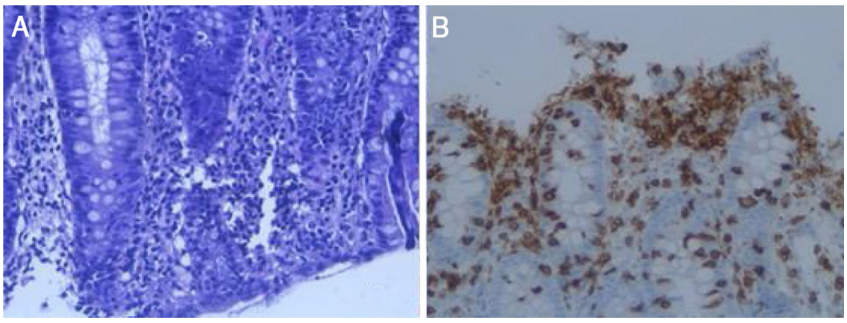


**Figure 1** (A) Haematoxylin-eosin stain showing more than 30 lymphocytes for every 100 enterocytes. (B) Immunohistochemistry stain with CD3 as a T lymphocyte marker.

its main characteristic is the absence of macroscopic abnormalities in the colon mucosa.[1] Hence, a pathology study of the colon mucosa is crucial, since it demonstrates the presence of intraepithelial lymphocytes (≥20 lymphocytes per field) in the colon mucosa.

Regarding aetiology, a genetic susceptibility has been found. In addition, it is reportedly linked to tobacco use and certain drug groups, including proton pump inhibitors, non-steroidal anti-inflammatory drugs and serotonin reuptake inhibitors.[1]

The main treatment consists of stopping the drug that triggered the diarrhoea. In some cases, it is advisable to use loperamide for managing symptoms. If the episode is not managed with these measures, treatment with budesonide can be added. In recent years, the use of mesalamine, cholestyramine and beclometasone has also been proposed, although there is not yet enough evidence in this regard.[2]

To date, only two cases of lymphocytic colitis associated with duloxetine have been reported in the literature,[3,4] which means there is a lack of evidence as to the best treatment for these patients. However, in the previously reported cases, as in the case reported herein, it was not necessary to use budesonide to improve the patient's lymphocytic colitis symptoms. The severity of and the treatment of this pathology may vary, depending on what triggered it. More studies are needed to establish guidelines for the most suitable management of this condition.

## Conflicts of interest

The authors declare that they have no conflicts of interest.

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# Low digestive haemorrhage due to giant-cell lung carcinoma metastasis☆

## Hemorragia digestiva baja por metástasis de carcinoma de pulmón de células gigantes

Lung cancer is the leading cause of cancer death in the world, with a 5-year survival rate of 10–20%. Nearly 50% of patients have metastatic disease at diagnosis, and a 5-year survival rate of less than 5%.[1] Giant cell carcinoma is a rare and poorly-differentiated variant that accounts for 0.1—0.4%[2] of cases of lung cancer.

Primary lung cancer usually metastasises to the brain, liver, adrenal glands, lymph nodes and bones.[1] The gastrointestinal tract is an atypical site of spread (0.5–1.3%), although autopsy studies have found that it could be underdiagnosed (4.7–14%),[1] due to spread through the blood and lymph nodes.

We present the case of an 81-year-old man, a former smoker who quit smoking 12 years earlier, with a history of left lower lobectomy plus lymphadenectomy for giant cell carcinoma in April 2018 (pT1bN0 with clear resection margins), with no remote disease observed in studies of spread (M0). During follow-up, no signs suggestive of relapse were

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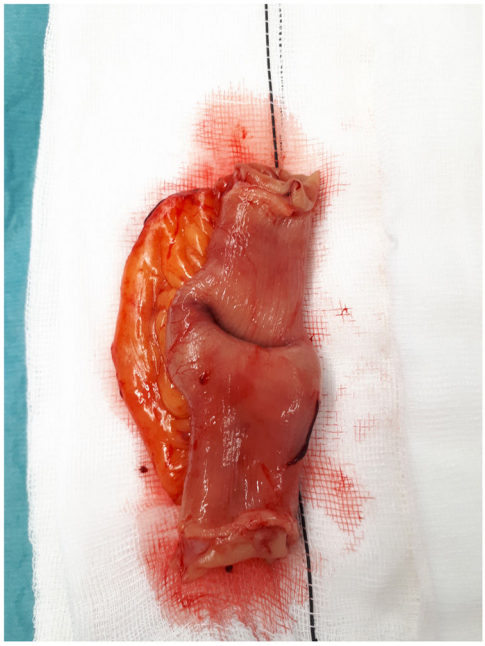

**Figure 1** Macroscopic image of the surgical specimen: a hard, umbilicated endoluminal tumour measuring around 7 cm × 7 cm in the proximal ileum.

seen, nor were there any abnormalities in tumour markers. The only finding worthy of note was a gradual development of anaemia.

The patient was admitted in May 2019, having been transferred from another hospital, for signs and symptoms of low gastrointestinal bleeding and several syncopal episodes, with a haemoglobin level as low as 6.39 mg/dl. He did not present any abdominal signs or symptoms, and examination revealed no findings of interest. The patient underwent a colonoscopy and a gastroscopy, which showed no lesions, and the study was completed with a capsule endoscopy which identified two small vascular lesions of an angiodysplastic nature: one in the proximal duodenum and the other in the proximal-medial jejunum. With these findings, an endoscopy was performed for purposes of haemostasis of these lesions. Given the persistence of the patient's signs and symptoms, a decision was made to perform thoracoabdominal computed tomography. This found a hypervascular mass measuring 25 mm in the mesentery of a loop of terminal ileum infiltration towards the intestinal lumen. In view of the findings of the computed tomography scan, a decision was made to perform an emergency exploratory laparoscopy. This revealed a hard, umbilicated endoluminal tumour measuring 7 cm × 7 cm in the proximal ileum (Fig. 1). A total of 20 cm of proximal ileum were then resected with end-to-end manual anastomosis. The patient had no complications in the postoperative period and was discharged on day 7 after the procedure.

The pathology study identified the tumour as giant cell carcinoma metastasis (positive for vimentin, mixed CK, CK7 and TTF, with no EGFR, BRAF or K-RAS mutation detected) consistent with metastatic lung cancer relapse. The patient was referred back to his oncologist, who started chemotherapy with paclitaxel and carboplatin. Radiological follow-up identified multiple bone lesions consistent with bone M1 lesions.

As already mentioned, metastasis of lung cancer to the gastrointestinal tract is uncommon; the main site of spread of this type of cancer is the small intestine (8.1%), followed by the stomach (5.1%) and the large intestine (4.1%).[1] The subtypes that most often metastasise to the small intestine are squamous cell, large cell and pleomorphic (which includes the giant cell subtype).

It is usually diagnosed by X-ray, ultrasound or computed tomography (diagnostic method of choice). Computed tomography angiography and capsule endoscopy are also useful in cases of gastrointestinal haemorrhage with no obstruction.[3,4] The differential diagnosis between a primary and a metastatic tumour can be difficult. Hence, immunohistochemistry plays a crucial role.[3]

Treatment must be focused in the context of metastatic lung cancer, although surgical resection of the bowel may be required in case of perforation, bowel obstruction or bleeding. Chemotherapy, as a neoadjuvant treatment, depends on the histopathology of the tumour and the condition of the patient,[3] as treatment in itself may increase the risk of perforation or bleeding.[5]

Despite surgical resection of intestinal metastases, around 50–60% of patients experience metastatic relapse, and one- and three-year survival rates are 44.4% and 33.3%, respectively.[5]

In conclusion, in patients with a history of lung cancer, the possibility of metastasis to the small bowel in a context of bleeding, obstruction or perforation must be included in the diagnostic algorithm.

In these cases, surgical resection is an option that contributes to solving the urgent condition as well as establishing a diagnosis and planning an oncological treatment strategy.

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# Portal and mesenteric thrombosis secondary to acute cytomegalovirus infection in an immunocompetent patient☆

## Trombosis portal y mesentérica secundaria a infección aguda por citomegalovirus en paciente inmunocompetente

Cytomegalovirus (CMV) infection is very common in the general population, with a positive IgG-based seroprevalence of 83%.[1] In immunocompetent patients, it is usually asymptomatic or causes flu-like signs and symptoms. Its most common complications are acute hepatitis and splenomegaly and there may be a risk of spleen rupture. Mesenteric vein thrombosis has been reported as a rare complication of acute CMV infection.[2]

We present the case of a 55-year-old woman who was admitted for asthenia, headache and epigastric discomfort (distension). On admission, laboratory testing showed 11,650 leukocytes/μl (63.2% lymphocytes/μl), C-reactive protein 21.2 mg/l, aspartate aminotransferase 119 U/l, alanine aminotransferase 120 U/l, gamma-glutamyl transferase 113 U/l, alkaline phosphatase 179 U/l, total bilirubin 0.5 mg/dl and lactate dehydrogenase 357 U/l. On admission, an abdominal ultrasound was performed which showed hyperechogenicity of the portal vessels suggestive of oedema, no signal with Doppler ultrasound in the portal vein and hyperechogenic matter in the superior mesenteric vein, consistent with thrombosis. The study was completed with computed tomography (CT) of the abdomen, which showed a filling defect in the right intrahepatic portal vein and inside the superior mesenteric vein consistent with thrombosis. Following the CT results, treatment was started with enoxaparin at a dose of 1.5 mg/kg/day (Figs. 1 and 2). A hypercoagulability study, with determination of cardiolipin antibodies, anti-β2-glycoprotein, lupus anticoagulant, activated C protein resistance, proteins C and S, homocysteine, functional antithrombin, factor V Leyden mutation and G20210A mutation, was negative. An autoimmunity study, a haematological study with a smear, proteinogram, ß2-microglobulin and JAK2 mutation were negative, and serological test for viral forms of hepatitis and infectious causes including *Brucella, Salmonella, Rickettsia conorii, Leishmania, Coxiella burnetii,* Epstein–Barr virus and CMV was only positive for the CMV IgM. The study was completed with a gastroscopy and magnetic resonance imaging of the head, which yielded no findings. Given the suspicion of CMV as the main cause of the thrombosis, polymerase chain reaction (PCR) testing for CMV in blood was performed, which measured 16,600 copies/mL, thus confirming acute CMV infection. Given the aggressive form of presentation, a decision was made jointly with the infectious disease group to administer valganciclovir 900 mg/12 h for 14 days. The patient responded well, with an improvement in her symptoms after several days of antiviral treatment. Following 6 months of anticoagulation therapy with enoxaparin, a follow-up CT scan of the abdomen showed full resolution of the patient's venous thrombosis. Subsequently, the hypercoagulability study was repeated and was again negative. PCR testing for CMV in blood was also repeated and

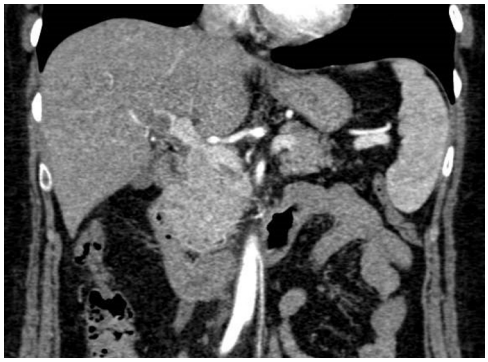

**Figure 1** Coronal slice from the abdominal CT scan showing the filling defect in the right portal vein.

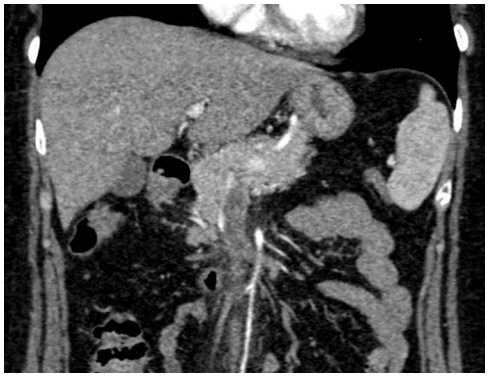

**Figure 2** Coronal slice from the abdominal CT scan showing the filling defect in the splenic vein.

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