

Figure 2 Total rupture of the urethra at the prostatomembranous junction (combined retrograde and anterograde urethrogram with suprapubic cystostomy).

Case 3. Male aged 12 years with pelvic trauma (crushing) and rupture of the prostatic urethra (Fig. 2). Primary endoscopic realignment was performed. Endoscopic internal urethrotomies were performed at 5, 6, 9 and 14 months post-surgery. Transurethral lithotripsies were performed at 5 and 18 months after the patient developed vesicular lithiasis. An end-to-end urethroplasty was performed at 2 years post-surgery, and since the stricture recurred, a transpubic urethroplasty was performed at 3 years post-surgery. At 14 years post-surgery, the patient suffered from urinary incontinence and retrograde ejaculation.

The management of urethral lesions in pelvic trauma survivors must focus on minimising the risk of future sequelae. There is still controversy surrounding its initial management in paediatric urology. Once urinary derivation by suprapubic catheterisation (vesicostomy) has been done, it is difficult to perform primary endoscopic realignment (the main approach in adults)¹⁻⁴ in young children due to lack of experience and of paediatric cystoscopic equipment (including a flexible cystoscope). Urethral stricture is the natural course of full ruptures, leading to difficult urination and often to post-void residual urine, lithiasis, urinary retention, and urinary tract infections. The rate of stricture recurrence is high for the endoscopic approach, and is also considerable after open urethroplasty (15–68%).¹⁻³

Another possible sequela, urinary retention, occurs less frequently (3-24%), although it is the complication that has

Premature constriction of the ductus arteriosus *

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the greatest impact on the quality of life of a child or adolescent that has survived pelvic trauma.^{1–5} Last of all, ED is also a very prevalent sequela and it has only been studied recently in patients that presented with posterior urethral injury during childhood.^{2,6} In a recent retrospective study of 60 patients that had urethral injuries in childhood, 47% exhibited ED (severe in 82% of them), mainly due to damage to vascular structures (vasculogenic aetiology).⁶

Posterior urethral injuries must not be underestimated in cases of pelvic trauma in children. While these injuries do not affect initial mortality, their treatment can be unsuccessful and impact the long-term quality of life in adulthood.

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To the Editor:

Severe premature constriction of the foetal ductus arteriosus is a rare condition that may result in serious foetal and neonatal morbidity. Its development is usually associated with maternal exposure to nonsteroidal antiinflammatory drugs,¹ a polyphenol-rich diet² or a tortuous ductus arteriosus,³ although there are cases in which the cause is not identified (idiopathic constriction of the ductus arteriosus).⁴

Since this is an important phenomenon, we present the characteristics of the pregnant women diagnosed with

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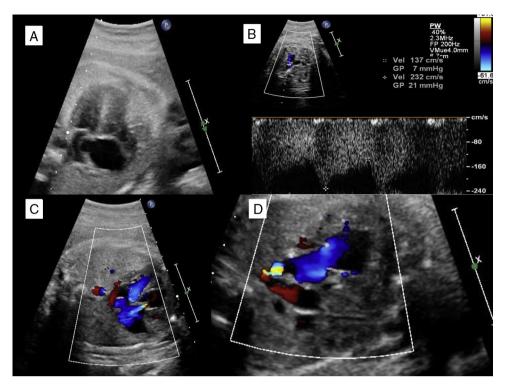


Figure 1 Severe premature constriction of the ductus arteriosus. (A) Four-chamber view showing dilatation of the right heart chambers with the foramen ovale flap bulging into the left atrium. (B) Doppler of the ductus arteriosus revealing a continuous blood flow with increased systolic and diastolic flow velocities. (C) Severe tricuspid regurgitation. (D) Three-vessel view showing a reduced ductal diameter.

premature intrauterine ductus arteriosus constriction in our department.

To that end, we performed a retrospective descriptive study by reviewing the medical records of the pregnant women who received care in the paediatric cardiology department of our hospital in the past three years (2011-2013) in whom echocardiography revealed the presence of premature constriction of the ductus arteriosus, and also of the subsequent evolution of the newborns.

We found evidence of premature constriction of the ductus arteriosus in 7 pregnant women, all of who were diagnosed in the third trimester at a mean gestational age of 35.4 weeks. Table 1 shows the characteristics of the pregnant women. The outcomes were favourable when the triggering factor was removed (5/7). One of the foetuses had a severe restriction of blood flow through the ductus arteriosus with dilation and dysfunction of the right heart chambers and severe tricuspid regurgitation (Fig. 1) that persisted in the neonatal period and was observed to have normalised in subsequent follow-up office visits.

Premature constriction of the foetal ductus arteriosus has been well described in the literature, although there are

Gestational age	Possible trigger	Impact on foetal haemodynamics	Maternal disease	Ethnicity	Disease in the newborn
At diagnosis					
35.4	Grapes	Yes	No	Caucasian	No
35.4	Paracetamol	No	No	Caucasian	No
32.0	Acetylsalicylic acid	No	Antiphospholipid syndrome	Caucasian	No
35.0	Hydroxychloroquine	No	Lupus	Latin American	No
35.0	Diclofenac and ibuprofen	Yes	Low back pain	Caucasian	Dilatation of right heart chambers and tricuspid regurgitation ++
35.6	No	No	No	Arabic	No
36.4	No	No	No	Caucasian	No

few published articles on the subject, which may be due to underdiagnosis of this condition. It must be suspected, particularly in the third trimester, if foetal echocardiography reveals dilatation and dysfunction of the right ventricle, tricuspid regurgitation and increased flow velocity in the ductus arteriosus measured by Doppler in the absence of structural heart disease.⁴ Identifying the possible cause of the condition requires taking a detailed history with particular emphasis on the diet of the mother and any medications taken during pregnancy.^{5,6} In our series, the type of maternal exposure most frequently associated with this condition corresponded to nonsteroidal anti-inflammatory drugs, as described in the literature,¹ although we found others such as exposure to hydroxychloroquine, which had not been described until now.

The early diagnosis of premature constriction of the foetal ductus arteriosus and the identification of its aetiology are essential in order to reverse or minimise haemodynamic alterations, as progression of this condition may lead to heart failure and foetal death.

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Review of para-infectious seizures from January 2012 to March 2014☆

Revisión crisis parainfecciosas de enero del 2012 a marzo del 2014

To the Editor:

Parainfectious seizures are afebrile convulsive seizures associated with minor infectious diseases, such as upper respiratory tract infections or acute gastroenteritis without electrolyte abnormalities or dehydration,¹⁻⁶ and are little-known in our country.^{1,3,4}

The aim of this retrospective descriptive study was to determine the incidence of parainfectious seizures in a tertiary hospital and to analyse the main characteristics of these seizures as well as their natural course. The inclusion criteria were having one or multiple afebrile seizures (body temperature equal or less than 37.9 °C) in association with a minor infection (upper respiratory tract infection or acute gastroenteritis without electrolyte abnormalities or clinical signs of dehydration); normal psychomotor development; and normal results in diagnostic tests. We excluded patients that had fever during the seizures, previously diagnosed with epilepsy, or with psychomotor retardation.

Our study (Table 1) included 11 patients ranging in age from 3 months to 5 years, 7 of who were male and 4 female. Only one of them had had a typical febrile seizure in the past.

The number of seizures ranged from a single seizure to a cluster of 10 seizures, and their duration from less than 1 min to 20 min (mean duration, 5.7 min), with 81.8% of seizures lasting less than 5 min. Generalised tonic-clonic seizures were the most frequent type (54.5%).

Of the 11 patients, 5 had upper respiratory tract infections, and 6 acute gastroenteritis.

Salmonella was isolated in 1 of the 5 stool cultures performed, and rotavirus in 2.

An acute-phase electroencephalogram was done in 10 of the 11 patients, and was normal in 40% (3 of the remaining patients had a slow EEG, and another 3 irritative features).

The neuroimaging tests performed included six magnetic resonance studies and one computer-assisted tomography, all of which were normal except in one patient that had non-obstructive non-progressive hydrocephalus.

Five patients required anticonvulsants to control their seizures in the emergency room, and were given benzodiazepines (diazepam or midazolam). Six of the patients that were hospitalised required anticonvulsants at a later point, and sodium valproate and levetiracetam were used most frequently. Only four patients required maintenance treatment at discharge, and one needed combination therapy.

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