de Aparato Digestivo, Hospital Universitario ''12 de Octubre'', Madrid, Spain*
[c] *Servicio de Oncología, Hospital Universitario ''12 de Octubre'', Madrid, Spain*
[d] *Unidad de Enfermedades Infecciosas, Hospital Universitario ''12 de Octubre'', Instituto de Investigación Hospital ''12 de Octubre'' (imas12), Madrid, Spain*

* Corresponding author.
E-mail address: santiagodecossio@gmail.com (S. de Cossío).

https://doi.org/10.1016/j.gastrohep.2019.10.002
0210-5705/

Hepatotoxicity caused by *Garcinia cambogia*☆

Hepatotoxicidad por *Garcinia cambogia*

Acute liver lesions due to the use of herbs and dietary supplements represent between 2 and 20% of cases of acute drug-induced hepatotoxicity in the West, with a progressive increase.[1] Among these compounds, the ones used to lose weight stand out for their frequency.[2] We have detected one case of hepatotoxicity due to one of these ñaturalëdrugs *(Garcinia cambogia [G. cambogia])*, which we believe it is the first reported in Spain.

A 64-year-old female patient, allergic to acetylsalicylic acid, without a medical or surgical history. She does not take routine medication.

She went to the emergency department because of a clinical picture of moderate non-colicky pain, in the epigastrium and right hypochondrium, nausea, vomiting and dark urine which had started 48 h prior to attending the emergency department.

☆ Please cite this article as: Mas Ordeig A, Bordón García N. Hepatotoxicidad por *Garcinia cambogia*. Gastroenterol Hepatol. 2020;43:134–135.

Her vital signs were normal, she was subicteric, and the examination only detected pain on deep palpation in the epigastrium and right hypochondrium, with no signs of peritoneal irritation.

Initial lab work: normal blood count, renal function and ionogram. Total bilirubin 2.9 mg/dl, direct bilirubin 1.8 mg/dl, GOT/GPT 577/720 U/L, ALP/GGT 273/624 U/L, prothrombin time 90%, amylase 60 U/L.

An abdominal CT scan was performed that showed only a distended gallbladder, with no inflammatory changes. The bile duct was not dilated, and the liver and other abdominal organs were normal.

She was admitted to examine the pain. The nausea and vomiting eased quickly. She tolerated an oral diet without problems.

An MRI-cholangiogram was performed that confirmed the normality of the main bile duct, detecting vesicular microlithiasis.

The markers of acute infection due to hepatitis A, B, C, and E viruses were negative, as were anti-cytomegalovirus IgM and Epstein-Barr virus. The autoantibodies (antinuclear, anti-smooth muscle, antimitochondrial and anti-LKM) were also negative. The immunoglobulin dosage was normal.

When we questioned the patient again, she explained that for about 15 days prior to admission she had taken between 1000 and 2000 mg daily of a compound that contained *G. cambogia,* in order to lose weight (body mass index 31). The patient had stopped taking this product coinciding with the onset of symptoms.

The analytical evolution was quickly favourable: at 48 h the bilirubin had dropped to 1.3 mg/dl, GOT/GPT were 223/395 U/L, and ALP/GGT were 230/488 U/L. Four days later, GOT/GPT were 36/143 U/KL, and ALP/GGT 171/373 U/L. The blood count and prothrombin rate remained normal.

As she was fully asymptomatic, the patient was discharged.

At 8 weeks from the first determination no analytical alterations were detected (bilirubin 0.9 mg/dl, GOT/GPT 23/18 U/L, ALP/GGT 82/42 U/L, normal prothrombin). The patient remained totally asymptomatic.

The liver injury was attributed to *G. cambogia* given the negativity of other causes, and the assessment by the CIOMS/RUCAM scale (Danan et al. Int. J. Med. Sci. 2015) which obtained a score of 9 (very probable or certain causality).

G. cambogia is a fruit of a plant that grows in South-east Asia and in Africa. It contains hydroxycitric acid, which is an inhibitor of an enzyme that is involved in the *de novo* biosynthesis of fatty acids and glycogen storage. It also causes appetite suppression. Because of these mechanisms it is used to lose weight. Its use dates back to about twenty years ago, and it was initially considered a product free of side effects. However, since 2005, at least 26 cases of acute hepatotoxicity attributable to this compound have been reported; in the majority the pattern was hepatocellular, while in three it was cholestatic. The symptomatology was nonspecific in all of them (more or less intense abdominal pain, nausea and vomiting, jaundice). The duration of treatment was highly variable, from 2 to 150 days. A total of 24% of patients developed severe acute liver failure that required an urgent liver transplant.[3,4] Fortunately, our patient presented a picture of hepatotoxicity that cured without problems.

In addition to the aforementioned review, the Latin American Hepatotoxicity Registry (Latin DILI Network) detected two more cases.[5] In this study, 10% of cases of liver toxicity caused by drugs were caused by herbs and dietary supplements.

The use of herbs and other products used as n̈aturalm̈edicines is not without risks. In cases of acute liver diseases of undetermined aetiology, the possibility of such causes (plants, herbs, infusions, etc.) should always be investigated. Suspending the intake of them, and not reintroducing them, is essential to avoid further liver damage.

References

1. Ortega-Alonso A, Stephens C, Lucena MI, Andrade RJ. Case characterization, clinical features and risk factors in drug-induced liver injury. Int J Mol Sci. 2016;17:714, doi: 10.3390.
2. Herrera S, Bruguera M. Hepatotoxicidad inducida por el uso de hierbas y medicamentos para perder peso. Gastroenterol Hepatol. 2008;31:447–53.
3. Kothadia JP, Kaminski M, Samant H, Olivera-Martínez M. Hepatotoxicity associated with use of the weight loss supplement *Garcinia cambogia*: a case report and review of the literature. Case Rep Hepatol. 2018. Article ID 6483605.
4. Corey R, Werner KT, Singer A, Moss A, Smith M, Noelting J, et al. Acute liver failure associated with *Garcinia cambogia* use. Ann Hepatol. 2016;15:123–6.
5. Bessone F, Hernández N, Lucena MI, Andrade RJ. and on behalf of the Latin DILI Network (LATINDILIN) and Spanish DILI Registry. The Latin American DILI Registry Experience: a succesful ongoing collaborative strategic initiative. In J Mol Sci. 2016;17:313, doi: 10.3390.

Antoni Mas Ordeig*, Noel Bordón García

Hospital El Pilar, Grupo Quironsalud, Barcelona, Spain

*Corresponding author.
E-mail address: antonimasordeig@gmail.com
(A. Mas Ordeig).

https://doi.org/10.1016/j.gastre.2019.08.010
2444-3824/