Pelvic organ prolapse in women with autosomal dominant polycystic kidney disease under tolvaptan treatment

Prolapso de órganos pélvicos en mujeres con poliquistosis renal autosómica dominante en tratamiento con tolvaptán

Pelvic organ prolapse (POP) in women is a highly prevalent condition that involves herniation of the pelvic organs through the vaginal walls. It can be described as anterior, middle or posterior compartment prolapse.[1]

The aetiology of POP is usually multifactorial, but risk factors (RF) such as age, history of vaginal delivery,[2] connective tissue abnormalities, pelvic surgeries such as hysterectomy and diseases that lead to increased intra-abdominal pressure such as obesity or chronic constipation contribute to its development.[3]

The clinical presentation is highly variable, ranging from asymptomatic (the most common) to a feeling of heaviness or the appearance of a lump in the perineal area, urinary or faecal incontinence and sexual dysfunction. Symptom severity is more conditioned by the position than by the stage of the prolapse[4,5] and is dynamic, influenced by the level of physical activity or the degree of bladder or rectal repletion.[3]

Autosomal dominant polycystic kidney disease (ADPKD) is the most common inherited renal disease with an incidence of one in 400–1000 live births; its presentation typically consists of the development and progressive growth of cysts throughout the renal parenchyma.[6]

The development of renal cysts in ADPKD begins in the embryonic stage and they continue to increase in size throughout the individual's life.[7]

In March 2017, tolvaptan (TVP) treatment was approved by the European Medicines Agency (EMA) for the treatment of ADPKD in patients meeting criteria for rapid progression in order to slow the course of the disease.

The increase in renal volume leads to an increase in intra-abdominal pressure that weakens the pelvic floor muscles, and is more prominent in patients with large renal volumes.

In addition, the high volumes of diuresis secondary to TVP treatment in these patients lead to bladder distension, which contributes to the weakening of this musculature.

For this reason, we decided to analyse the prevalence of pelvic organ prolapse (POP) symptomatology in patients with ADPKD classified as rapid progressors and undergoing treatment with TVP.

In our series, of the total number of patients treated with TVP, seven were women, of whom three (43%) had POP symptoms. The most common symptomatology was the appearance of a genital mass and/or urinary incontinence. The average age of the patients was 42 years. Two patients were multiparous, mean body mass index was 31 (20–44), one of them had pelvic surgery (hysterectomy) as a risk factor and a history of cystocele and rectocele.

All three patients were on maximum dose TVP treatment (120 mg/day) with a mean diuresis volume of 6 l (6–6) and a mean urinary osmolality of 265 mOsm/kg (190–387).

Rehabilitation treatment led by the pelvic floor unit was effective in one patient. For the other two patients, the dose of TVP had to be lowered, and the drug had to be discontinued in one of them due to lack of improvement and limiting symptomatology.

These results suggest that the occurrence of POP may be common in women receiving TVP, with obesity being a risk factor for POP, as well as having a history of POP.

Asymptomatic status prior to initiation of TVP treatment does not rule out subclinical disease, which may be precipitated by increased bladder volume. Targeted anamnesis may be sufficient for screening prior to the start of treatment, as well as during follow-up, although more attention should be paid to patients with a history of POP or risk factors such as those discussed above.

In women with predisposing RF for developing POP, assessment and follow-up by the pelvic floor unit would be advisable from the start of treatment with tolvaptan.

In addition, personalised treatment should be considered, with the possibility of adjusting the drug dose according to urinary osmolality,[8] which could be useful in limiting the aquaretic effect and its complications in patients with risk factors for POP.

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Kluyvera ascorbata sepsis in a patient on hemodialysis

Sepsis por *Kluyvera ascorbata* en un paciente en hemodiálisis

Dear Editor,

In patients with chronic kidney disease on renal replacement therapy, after cardiovascular disease, infections are the second leading cause of hospitalisation and death.[1] Vascular access is the main source of bacteraemia, and in this group of patients sepsis the risk of death is increased 100 times.[2] The most common micro-organism involved in up to 80% of haemodialysis catheter infections is Staphylococcus aureus, but other germs, including non-fermenting Gram-negative bacilli, have also been reported.[3] There are several pathogens of the Enterobacteriaceae family, including Kluyvera strains,[4] which rarely affect humans, but when they do, they can cause severe infection and death. We believe it is of interest to present the case of a patient on haemodialysis with *Kluyvera ascorbata* (*K. ascorbata*) infection which, to our knowledge, has not been previously reported.

Case report

This was a 66-year-old woman, partially dependent for basic activities of daily living, smoker of 20 cigarettes a day, with a history of obesity, chronic obstructive pulmonary disease, hypertension, treated and cured cervical adenocarcinoma, urinary sepsis and long-standing type 2 diabetes mellitus (DM2) with micro- and macrovascular damage which had led to chronic diabetic kidney disease and the initiation of haemodialysis one year before. A right brachiocephalic arteriovenous fistula had been created, but did not mature. A right jugular tunnelled catheter was then inserted, but had been replaced five months ago due to dysfunction. She was referred from a dialysis centre for sudden onset of hypotension and dyspnoea with desaturation after sealing of the branches of the dysfunctional catheter with urokinase. As anaphylactic reaction was suspected, the patient was given adrenaline 5 mg by nebulisation and 0.5 mg intravenously, hydrocortisone 200 mg intravenously, dexchlorpheniramine 5 mg/mL intravenously and ventilatory support. Blood tests showed hyperkalaemia of 6.8 mEq/l as the only notable finding and a chest CT scan ruled out lung disease.

When the patient arrived at our centre, the referral hospital for the area, she was hypotensive and tachycardic and with a low level of consciousness. A greenish exudate was observed at the catheter entry orifice, so it was decided to remove it, sending the tip for culture, as well as taking samples for blood and urine cultures, and inserting a temporary catheter in the left jugular vein. A repeat blood test showed leuco-

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