Case Report

Infertility in adults with polycystic kidney disease

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Introduction

Because of a marked pituitary dysfunction, which is not reversed by dialysis, infertility is frequently a problem in male patients with chronic renal failure (CRF). CRF is associated, in fact, with impaired gonadal steroidogenesis and gametogenesis, elevated gonadotropin levels and attendant alterations in hypothalamic regulation of pituitary-gonadal function [1]. Therefore, any additional cause of infertility can further and dramatically decrease the ability of patients with CRF to conceive. These additional causes can be related to metabolic conditions, such as protein-caloric malnutrition (a frequent complication of CRF, and capable of inducing Leydig cell insufficiency) [2], or extra-renal complications of the primary disease, such as adult polycystic kidney disease (ADPKD).

Male patients with ADPKD, a disease responsible for ~6% of all cases of end-stage renal failure in Italy (unpublished data from the 1999 report of the Italian Society on Nephrology), have an increased rate of infertility even before developing advanced renal insufficiency.

Case report

A 38-year-old white married man was admitted in our unit because of infertility. Diagnosis of ADPKD had been made at the age of 26 years. No other relevant disease had been diagnosed before his admission. His serum creatinine was 0.8 mg/dl and creatinine clearance was 98 ml/min. Diagnostic criteria for infertility included: history, physical examination, abdominal and transrectal ultrasonography, nuclear magnetic resonance imaging, hormonal studies and semen analyses. The patient denied genitourinary symptoms or previous surgery. Physical examination did not reveal testicular abnormalities. Abdominal ultrasonography, however, showed multiple large cysts in both kidneys and the liver. Transrectal ultrasonography revealed a normal prostate, but both seminal vesicles appeared enlarged, with diameters of 2 and 3.2 cm, respectively. Abdominal nuclear magnetic resonance confirmed this finding. Serum levels of follicle-stimulating hormone, luteinizing hormone and PRL were normal, both at baseline and after stimulation with gonadotropin-releasing hormone, TRH and β-estradiol. Nevertheless, semen analyses showed ipozoospermia with severe oligo-terato-zoospermia, and we made a diagnosis of infertility secondary to ductal obstruction from dilation of seminal vesicles [3].

Discussion

The association between polycystic kidney disease and infertility due to dilatation of seminal vesicles is an important, although often unrecognized, cause of infertility in patients with ADPKD. The mechanism by which enlarged vesicles can cause infertility is the obstruction of the ejaculatory ducts, which leads to azoospermia or severe oligospermia. The pathogenesis of cyst formation in ADPKD has not been clearly defined, but it is likely that it is due to the same basement membrane defect that allows cyst formation in multiple organs [4]. Unfortunately, ADPKD is sometimes associated with other causes of infertility as described by Okada et al. [5] who, studying infertile patients with 9+0 immotile spermatozoa, found a high prevalence (four out of 16 cases) of ADPKD, hypothesizing a genetic linkage between these two conditions. However, dilatated seminal vesicles are probably the most frequent andrological complication and cause of infertility. Danaci et al. [6] reported that as many as 60% of patients with ADPKD have seminal cysts. In contrast, only 5.2% of andrological patients without ADPKD have that abnormality [7].
Despite such an elevated prevalence, little is still known about this extrarenal complication. For example, it is never mentioned in textbooks of internal medicine or in most textbooks of nephrology. Nevertheless, the nephrologist should be aware of the possibility of such a complication, because, although transurethral resection of the ejaculatory duct usually does not improve the semen quality, early epididymal sperm aspiration for in vitro fertilization can be effective in treating infertility. Testicular biopsy, in fact, often shows normal spermatogenesis despite oligoteratospermia [3].

**Conclusion**

Because of the high prevalence in ADPKD of seminal cysts that can cause obstruction of the ejaculatory duct and other abnormalities such as axonemal 9+0 defect, consideration should be given to fertility in the initial evaluation of these patients, before the onset of uraemia further reduces their ability to conceive. Patients with ADPKD, whose analysis of ejaculation is still normal, should be offered the opportunity for sperm cryopreservation, while those with oligoteratospermia and normal testicular biopsy could be considered for early microscopic epididymal sperm aspiration for in vitro fertilization.

**References**


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