URINARY ASCITES AFTER TRANSURETHRAL RESECTION IN A CIRRHOTIC PATIENT

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Abstract

Urinary ascites is a rare entity, usually secondary to iatrogenic or traumatic bladder perforation. Given the infrequency of the entity, it is a diagnosis for which high clinical suspicion is required, and when a patient presents with ascites, it is common to attribute it to other causes. Especially in cirrhotic patients, where ascites is most often secondary to portal hypertension, although this is not always the cause. It is therefore important to always make an appropriate differential diagnosis, assessing the patient's history, the characteristics of the ascitic fluid and the response to treatment. We present the case of a patient with cirrhosis, with a history of transurethral resection (TUR) of a bladder tumour, which was initially classified as oedema-ascites decompensation and after completing the diagnostic process was attributed to urinary ascites.

Keywords: uroperitoneum, ascites, cirrhosis.

Introduction

Ascites is a clinical situation of special frequency and relevance in digestive pathology, being usually related, in our area, to cirrhosis or digestive tumours. However, any patient who begins with ascites must undergo an appropriate diagnostic process, evaluating and ruling out the different causes of ascites. Among the causes of ascites, an extremely infrequent one that can be observed in patients who have undergone bladder or laparoscopic surgery is ascites of urinary origin due to perforation of the bladder dome, which constitutes the peritoneal portion of the urinary bladder. Diagnosis is based on ascitic fluid analysis, surgical history and computerised tomography (CT). Treatment is based on bladder catheterisation and management of the perforation, which is usually carried out conservatively.

Clinical case

We report a 92-year-old patient, with a history of Child A5 cirrhosis, who underwent transurethral resection of the bladder as treatment for bladder neoplasia. The patient reported progressive abdominal distension, without associated peripheral edema, with evidence of grade 2 ascites and oliguria. An abdominal ultrasound scan showed a heterogeneous liver ultrastructure with blunt and irregular borders compatible with chronic liver disease and moderate ascites. Laboratory tests showed creatinine of 6 mg/dl, CRP of 136, normal liver markers and normal coagulation parameters. A diagnostic paracentesis was performed with leukocytes 2450 with 65% PMN, creatinine

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in ascitic fluid of 2.4 mg/dL, urea 89 mg/dL and albumin 1.6 g/dL. With a serum-ascitic albumin gradient of 1.5. A diagnosis of first edema-ascitic decompensation and spontaneous bacterial peritonitis (SBP) was made. For which, treatment was started with albumin at a dose of 1.5 g/kg on the first day and 1g/kg on the third day, together with antibiotic coverage with 3rd generation cephalosporins. Given the acute renal failure, diuretics were not initiated.

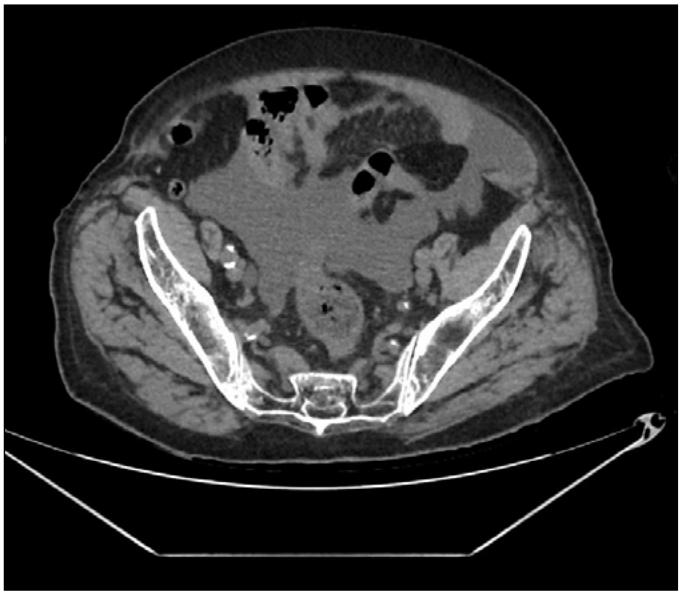

Figure 1. ***This image shows an axial CT scan of the abdomen, without contrast, showing free fluid corresponding to ascites.***

However, after indicating bladder catheterisation for diuresis quantification, there was an evident decrease in ascites and a rapid improvement in renal function to normal figures. After this, and given the history of TUR, urology was contacted and a Uro-CT scan was requested. This test revealed a perforation in the bladder dome and intraperitoneal contrast material, and the case was classified as urinary ascites secondary to this perforation. Given that the patient was stable and a progressive resolution of the ascites was observed with bladder catheterisation, conservative treatment of the perforation was decided upon. After maintaining bladder catheterisation and antibiotic coverage for 3 weeks, and in view of the patient's clear improvement, a new control Uro-CT scan was performed, which confirmed closure of the perforation, after which the patient was discharged.

Discussion

Ascites consists of the presence of serous fluid in the peritoneal cavity, the causes of this condition are varied, one of

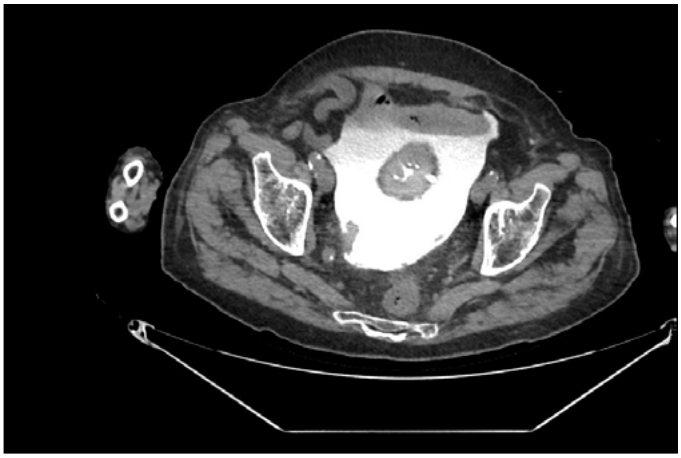

Figure 2. ***CT abdomen and pelvis, with contrast in the excretory phase, showing contrast leakage secondary to bladder perforation.***

the most frequent being portal hypertension, usually linked to hepatic cirrhosis (up to 75% of the causes of ascites). There are also other common causes such as those related to a decrease in plasma oncotic pressure, such as heart failure or nephrotic syndrome[1].There are other causes such as tuberculosis, secondary to peritoneal carcinomatosis or pancreatic carcinomatosis. There may also be a large group of rare causes including urinary ascites.

Urinary ascites is a rare diagnosis characterised by the presence of ascites attributable to bladder perforation, usually following a therapeutic procedure on the urinary bladder[2]. Elevated urea and creatinine levels are usually found in ascitic fluid analysis. In addition, an even higher elevation of creatinine is often found in the blood, and diuresis is reduced due to the passage of urine into the peritoneal space. The higher elevation of plasma creatinine is explained by the fact that the peritoneum allows creatinine to pass from the abdominal cavity into the plasma[3]. A very characteristic feature is the decrease in plasma creatinine levels and resolution of ascites after bladder catheterisation. A key factor in its diagnostic evaluation is the history of an invasive procedure on the urinary bladder and late-phase contrast-enhanced CT scan, which allows the existence of bladder perforation to be detected. Treatment consists of reducing the passage of urine into the peritoneal cavity by bladder catheterisation and closure of the defect which, depending on its characteristics and the patient's clinical condition, can be managed conservatively or by surgical intervention.

In our case, despite the initial diagnosis of oedema-ascitic decompensation, the non-advanced stage of cirrhosis, the absence of hyponatraemia or plateletopenia, the

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characteristics of the fluid and the decrease in ascites after catheterisation led us to suspect an alternative diagnosis, which was confirmed after performing the Uro-CT scan. The particularity of this case probably lies in the fact that it is a patient with hepatic cirrhosis, which, being the most frequent cause of ascites, does not, from the outset, lead us to orient the differential diagnosis towards a history of recent bladder surgery.

In summary, ascites requires a comprehensive diagnostic approach in order to find the etiological cause. We must not forget that even in cirrhotic patients presenting with ascites, we must always be attentive to the patient's characteristics and history, as well as the analysis of ascitic fluid and imaging tests, in order to make an appropriate differential diagnosis.

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