Study and treatment of the couple in post-coital urinary tract infection in women☆

Estudio y tratamiento de la pareja en ITU poscoital de la mujer

Dear Editor,

Recurrent uncomplicated urinary tract infections (UTIs) are frequent among healthy young women.[1] In some women, there is a clear relationship with sexual activity; the frequency of is sexual intercourse described as an important contributing factor, and it has even been reported that women having sexual intercourse on a daily basis are at 9 times' greater risk.[2–4] While changes in sexual partner during the previous year is a known risk factor, it is unusual to studied the couple.

We present the case of a 39-year-old woman with a stable partner who presented with post-coital voiding syndrome. She had no prior relevant medical history, nor repeated UTIs, and did not take oral contraceptives or use spermicide. It was discovered to have positive culture for *Escherichia coli* culture. She therefore received treatment with ciprofloxacin and became asymptomatic. Two months later, she again presented with post-coital voiding syndrome; this time, it was caused by *Citrobacter koseri*. She received treatment with fosfomycin and nutritional-hygienic measures that included post-coital voiding. After 10 months without symptoms, she again presented with post-coital voiding syndrome, again with the *Citrobacter koseri* culture. She therefore began another course of antibiotics with fosfomycin and compliance with hygienic measures was stressed.

Three months later, when the voiding syndrome occurred again, a new culture was requested and her asymptomatic partner was studied, using a glans swab. The man's culture

Table 2 – Swab culture from male glans.

Date	02/03/2015	02/03/2015
Microorganism	*Escherichia coli*	*Citrobacter koseri*
Resistance marker		
Gentamicin	Sensitive ≤1	Sensitive ≤1
Cefalotin	Sensitive 16	Sensitive 4
Cefuroxime	Sensitive 4	Sensitive 4
Nitrofurantoin	Sensitive ≤16	Sensitive ≤16
Amoxicillin/clav.	Sensitive 8	Sensitive ≤2
Ampicillin	Resistant ≥32	Resistant ≥32
Nalidixic acid	Sensitive ≤2	Sensitive ≤2
Ciprofloxacin	Sensitive ≤0.25	Sensitive ≤0.25
Trimeth/sulfam	Sensitive ≤20	Sensitive ≤20
Fosfomycin	Sensitive ≤16	Sensitive ≤16

Table 1 – Bacteria in female urine cultures.

Date	18/11/2013	27/01/2014	13/11/2014	22/02/2015
Microorganism	*Escherichia coli*	*Citrobacter koseri*	*Citrobacter koseri*	*Citrobacter koseri*
Resistance marker				
Gentamicin	Sensitive ≤1	Sensitive ≤1	Sensitive ≤1	Sensitive ≤3 1
Cefalotin	Sensitive 16	Sensitive 4	Sensitive 8	Sensitive 4
Cefuroxime	Sensitive 4	Sensitive 4	Sensitive 4	Sensitive 4
Nitrofurantoin	Sensitive ≤16	Sensitive ≤16	Sensitive 32	Sensitive 32
Amoxicillin/clav.	Sensitive 8	Sensitive ≤2	Sensitive 4	Sensitive 4
Ampicillin	Resistant ≥32	Resistant ≥32	Resistant ≥32	Resistant ≥32
Nalidixic acid	Sensitive ≤2	Sensitive ≤2	Sensitive ≤2	Sensitive ≤2
Ciprofloxacin	Sensitive ≤0.25	Sensitive ≤0.25	Sensitive ≤0.25	Sensitive ≤0.25
Trimeth/sulfam	Sensitive ≤20	Sensitive ≤20	Sensitive ≤20	Sensitive ≤20
Fosfomycin	Sensitive ≤16	Sensitive ≤16	Sensitive ≤16	Sensitive ≤16

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showed 2 germs, *Escherichia coli* and *Citrobacter*, with identical sensitivity to the patient's cultures (Tables 1 and 2). Both started treatment with single-dose fosfomycin and no further new infections developed.

The study and treatment of couples with post-coital UTI in women may be helpful in managing recurrent UTIs.

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TSC2/PKD1 contiguous gene syndrome☆

Síndrome de genes contiguos *TSC2/PKD1*

Dear Editor,

We present the case of a 22-month-old girl assessed by a multidisciplinary team as she had seizures that were difficult-to-manage that started when she was 6 months old and she had bilateral polycystic kidney disease. Her parents were healthy and not consanguineous. She was born as a result of her mother's second pregnancy, an unmonitored single-foetus pregnancy complicated by HELLP syndrome. She was delivered by Caesarian section after 36 weeks, required resuscitation at birth and had generalised cyanosis. Her birth weight was 1700 g (DE −3.8) and her birth length was 46 cm (DE −2.5). She was hospitalised at 8 months of age due to tonic–clonic seizures, followed a torpid course with global delay in maturation delay.

On physical examination, she had a tendency towards dolichocephaly with an unusual facies characterised by a prominent forehead; a concave nasal bridge with a flattened tip; upturned nostrils; and a short, wide philtrum. She had 7 hypochromic lesions; 2 were ash-leaf that corresponds to the largest in her lumbar region and on her left thigh (Fig. 1a).

An MRI scan showed patch-shaped multi-focal areas with hyperintense behaviour on T2 flair in both parietal hemispheres not leading to a mass effect on the surrounding structures. A sleep electroencephalogram was abnormal due to paroxysmal activity with bursts of points and acute waves in the fronto-polar and right temporal regions. An eye ultrasound showed a raised lesion with a hypoechoic internal structure in the papillary area, consistent with an astrocytic hamartoma of the optic nerve of the right eye. A transthoracic echocardiogram showed tumour in the right ventricular outflow tract suggestive of rhabdomyoma neither causing obstruction nor resulting in haemodynamic repercussions. A kidney ultrasound showed an increase in volume in both kidneys, 9.4 cm × 4.0 cm for the left kidney and 9 cm × 3.6 cm for the right kidney. It revealed the formation of fluid-filled cysts, some of which had thick walls. The largest one, in the lower pole of the right kidney, was 6 cm × 3.1 cm (Fig. 1b). A kidney profile, as well as other serum analysis, was found to be within the reference values.

DNA was extracted, and mass sequencing of the *TSC1* and *TSC2* genes was performed on the MiSeq (Illumina) platform. This revealed 2 polymorphisms in heterozygosis in *TSC1*, GAA/GAG, c.1335A>G, p.Glu445Glu and ATG/ACG, c.965T>C, p.Met322Thr with rs7862221 and rs1073123 references, respectively. A multiplex ligation-dependent probe amplification study (MLPA P337) showed a deletion in heterozygosis of the

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