Table 1 Analytical values before and during admission. A clear predominance of cytolysis (*R*-ratio >5) can be seen, consistent with the alterations described in treatment with sunitinib.

Parameter	10 days before admission	Upon admission	One day after admission	Two days after admission
Bilirubin (md/dl)	0.79	7.13	7.51	11.47
GGT (U/l)	14	110	114	119
ALP	105	230	243	265
AST (U/l)	29	563	N/A	835
ALT (U/l)	19	853	945	1082
R-ratio	0.50	10.30	10.80	11.34
Platelets ($\times 10^3/\mu l$)	377	45	41	19
TP (%)	107	35	29	Incalculable

References

1. Ibrahim EM, Kazkaz GA, Abouelkhair KM, Bayer AM, Elmasri OA. Sunitinib adverse events in metastatic renal cell carcinoma: a meta-analysis. Int J Clin Oncol. 2013;18:1060–9, http://dx.doi.org/10.1007/s10147-012-0497-2.
2. Weise AM, Liu CY, Shields AF. Fatal liver failure in a patient on acetaminophen treated with sunitinib malate and levothyroxine. Ann Pharmacother. 2009;43:761–6, http://dx.doi.org/10.1345/aph.1L528.
3. Taran A, Ignatov A, Smith B, Costa SD, Bischoff J. Acute hepatic failure following monotherapy with sunitinib for ovarian cáncer. Cancer Chemother Pharmacol. 2009;63:971–2, http://dx.doi.org/10.1007/s00280-008-0814-7.
4. Naranjo CA, Busto U, Sellers EM, Sandor P, Ruiz I, Roberts EA, et al. A method for estimating the probability of adverse drugs reactions. Clinical Clin Pharmacol Ther. 1981;30:239–45, http://dx.doi.org/10.1038/clpt.1981.154.
5. Sunitinib. LiverTox: clinical and research information on drug-induced liver injury [Internet]. Bethesda (MD): National Institute of Diabetes and Digestive and Kidney Diseases; 2012. Available from: https://www.ncbi.nlm.nih.gov/books/NBK548118/ [updated 27.06.18].

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Adrenal uptake in PET/CT in a patient with pancreatic neoplasm: Not always metastasis☆

Captación adrenal en PET/TC en paciente con neoplasia de páncreas: no siempre metástasis

A 62-year-old woman with a history of hysterectomy and oophorectomy for uterine cancer, left parathyroidectomy for Warthin's tumour and bladder neoplasia treated with transurethral resection (TUR), complaining of back pain.

Abdominal CT (Fig. 1A/B) and abdominal MRI (Fig. 1C/D/E) were performed, showing a 29-mm left adrenal nodule, well defined, hypointense and homogeneous in all sequences, compatible with adenoma and an ampullary lesion of 15 mm, solid hypointense in all sequences, compatible with ampullary tumour.

Both a liver function test and a hormonal study were normal, ruling out a hyperfunctioning adrenal lesion (dexamethasone suppression test, catecholamines, aldosterone and baseline renin activity).

Endoscopic ultrasound (EUS): ampullary adenoma with growth in the bile duct (N0). Biopsy: signet ring cell adenocarcinoma inside villous adenoma.

In the presence of a left adrenal lesion in a patient with confirmed ampullary malignancy, PET/CT was performed, which showed focal left adrenal uptake (SUVmax 4.6), without ampullary uptake. Fine-needle aspiration biopsy (FNAB) was performed for the adrenal lesion, with unsatisfactory cytology results.

Faced with focal adrenal uptake on PET/CT, without histological confirmation of metastasis in a patient suitable for surgical intervention, a left adrenalectomy was performed with a negative intraoperative study for malignancy, so the proposed oncological surgery was continued and a cephalic pancreatoduodenectomy was performed.

Histological report: left adrenal adenoma and ampullary signet ring cell adenocarcinoma in ampullary villous adenoma, with free margins (pT1aN0).

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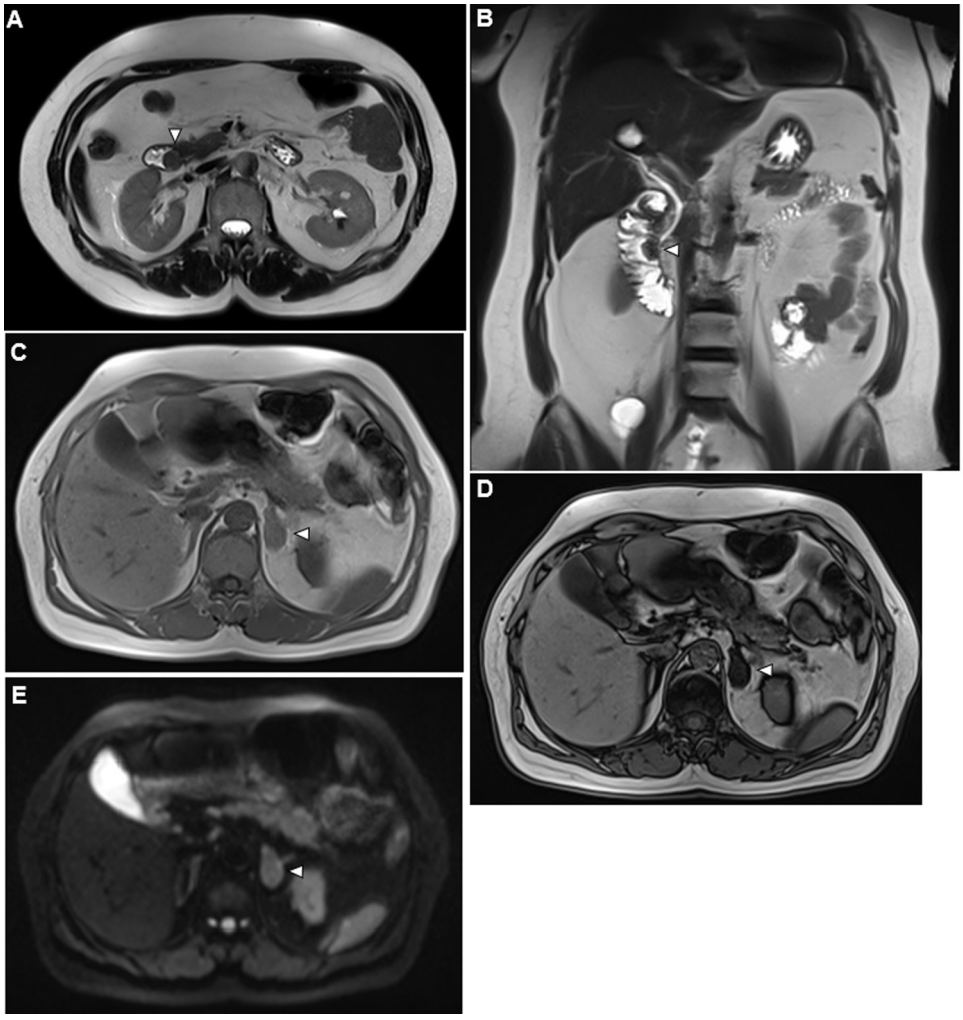

Figure 1 Axial (A) and coronal (B) T2 MRI shows a solid mass protruding inside the second duodenal portion at the location of the ampulla of Vater (arrowhead). This image is compatible with an ampullary tumour. MRI (C-D-E). A left adrenal lesion (arrowhead) is observed, showing signal drop between in-phase (C) and out-of-phase (D) images weighted in T1. This absence of signal indicates the presence of intracellular fat, considered diagnostic of adrenal adenoma. No restricted diffusion was demonstrated (E).

Up to 50% of all incidental adrenal masses in patients with known malignancy would harbour metastases.[1] PET/CT is the standard in patients with solitary adrenal lesion and known malignancy, because most adrenal adenomas have low metabolic activity, while malignant adrenal lesions have high metabolic activity,[1,2] with sensitivity and specificity of 97% and 91%, respectively.

According to the literature, focal adrenal uptake on PET/CT reaches a false positive rate of up to 20%.[1,2] Currently, and with these data, there is no consensus on the best measure (adrenal SUVmax versus the adrenal-to-liver SUV ratio), nor on the SUVmax cut-off point, to distinguish between malignant and benign adrenal lesions.[3]

More studies are needed on the incidental findings of PET/CT in patients with pancreatic cancer. Its use should be considered in patients at high risk of metastasis. There is no specific literature focusing on adrenal metastases of cancer from benign prostatic hyperplasia (BPH).

Therefore, a focal adrenal uptake on PET/CT is highly suggestive of malignancy and more diagnostic studies are necessary. Adrenal biopsy should be considered,[1,2] and in the event of inconclusive results, the risk and advantages of cancer surgery should be included in the decision-making and preoperative counselling with the patient and the treating oncologist.

Conflicts of interest

The authors declare that they have no conflicts of interest.

References

1. Lang BH, Cowling BJ, Li JY, Wong KP, Wan KY. High false positivity in positron emission tomography is a potential diagnostic pitfall in patients with suspected adrenal metastasis. World J Surg. 2015;39:1902–8, http://dx.doi.org/10.1007/s00268-015-3035-3. PubMed PMID: 25809060.
2. Kuritzkes B, Parikh M, Melamed J, Hindman N, Pachter HL. False-positive rate of positron emission tomography/computed tomography for presumed solitary metastatic

adrenal disease in patients with known malignancy. Ann Surg Oncol. 2015;22:437–40, http://dx.doi.org/10.1245/s10434-014-4031-9. PubMed PMID: 25160737.
3. Watanabe H, Kanematsu M, Goshima S, Kondo H, Kawada H, Noda Y, et al. Adrenal-to-liver SUV ratio is the best parameter for differentiation of adrenal metastases from adenomas using 18F-FDG PET/CT. Ann Nucl Med. 2013;27:648–53, http://dx.doi.org/10.1007/s12149-013-0730-8. PubMed PMID: 23625579.

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DRESS syndrome and drug-induced liver injury owing to antituberculous treatment

Síndrome de DRESS y hepatotoxicidad por tuberculostáticos

Drug-induced liver injury by antituberculous drugs (anti-TBC) is an idiosyncratic drug reaction, more often associated with isoniazid. Clinical manifestations may appear between 1 and 12 weeks after the onset of treatment, although they usually do between 2nd and 4th week.[1,2]

A severe form of drug toxicity is DRESS syndrome (Drug Reaction with Eosinophilia and Systemic Symtoms). It is characterized by fever, rash, eosinophilia and in many cases hepatotoxicity. This is a serious and potentially fatal reaction, with a mortality rate of around 20% if the responsible drug is not discontinuated, so early diagnosis is of utmost importance. Often it is not the skin involvement, but the presence of hepatotoxicity is a crucial factor in prognosis.[2,3]

A 69-year-old male presented to Emergency Room due to fever and rash. The physical examination showed jaundice and a cutaneous maculopapular rash with facial edema. Laboratory tests identified hyperbilirubinemia (total bilirubin 10.32 mg/dl, conjugated bilirubin 9.72 mg/dl), elevated transaminases (ALT 2095 U/L, AST 1208 U/L, GGT 364 U/L and alkaline phosphatase 566 U/L), leukocytosis with eosinophilia (WBC of 13,800/μL with 17.6% eosinophils) and coagulopathy (INR 2.31. Abdominal ultrasound showed no relevant findings.

The patient had recently been diagnosed with tuberculosis so he had started first-line anti-TBC treatment with rifampicin, isoniazid, ethambutol and pyrazinamide five weeks before the onset of the clinical picture. The search for the following alternative causes was negative: viral infections (HAV, HBV, HCV, HEV, EBV, CMV), alcohol-related liver disease, autoantibody titers, serum IgG, ceruloplasmin levels and 24 urine cooper. Liver biopsy was considered, but finally it was not done because the condition presented with typical manifestations, alternative causes were ruled out and the patient evolved favorably.

Due to the temporal relationship between drug therapy and the appearance of the clinical picture and the exclusion of other causes of liver disease, the diagnosis of DRESS syndrome with acute drug-induced liver injury (DILI) with hepatocellular pattern was established. It was decided to stop treatment and prednisolone was prescribed in descending pattern.

The outcome was favorable with 8 weeks of treatment normalizing liver function and transaminases levels. However, upon restarting a 2nd and 3rd line of anti-TBC treatment, our patient presented two successive episodes the second episode presented cutaneous manifestations with mild asymptomatic liver enzyme elevation and in the third episode he suffered facial edema and acute DILI with hepatocellular pattern requiring new hospital admission and systemic steroids treatment (Table 1).

During the follow-up period, the tuberculous disease worsened; with pulmonary, pleural, lymph node and peritoneal progression and the appearance of a chest wall abscess. Therefore, it was decided to immediately restart a new 4th-line anti-TB therapy, avoiding the potentially hepatotoxic drugs. Such therapy was based on streptomycin, moxifloxacin, ethambutol and linezolid. Surgical drainage of the chest wall abscess was also performed. The patient completed 12 months of anti-TB treatment, achieving complete clinical and bacteriological cure of tuberculosis disease, without signs of liver damage during this period.

Several cases of Drug-induced liver injury and DRESS syndrome induced by anti-TBC drugs have been reported in the literature. The main associated drugs are the first-line anti-TBC drugs; isoniazid, rifampin and pyrazinamide, especially when used in association as in the case we present.[1,2,4] For the diagnosis of drug-induced liver injury and/or DRESS syndrome, clinical suspicion is very important, together with a comprehensive anamnesis of drug use, which will allow us to consider the temporal relations between drug intake and the development of the clinical picture The exclusion of alternative causes of liver damage is of utmost importance.[1,4]

In the case we report, due to the successive changes of anti-TBC treatment, it is not possible to define with certainty which drugs are responsible for the liver damage episodes. Rechallenge tests were not performed due