management once anti-tuberculosis treatment has been instigated has not been established, given the patient's excellent initial virologic response we decided to continue the former; however, we discontinued boceprevir as its efficacy is reduced on interaction with rifampicin and isoniazid.

Adverse effects on the immune system are virtually unknown with the new direct-acting antiviral drugs and interferon-free therapies, so an increase in infections is not likely. The new antiviral agents also have fewer pharmacological interactions.[8] Nevertheless, for treatments based on sofosbuvir, ledipasvir, daclatasvir, dasabuvir and ombitasvir, the anti-tuberculosis agent rifampicin is contraindicated due to its P-glycoprotein-inducing effect, which significantly reduces plasma drug concentrations.

References

1. Londoño MC, Perelló C, Cabezas J, Cañete N, Lens S, Mariño Z, et al. The addition of a protease inhibitor increases the risk of infections in patients with hepatitis C-related cirrhosis. J Hepatol. 2015;62:311–6.
2. Hametner S, Monticelli F, Kern JM, Schöfl R, Ziachehabi A, Maieron A. Tuberculous sepsis during antiviral HCV triple therapy. J Hepatol. 2013;59:637–8.
3. Saitou Y, Hatazi O, Aonuma H, Ogura S, Yamamoto N, Kobayashi T. Pulmonary tuberculoma in a patient with chronic hepatitis C: a clinical pitfall in the treatment strategy. Intern Med. 2014;53:1669–74.
4. Babudieri S, Soddu A, Murino M, Molicotti P, Muredda AA, Madeddu G, et al. Tuberculosis screening before anti-hepatitis C virus therapy in prisons. Emerg Infect Dis. 2012;18:689–91.
5. Belkahla N, Kchir H, Maamouri N, Ouerghi H, Hariz FB, Chouaib S, et al. Reactivation of tuberculosis during dual therapy with pegylated interferon and ribavirin for chronic hepatitis C. Rev Med Interne. 2010;31:e1–3 [Article in French].
6. Pérez-Elías MJ, García-San Miguel L, González García J, Montes Ramírez ML, Muriel A, Machín-Lázaro JM, et al. Tuberculosis complicating hepatitis c treatment in HIV-infected patients. Clin Infect Dis. 2009;48:e82–5.
7. Killingley B, Bhagani S, Slapak G, Yee TT, Johnson M. Tuberculosis complicating treatment of hepatitis C in an HIV-infected haemophilia A patient. Haemophilia. 2006;12:545–7.
8. Toronto General Hospital. University Health Network. Hepatitis C Drug Information for Healthcare Professional; 2015. Available from: http://www.hcvdruginfo.ca [accessed 12.04.15].

Laura Rodríguez-Martín[a,*], Pedro Linares Torres[a], Marta Aparicio Cabezudo[a], Nereida Fernández-Fernández[a], Francisco Jorquera Plaza[a], José Luis Olcoz Goñi[a], Esperanza Gutiérrez Gutiérrez[b], Eva María Fernández Morán[c]

[a] *Servicio de Aparato Digestivo, Complejo Asistencial Universitario de León, León, Spain*
[b] *Servicio de Farmacia, Complejo Asistencial Universitario de León, León, Spain*
[c] *Servicio de Análisis Clínicos, Complejo Asistencial Universitario de León, León, Spain*

* Corresponding author.
E-mail address: laura.rm.86@gmail.com
(L. Rodríguez-Martín).

Pseudoachalasia in a patient with a history of non-Hodgkin lymphoma☆

Seudoacalasia en paciente con antecedentes de linfoma no Hodgkin

We present the case of a 48-year-old man with a personal history of non-Hodgkin lymphoma (mucosa-associated lymphoid tissue [MALT]-type) with intestinal involvement, diagnosed in 2004 and treated with chemotherapy. The patient has remained in remission since then.

The patient attended our clinic with a 3-month history of progressive dysphagia, chest pain irradiating to the back and weight loss of 10 kg. Findings for a gastroscopy were normal, while a barium swallow test revealed a 3-cm stenosis at the level of the gastro-oesophageal junction with no mass effect, suggesting incipient achalasia. The oesophageal body was normal in calibre, with no dilation or air-fluid level. Four-channel perfusion oesophageal manometry was performed in decubitus. The upper oesophageal sphincter was located at between 25.3 cm and 20 cm from the nasal ala and had a slightly increased resting pressure and normal function. Good pharyngo-oesophageal coordination was observed. The oesophageal body study with at least 10 swallows of 5 mL of water resulted in negative baseline pressure with respect to the fundal pressure. Waves in the body were low pressure and simultaneous. The lower oesophageal sphincter was located at between 44.5 cm and 40.3 cm, had an increased resting pressure (mean baseline pressure 40.6 mmHg) and showed an absence of complete relaxation during the swallow.

The patient progressed within a few days to aphagia with sialorrhoea. Given the rapid evolution of symptoms, a chest-abdominal computed tomography (CT) scan was performed to rule out pseudoachalasia (Fig. 1) with results pointing to a mass in the posterior–inferior mediastinum, obliterating the distal oesophagus and spreading to invade the mediastinal fat and right lung base and to surround the inferior vena cava. The plane of cleavage was lost with the inferior vena cava (although there was no clear infiltration) and also with the diaphragm. These findings suggested possible oesophageal carcinoma extending to surrounding structures or, less likely, pulmonary neoplasm with mediastinal infiltration. Pathological lymphadenopathies were also identified in the mediastinum, right retrocrural space, retroperitoneum (the largest, in the

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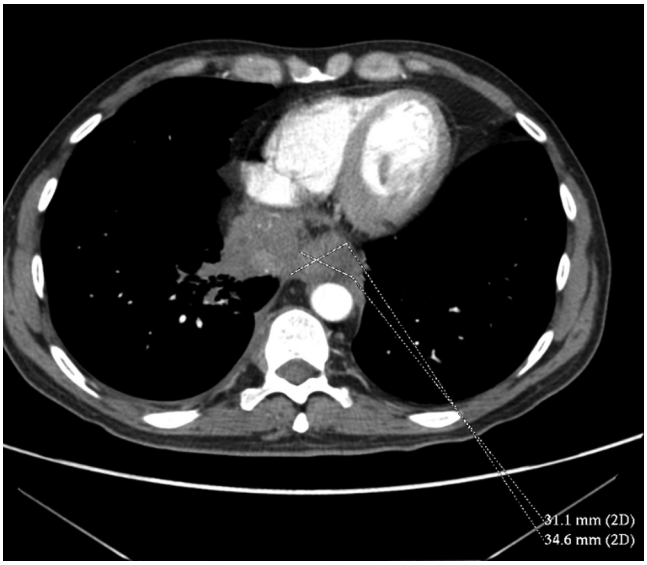

Figure 1 Chest computed tomography: mass in the posterior–inferior mediastinum obliterating the oesophagus and surrounding the inferior vena cava.

coeliac trunk, measuring 28 mm), iliac chains and inguinal regions.

Repeat gastroscopy showed only concentric, punctiform stenosis at the level of the lower oesophageal sphincter that prevented the endoscope from passing. Biopsies taken at this level ruled out tumour infiltration.

Endoscopic ultrasound with aspiration of one of the mediastinal lymphadenopathies was inconclusive due to extraction of inflammatory fibrinous material corresponding to an area of extensive necrosis.

In view of this result, positron emission tomography-computed tomography (PET-CT) was performed, with the finding of an intrathoracic-extrapulmonary mass at the intercostal level of the right upper hemithorax. Biopsies again showed intense necrosis.

Finally, an exploratory laparoscopy was carried out, with resection of the coeliac axis lymphadenopathy. The findings were consistent with marginal zone B-cell non-Hodgkin lymphoma (positive for CD20).

The patient was started on chemotherapy by the haematology department, with rapid resolution of his dysphagia. He is currently in remission.

Pseudoachalasia is a rare oesophageal motor disorder—generally secondary to neoplasm—that presents with symptoms and radiological and manometric signs that are indistinguishable from achalasia.[1] Pseudoachalasia affects approximately 4% of patients who present with signs and symptoms suggestive of achalasia.[2]

Distinguishing between the 2 conditions is crucial. Achalasia, a primary oesophageal motor disorder of unknown cause, consists of non-relaxation of the lower oesophageal sphincter and aperistalsis of the oesophageal body, whereas pseudoachalasia is mostly caused by a neoplasm resulting from mechanical compression or infiltration of the myenteric plexus by malignant cells or even secondary to a paraneoplastic syndrome.

A total of 264 cases of pseudoachalasia have been described in the medical literature, most secondary to neoplasms (53.9% primary, 14.9% secondary), 11.9% post-surgical (oesophageal or gastric surgery) and, exceptionally, 2.6% secondary to paraneoplastic syndrome.

In older patients who present with rapidly progressive dysphagia and weight loss, whose barium study, upper gastrointestinal endoscopy and manometry results are consistent with achalasia, neoplasm should be ruled out as the primary cause of symptoms.[3]

References

1. Liu W, Fackler W, Rice TW, Richter JE, Achkar E, Goldblum JR, et al. The pathogenesis of pseudoachalasia: a clinicopathologic study of 13 cases of a rare entity. Am J Surg Pathol. 2002;26:784–8.
2. Campo SM, Zullo A, Scandavini CM, Frezza B, Cerro P, Balducci G. Pseudoachalasia: a peculiar case report and review of the literature. World J Gastrointest Endosc. 2013;5:450–4.
3. Gockel L, Eckardt VF, Schmitt T, Junginger T. Pseudoachalasia: a case series and analysis of the literature. Scand J Gastroenterol. 2005;40:378–85.

Judit Cotta Rebollo[a,*], Elena Toscano Castilla[a], Marta Lozano Lanagrán[a], Francisca Martín Ocaña[a], Ángeles Cristina Pérez Aísa[a,b], Francisco Fernández Cano[a], Cristina González Artacho[a], Pedro Rosón Rodríguez[a], Francisco Melgarejo Corder[a,c]

[a] *Sevicio de Aparato Digestivo, Hospital Quirón, Málaga, Spain*
[b] *Hospital Costa del Sol, Málaga, Spain*
[c] *Servicio de Aparato Digestivo, Hospital Regional Carlos Haya, Málaga, Spain*

[*] Corresponding author.
E-mail address: juditcotta@gmail.com (J. Cotta Rebollo).