Interesting images

Inferior mesenteric venous thrombosis and $^{18}$F-FDG PET/CT

Trombosis venosa mesentérica inferior y $^{18}$F-FDG PET/TC

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An 89-year-old male, with rectal adenocarcinoma, stage II A, treated with transanal resection, end anastomosis and radiotherapy is reported. At the last follow-up, he shows significant elevation of CEA serum levels, changes in bowel and an extraparietal rectal nodule. $^{18}$F-FDG PET/CT scan showed a tumor local recurrence, pelvic lymph node involvement, peritoneal metastatic implants and liver metastasis.

The inferior mesenteric vein and its branches, up to splenoportal axis, have close contact with the local recurrence, showing intraluminal hypermetabolic content in its entire length, compatible with thrombosis by tumor invasion. Histopathologic analysis of rectal mass discovered sigma cancer (Figs. 1 and 2).

Mesenteric vein thrombosis (MVT) causes between 5 and 15% of cases mesenteric ischemia and mainly affects the

Fig. 1. From left to right, coronal images of CT, PET, fused PET/CT and maximum intensity projection, showing increased uptake of FDG following a linear path that coincides in the inferior mesenteric vein, compatible with massive thrombosis. Linear hypermetabolic foci are observed perpendicular to the main path of the vein at its caudal half, coinciding with compromised venous tributaries branches in the area. The maximum intensity projection shows a hypermetabolic focus in segment VIII hepatic dome, which is not apparent on CT lesion, which was interpreted as metastasis.

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superior mesenteric vein. Currently it is considered that most of patients (60%) with MVT have a secondary etiology; a previous abdominal surgery and hypercoagulable states have been identified as the conditions most commonly associated.¹

In this context, pathophysiological mechanism is the direct extension of the rectal tumor cells through the upper rectal hemorrhoidal veins to splenoportal mesenteric axis. The PET/CT diagnostic criteria for a venous thrombosis tumor include focal or linear uptake along the affected vessel.² The main treatment is anticoagulation therapy and, when there are signs of intestinal ischemia, surgery associated with early anticoagulation. The mortality reported in the literature is 20–50%.³

References