

Evaluation of Testicular Artery Blood Flow in Infertile Patients with Varicocele

Thesis

**Submitted in partial fulfillment for the master degree
in Dermatology, Andrology & STDs**

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2010

Abstract

The aim of the study was to measure the testicular artery blood flow parameters in infertile patients with varicocele as compared with normal fertile men. The study was carried out on 60 infertile patients with varicocele and 20 fertile patients with varicocele, also 20 fertile men with no varicocele were included as control group. All patients were subjected to history taking, general and local genital examination. Conventional semen analyses were performed twice in patient group. Hormonal profile was done to the infertile patient group. Scrotal color Doppler ultrasound measurements of vein diameter, testicular volume and testicular artery blood flow parameters were done for both patients and control groups. The results showed that evaluation of testicular artery blood flow shows that it is most high in infertile patients with varicocele regardless of the degree of vein diameter, becomes less higher in fertile patients with varicocele and is of least value in fertile patients with no varicocele.

Key words: Varicocele, Testicular artery, Scrotal color Doppler ultrasound.

Acknowledgement

*First and foremost, thanks to **Allah**, the most beneficial and most merciful.*

*I would like to express my deep thankfulness and gratitude to **Dr. Ahmad Mahmoud Salem**, Professor of Andrology and STDs, Faculty of Medicine, Cairo University for his valuable advices and support throughout the work.*

*I am greatly honored to express my sincere appreciation to **Dr. Sahar Nassef**, Professor of Internal & Vascular Medicine, Faculty of Medicine, Cairo University for her continuous, sincere and valuable help and support in every detail of this study.*

*Many thanks and respect to my dear teacher **Dr. Amr Mohammad Gadalla**, Assistant Professor of Andrology and STDs, Faculty of Medicine, Cairo University for his continuous help, generous advice throughout this work and his guidance to me since the start of my residency in the Andrology and STDs Department.*

*I would also like to thank all my **Professors and Colleagues** in the Department of Andrology and STDs, Faculty of Medicine, Cairo University for their kind help and support, with special thankfulness and gratitude to **Dr. Alaa Abd Al Aal**, Lecturer of Andrology and STDs for his kind help throughout the study.*

*Finally, I would also like to dedicate this work to **my dear mother**, my brothers, my beloved wife and my son and to the one I miss the most and cherish his memory my late beloved and dear father **Dr. Raef Sadek**.*

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List of Abbreviations

CDUS: Colour Doppler Ultrasound.

Testicular Vol: Testicular volume.

TAvol flow: Testicular artery volume flow.

PSV: Peak systolic velocity.

EDV: End diastolic velocity.

TAm_{ax} flow: Testicular artery maximum flow velocity.

TAm_{in} flow: Testicular artery minimum flow velocity.

PI: Pulsatility index = $(PSV - EDV) / \text{mean velocity}$.

RI: Resistivity index = $(PSV - EDV) / PSV$.

S/D ratio: (PSV / EDV) ratio.

FSH: Follicular stimulating hormone.

Introduction
&
Aim of The Work

Introduction

Varicocele can be defined as an abnormal elongation, dilation, tortuosity and reflux of blood of the veins of the pampiniform plexus. Idiopathic varicocele is usually asymptomatic. It presents as an asymmetry in scrotal size with changes in spermiogram parameters, and is rarely associated with testicular pain (*Noske and Weinder, 1999*).

Numerous clinical studies have demonstrated an association between varicocele and testicular dysfunction; however, the pathophysiological effects of varicocele remains poorly understood which includes testicular hypoxia and venous stasis (*Takahara et al. 1991*), the retro-grade flow of adrenal metabolites from the renal vein to internal spermatic vein, nitric oxide (*Mitropoulos et al.1996*), reactive oxygen species (*Sharma et al.1996*) and regulators of apoptosis (*Baccetti et al.1996*).

However the well-documented hypothesis is that varicocele induces testicular damage by causing hyperthermia (*Harrison et al. 1990*) that some researchers have attributed to an increase in arterial blood to the testes which leads to vasodilatation as seen in experimental left varicocele in both rat and dog models (*Turner et al. 1990*).

The arterial blood supply to the testis comes from the testicular artery, vasal artery, and cremasteric artery. At the level of the testis, all three arteries anastomose to allow adequate blood supply and perfusion to the testis (*Parrot et al. 1994*).

Color Doppler ultrasonography (CDUS) can be used as a non-invasive method for demonstrating the structure and pathologies of the testicular artery and venous anatomy with a high sensitivity and specificity (*Trum et al. 1996*). There was no significant difference between blood flow measurements obtained superior to the testis and where the artery enters the testis (*Ross et al. 1994*). The blood supply to the testis was later defined by a new concept describing a segmental vascular organization in testicular vascularity. This concept means that substance uptake to the germinal epithelium and Leydig cells may be caused by a higher blood flow rather than by diffusion (*Ergun et al. 1996*).

It was demonstrated that arterial blood flow decreased in monkeys with experimental left varicocele suggesting that increased pressure on the venous side of testicular capillary beds might decrease arterial blood flow and cause testicular damage (*Harrison et al. 1986*). Decreased arterial blood flow may be due to resistance in the segmental vascular bed, increased venous pressure and reflux of renal and adrenal metabolites (*Aydos et al. 1993*). Defective energy metabolism and decreased testicular blood flow may result from experimental varicocele in animals (*Hsu et al. 1994*).

It is difficult to measure the testicular blood flow in humans because of the low flow rates, the complexity of the anatomical structure and the small size of the testicular arteries. Theoretically, the ideal site for measuring the testicular artery blood flow is the point where the artery enters the testis. However, this is not possible because of the anatomical complexity which stands as one of the reasons for the contradictory results in the literature (*Trum et al. 1996*).

The basic feature in varicocele is the change in vascular parameters, where to explain the etiology of varicocele, it has been hypothesized that increased arterial blood flow to the testes at puberty exceeds the venous capacity, resulting in venous dilation and varicocele. This is consistent with the findings noted in all the animal models; however confirmation in humans is required (*Nagler et al. 1987*).

In a recent study, the testicular artery blood flow (TABF) was found to be significantly decreased in men with varicocele, which may be a reflection of impaired microcirculation that leads to defective energy metabolism and in turn impaired spermatogenesis (*Tarhan et al. 2003*).

Aim of The Work

To measure the testicular artery blood flow parameters in infertile patients with different grades of varicocele as compared with normal fertile men.

Review of Literature

ANATOMY OF THE TESTIS

The left testis lies at a lower level than the right within the scrotum; rarely this arrangement is reversed. Each testis is contained by a tough white fibrous capsule, the tunica albuginea, and invaginated anteriorly into a double serous covering, the tunica vaginalis, just as the intestine is invaginated anteriorly into the peritoneum. Along the posterior border of the testis, rather to its lateral side, lies the epididymis, which is divided into an expanded head, a body and a pointed tail inferiorly. Medially, there is a distinct groove, the sinusepididymis, between it and the testis. The epididymis is covered by the tunica vaginalis except at its posterior margin which is free or, so to say, 'extra-peritoneal'. The testis and epididymis each bear at their upper extremities a small stalked body, termed respectively the appendix testis and appendix epididymis (hydatid of Morgagni). The appendix testis is a remnant of the upper end of the paramesonephric (Müllerian) duct; the appendix epididymis is a remnant of the mesonephros. These structures, being stalked, are liable to undergo torsion (*Harold et al., 2006*).

ARTERIAL BLOOD SUPPLY:

The principle arterial blood supply of the testis is via the testicular artery (internal spermatic artery) which arises from the aorta at or immediately below the level of the renal vessels (at the level of the second lumbar vertebra) and runs its course with the spermatic cord through the inguinal canal giving a branch to the

epididymis. At the back of the testis it divides into medial and lateral branches which do not penetrate the mediastinum testis but sweep around horizontally within the tunica albuginea where their branches penetrate the testicular parenchyma (*Sinnatamby et al., 1999*).

There are 5 patterns of testicular artery terminations:

First pattern it divides into upper polar or segmental and lower polar or segmental branches. *Second pattern* it gives upper polar, middle segmental and lastly it continues to the lower end of the testis. *Third pattern* it divides into upper, middle and lower segmental branches. *Fourth pattern* it gives upper polar only and continues by itself along the mediastinum towards the lower end of the testis then it is directed to supply the antero-lateral aspect of the testis. *Fifth pattern* it descends without extra-testicular branches along the mediastinum testis (*Taymour et al., 2002*).

Two other arteries share in the blood supply of the testis, these are the vasal artery and the cremastic artery. The artery of the vas (differential artery) arises from the inferior vesical artery and lies in contact with the vas until it reaches the epididymal head where it branches into capillary network supplying the tail and body of the epididymis. The cremastic (external spermatic artery) arises from the inferior epigastric artery and runs its course towards the internal inguinal ring where it enters the coverings of the spermatic cord and continues to anastomose with the capillary network of the differential and internal spermatic arteries (*Sinnatamby et al., 1999*).

Fifty six percent to 69% of patients have a solitary testicular artery. The testicular artery anastomoses with the artery to the vas, supplying the vas deferens and epididymis, which arises from the inferior vesical branch of the internal iliac artery. This cross-connection means that ligation of the testicular artery is not necessarily followed by testicular atrophy (*Beck et al., 1992; Yamamoto et al., 1995*).

Contrary to classical descriptions of testicular arterial anatomy that depict a single testicular artery branching at the level of the scrotum (*Warwick and Williams, 1973; Woodburne, 1978*), several investigators, using both clinical and histologic analysis, have documented the presence of multiple arterial branches within the inguinal spermatic cord as far proximally as the internal ring (*Beck et al, 1992; Jarow et al, 1992; Ergun et al, 1997; Hopps et al, 2003*). Using loupe magnification, Jarow et al (1992) examined the spermatic cords of 12 men who underwent inguinal varicocelectomy and reported a mean of 2 testicular arteries (range, 1–3). Histologic analysis of 17 adult spermatic cords examined at autopsy revealed a mean of 2.4 arteries at the level of the proximal inguinal canal (*Jarow et al, 1992*).

Reporting on the intraoperative anatomy of 83 infertile men who underwent microsurgical varicocelectomy at the inguinal level, 1 artery was identified in 69% of dissections, 2 arteries in 27% of dissections, and 3 or more arteries in 4% of spermatic cords (*Beck et al, 1992*). In a follow-up report, the same group identified 2 arteries in 42% of all dissections and 3 arteries in 33% of the spermatic cords during microsurgical varicocelectomy at the subinguinal level (*Hopps et al, 2003*).