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Two Woeful Cases of Iatrogenic Hypergonadotropic Hypogonadism: The Inguinal Hernia Repair Should Always be as Cautious as Necessary to Avoid Turning a Potential Risk of Fertility Disorder into an Actual One

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Abstract **Case Report**

A bilateral inguinal herniorrhaphy had driven a testicular atrophy and a iatrogenic hypergonadotropic hypogonadism in two males aged 40 and 31 years. That iatrogenic hypogonadism had shaken the marital life of the 40 years old male despite he was already father of two children. It had almost destroyed the hope of the 31 years old male to become a father. These two woeful cases remind surgeons that the inguinal hernia repair should always be as cautious as necessary to preserve fertility and sexual life.

Keywords: Hypergonadotropic bilateral inguinal Fertility Disorder.

INTRODUCTION

The inguinal hernia repair is a common operation [1]. Despite it potentially encompasses the risk of damaging the spermatic cord's components in males; it rarely leads to a testicular destruction or infertility [2-5]. We present two unfortunate cases of iatrogenic hypergonadotropic hypogonadism triggered by a bilateral inguinal herniorrhaphy.

OBJECTIVE

To report two cases of post-herniorrhaphy testicular atrophy and infertility

CASE PRESENTATION

CASE 1

A 40 years old male complained of an anejaculation. He was married and father of two children. He had undergone a bilateral inguinal herniorrhaphy 7 months earlier. On examination, his scrotum was shrunk and his testicles were not palpable (figure 1). According to him, his right testicle had always been tiny since he was a child whereas his left testicle's volume had drastically decreased since his hernia repair was performed. His FSH level was 7.78 x 10^{-2} IU/mL i.e. 6.27 times the upper limit of normal range (1.5 x 10^{-3} – 1.24 x 10^{-2}). His LH level was 3.76 x 10^{-2} IU/mL i.e. 4.37 times the upper limit of normal range (1.7 x 10^{-3} – 8.6 x 10^{-3}). His prolactin level was 27.08 ng/mL i.e. 1.69 times the upper limit of normal range (2 – 16). His testosterone level was 0.025 ng/mL i.e. 10^{-2} time the lower limit of normal range (2.5 – 8.4). Thus, the patient presented a hypergonadotropic hypogonadism. As he wanted to have no more children, we treated him with testosterone 50 micrograms as a daily ointment of the skin (alternatively the skin on the right shoulder, the skin on the left shoulder and the abdominal skin below the umbilicus). 33 months later, he changed his mind on the further paternity matter as his wife pressured him to have a new child. Nevertheless, the couple abandoned the idea of having a new child by means of an assisted reproduction technique as it both could not afford the expenses related to the method and did not intend to resort to a donor's sperm.

Fig-1: A: Bilateral scars of herniorrhaphy in the 40 years old male B: Void and shrunk scrotum in the same 40 years old male

CASE 2

A 31 years old male consulted for an azoospermia. Twenty-nine months earlier, he underwent a bilateral inguinal herniorrhaphy combined with a bilateral varicocelectomy. The latter was performed to better his sperm motility. In fact, the patient who had resorted to medical care for a primary infertility had presented a decreased sperm motility along with a normal sperm count, i.e. 23.5 millions of spermatozoids per milliliter of ejaculate.

On workup, his levels of testosterone, bioavailable testosterone and prolactin were normal and were respectively 7.25 ng/mL (3.0 ≤ normal ≤ 10.6), 1.34 ng/mL (1.00 ≤ normal ≤ 3.70) and 5.6 ng/mL (3.46 ≤ normal ≤ 19.40). His levels of LH was 1.4 x 10^{-2} IU/mL, i.e. 2 x 10^{-3} unit above the upper limit of normal range (5.7 x 10^{-4} – 1.2 x 10^{-2}). His FSH level which was highly elevated amounted to 3.4 x 10^{-2} IU/mL i.e. 2.8 times the upper limit of normal range (9.5 x 10^{-4} – 1.2 x 10^{-2}). His sperm count was 0 spermatozoid per milliliter of ejaculate. Moreover, his ultrasonography-estimated right and left testicle volume had dropped from 10.79 mL and 10.67 mL to 1.5 mL and 2 mL in 22 months after the surgery. Thus, the non-mesh hernia repair had driven an atrophy of both testicles and a non-obstructive azoospermia. We referred the patient to an assisted reproduction center. A testicular biopsy might still allow the extraction of his own spermatids. Or he might resort to a donor's sperm.

DISCUSSION

Each one of the two patients exhibited a history of a bilateral inguinal herniorrhaphy with subsequent progressive atrophy of the testicles. The time from surgery to testicular atrophy was short: less than one year in one patient and less than two years in the other one. Nevertheless, testicular atrophy may develop beyond two years after inguinal hernia repair [4]. The testicles were completely lost in one patient. The latter had no more erection and no more ejaculation. The other patient had lost 16.3% of his total testicular volume and had developed an azoospermia. Either the full fertility of one patient or the sub-fertility

of the other patient was turned into a hypergonadotropic hypogonadism type of infertility. The operation may have incidentally triggered a compression, a ligation, a cutting or other fibrotic or thrombotic lesions of the spermatic cords' components [6, 7]. Anyway, the damage can hardly be reverted in either one of the patients. The 40 years old patient may have been lucky to have two children before getting an iatrogenic hypogonadism. Yet the hypogonadism has disrupted the equilibrium of his sexual and marital life as he faces an erectile dysfunction and a secondary infertility. In the 31 years old patient the situation is more desperate as he turned from decreased sperm motility to an azoospermia while seeking a medical solution for an infertility issue.

CONCLUSION

The two patients got a bilateral testicular destruction from a bilateral inguinal herniorrhaphy. Those two unfortunate cases remind surgeons those so-called or so-thought light procedures such an inguinal hernia repair can lead to sorrowful outcomes if they are neglectfully carried out.

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