Acute renal failure following IVF: Case report

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IVF is one of the most comprehensively registered interventions in clinical medicine. IVF is regarded as safe with very few complications. We report a woman who developed acute renal failure due to compression of both ureters from enlarged stimulated ovaries. The condition was diagnosed using ultrasound and magnetic resonance imaging (MRI). The condition was treated with insertion of double-J stents in both ureters and resolved without need of dialysis. Compression of the ureters due to enlarged ovaries should be considered if a patient develops acute renal failure following IVF.

Key words: acute renal failure/IVF/OHSS/ultrasound

Introduction

During the last 25 years, IVF has become an important treatment option in patients with infertility. Following hormone stimulation, the oocytes are collected from the ovaries transvaginally using ultrasound guidance. The procedure is regarded as safe. Two days after fertilization in vitro, the embryos are implanted in the uterus. The most common complications are haemorrhages, pelvic abscesses and pain (Yuzpe et al., 1989). There are also some reports of ureter damage after puncture by the collecting needle (Coroleu et al., 1997; Miller et al., 2002). We report a case where a woman developed acute renal failure due to compression of both ureters from enlarged stimulated ovaries.

Case report

A 30-year-old woman with infertility due to tubal impairment was accepted for IVF treatment. She had no history of renal problems. There had previously been two unsuccessful treatments in another hospital. She had been operated with laparoscopic removal of bilateral sactosalpinges. The ovaries were embedded in heavy adhesions and contained several clear fluid cysts. Adhesiolysis was not performed. Further investigation revealed that she had regular periods of 32–35 days and had normal hormonal status. She had a body mass index (BMI) of 31. Her husband was normospermic. She underwent four new attempts in our clinic using the same hormone stimulation regimen as for the previous procedures with a long downregulation using GnRH agonist (Synarel® nasal spray; Searle, UK) followed by stimulation with FSH (Puregon®; Organon, The Netherlands) 150 IU o.d. for 5 days, and thereafter 125 IU o.d. The first attempt yielded seven oocytes, and two good embryos were transferred. No pregnancy or complication occurred. In her second attempt, the fourth in total, ultrasonography on her 14th day of stimulation showed a total of 14 follicles of 17 mm and several follicles of <12 mm in addition to two cystic structures of 40 mm. After the first three attempts, ultrasonography had shown no evidence of hyperstimulation. During her last attempt, no scan was performed prior to the scan on day 14. She was then administered 6500 IU of HCG (Ovitrelle®; Serono, Switzerland). The cycle was monitored following the Nordic model consisting only of ultrasound folliculometric measurements and no serum estradiol (E₂) measurements (Wikland et al., 1994).

Transvaginal, ultrasound-guided follicle aspiration was performed 36 h after HCG administration. It was performed under light sedation and paracervical anaesthesia using a total of 10 ml of lidocaine 1.0%, yielding 18 oocytes. The left ovary was situated behind the uterus and was difficult to reach by puncture. During the collecting procedure, she reported pain in the lower left part of the abdomen. Because of this, some of the follicles were not aspirated and 25 mg of pethidine had to be administered twice. Ten oocytes were fertilized, of which four showed normal cleavage and an embryo score of 2.1. Two embryos, each with a four-cell score of 2.1 were transferred 2 days after oocyte collection and two embryos were cryopreserved. Luteal support was given daily with vaginal micronized progesterone (Crinone, Serono). A slight abdominal pain prevailed and non-steroidal anti-inflammatory drugs (NSAIDs) and paracetamol were prescribed.

She reported decreased urine output. From day 2 following aspiration, she developed anuria. At the same time, she presented gastrointestinal symptoms of nausea and vomiting and refrained from eating and drinking. At the day of embryo transfer, she reported lower abdominal pain and she was consequently admitted to the Department of Gynaecology.
Upon admission to the hospital, her serum creatinine was 329 \( \mu \text{mol/l} \). Renal ultrasound showed slight to moderate hydronephrosis, most pronounced in the left kidney. A percutaneous pyelostomy was performed by ultrasound guidance on this side. Vaginal ultrasound showed enlarged ovaries measuring \( 7 \times 9 \text{ cm} \) (right) and \( 3 \times 6 \text{ cm} \) (left), and no sign of ascites.

During the following 24 h, there was still no urine output via the pyelostomy catheter, only blood. She was given an indwelling bladder catheter which drained only 300 ml of urine/24 h. At this time, her serum creatinine increased to 617 \( \mu \text{mol/l} \). Because of persistent oliguria, it was decided to start haemodialysis the next day. However, before the dialysis was started, her urine output increased both from the pyelostomy and to some lesser degree from the bladder catheter. The serum creatinine started to decline, and dialysis was not needed. A magnetic resonance imaging (MRI) scan showed bilateral hydronephrosis (Figure 1), and very large ovaries (right, 13 cm antero-posterior diameter, 20 cm cranio-caudal diameter; left, 9 cm antero-posterior, 10 cm cranio-caudal) which led to bilateral compression of the ureters (Figure 2a–c). Double-J stents were inserted in both ureters via cystoscopy. The post-operative course was uneventful. She was discharged after 8 days with a serum creatinine value of 126 \( \mu \text{mol/l} \). Four weeks later, her serum creatinine had declined further to 80 \( \mu \text{mol/l} \) and the double-J stents were removed. Ultrasonography after another 6 weeks showed no sign of hydronephrosis. Unfortunately, no pregnancy developed.

**Discussion**

Transvaginally, ultrasound-guided oocyte retrieval has become the gold standard for IVF therapy (Lenz *et al.*, 1981; Wikland *et al.*, 1985; Tanbo *et al.*, 1988). It is considered as a well tolerated, cost effective and safe procedure. A few cases of ureteral damage due to puncture of the ureter by the collecting needle have been described. In one case, the ureter was compressed by a stimulated ovary in a patient with a transplanted pelvic kidney (Khalaf *et al.*, 2000). To our knowledge, however, there are no reports of development of oliguric renal failure in a patient with two normal kidneys. The diagnosis of ureteral compression was confirmed by MRI scan, a procedure without ionizing radiation and which should not cause any harm to fertilized embryos.

![Figure 1. MRI scan showing the stimulated ovaries compressing both ureters resulting in bilateral hydronephrosis.](https://academic.oup.com/humrep/article-abstract/20/8/2250/618504/2251)

![Figure 2. (a–c) Axial MRI sections showing compression of the ureters (arrows) and the enlarged ovaries.](https://academic.oup.com/humrep/article-abstract/20/8/2250/618504/2252)
The differential diagnoses considered in this case included retroperitoneal haemorrhage with a haematoma obstructing the urinary tract, and a pre-renal insufficiency due to hypotension. The use of NSAIDs might have worsened the situation. The MRI scan showed no sign of haematoma and there was no report of hypotension at any point. Bilateral ureteral damage caused by the collecting needle was considered to be very unlikely. However, it is possible that a combination of two separate mechanisms may have produced the bilateral obstruction. First, the patient did report pain in the left pelvic region during oocyte collection. This might have been due to ureteral damage. Secondly, on MRI, the right ovary was much larger than the left. We consider it to be most likely that the obstruction on the right side was due to external compression while the obstruction on the left was due to ureteral damage. However, cystoscopy showed no bleeding from the left ureteral orifice. Severe pelvic adhesions may have worsened the situation by limiting the normal movement of the ovaries.

Ovarian hyperstimulation syndrome (OHSS) is a common complication in assisted reproductive technologies. It is seen to occur in ~10% of the treatments, and the severe form is observed in 0.5–2% of IVF cycles (Olivennes, 2003). OHSS is usually described by enlarged multicystic ovaries, ascites and haemoconcentration. Acute renal failure due to a hypovolaemic state following production of protein-rich ascites in patents with OHSS has been reported (Winkler et al., 1992), but in this case there were very few ascites and only slight haemoconcentration. The most pronounced finding was the huge enlargement of the ovaries. The patient was not diagnosed as having a polycystic ovarian condition; she had regular periods and normal hormonal levels, and she also had a normal reaction on the three previous treatments. However, the enormously enlarged ovaries following an approved stimulation protocol could indicate some sort of polycystic ovaries.

Even though the complication risk related to IVF is low, one should be aware of a possible compression or damage to the ureters with subsequent development of acute renal failure. To prevent permanent kidney damage and to ensure safe development of the pregnancy, the condition has to be diagnosed and treated without delay. As ionizing radiation may be harmful to the fetus, MRI together with ultrasonography should be the imaging modalities of choice in the diagnostic work-up of such a patient.

References


