MEAN TUBULAR DIAMETER (MTD) IN CADAVERIC VERSUS CRYPTORCHID TESTES

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ABSTRACT

Cryptorchidism refers to hidden testicle. It is a clinical condition where one or both testicles have been retained from entering the scrotum during the later part of the foetal period. Along with irreversible damage at both gross and cellular levels there is impairment of endocrine and reproductive functions of the testicles. As the incidence of cryptorchidism is very low and previously reported research and data on comparative findings between cadaveric and cryptorchid testes in the state of Bihar was not available this study was undertaken.

KEYWORDS: Cryptorchidism, testicle, scrotum, comparative.

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INTRODUCTION

The testis is a white, ovoid organ that is normally 15 to 25 ml in volume[1], has a length of 4.5 to 5.1 cm[2], a diameter of 2.5 cm[3] and weighs 10-15 grams[3]. The testicles are the only organs in humans that are located outside the body and have two main functions: to produce hormones, in particular testosterone and to produce male gametes, the spermatozoa[4]. Leydig cells are the prime source of testosterone[5,6,7] and contribute to about 5-12% of testicular volume[8,9,10]. Parenchyma of the testicle is divided into compartments separated by connective tissue septa [3,11]. Each septum divides seminiferous tubules into lobes [11]. Each seminiferous tubule contains developing germ cells and interstitial tissue[11]. In humans, interstitial tissue comprises 20-30% of total testicular volume[12].

Seminiferous tubules are long, highly coiled, looped structures, both ends of which usually terminate in the rete testis [11]. Each seminiferous tubule is about 200µ in diameter [3]. They are surrounded by several layers of peritubular tissue[13]. Cryptorchid testicles of adults are much smaller than normal and there is no doubt that undescended testicles not operated upon early in life are seriously damaged[14].

MATERIALS AND METHODS

Both apparently normal testes were carefully dissected and removed from an adult cadaver. These were the control testes and labeled as “C”. Four museum specimens of orchidectomized cryptorchid testes were examined both morphologically and histologically.
These were labeled as $T_1$, $T_2$, $T_3$ & $T_4$ respectively. Their findings were contrasted with morphological and histological findings of apparently normal testes obtained from cadaver. The focus of the present study is to find out justifiable deviations present in a cryptorchid testis from a normal testis. Both “C” as well as “$T_1$, $T_2$, $T_3$ & $T_4$” were measured in three dimensions using ruler and thread, they were weighed on an electronic weighing scale. After that both control and specimens were subjected to tissue processing using Bouin’s fluid as a fixative. This fluid rapidly coagulates the tissue and yet well preserves the tubular cytoarchitecture. It also imparts a bright yellow colour to the tissue which makes it clearly visible during embedding and section cutting. Routine processing was performed and the tissues were stained according to Ehrlich’s H & E method. Lastly the stained sections of each specimen were studied under the Carl Zeiss Observer Z1 microscope at various magnifications and the following information was recorded.

- Mean Tubular Diameter (MTD) in microns ($\mu$)
- Presence or absence of spermatogonia
- Presence or absence of Leydig cells
- Condition of the basement membrane
- Presence or absence of peritubular fibrosis
- Presence or absence of Sertoli cells

**OBSERVATIONS**

In the present study the observations were as follows.

Mean Tubular diameter (MTD) was calculated by measuring the average of vertical and horizontal diameters of 20 randomly selected seminiferous tubules (ST) in each of “C, $T_1$, $T_2$, $T_3$ & $T_4$.”

**Fig. 1:** Histological observation of Control Testis in 10X.

**Fig. 2:** Histological observation of T1 in 10X.

**Fig. 3:** Histological observation of T2 in 10X.

**Fig. 4:** Histological observation of T3 in 10X.

**Fig. 5:** Histological observation of T4 in 10X.
Table 1: Showing morphological findings of “C, T₁, T₂, T₃ & T₄”.

<table>
<thead>
<tr>
<th>Specimen</th>
<th>Length (cm)</th>
<th>C</th>
<th>T₁</th>
<th>T₂</th>
<th>T₃</th>
<th>T₄</th>
</tr>
</thead>
<tbody>
<tr>
<td>ST</td>
<td></td>
<td>4.4</td>
<td>1.2</td>
<td>2.6</td>
<td>2.8</td>
<td>3.2</td>
</tr>
<tr>
<td>ST-01</td>
<td></td>
<td>2.8</td>
<td>0.9</td>
<td>1.6</td>
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<td>2.2</td>
</tr>
<tr>
<td>ST-03</td>
<td></td>
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<td>0.6</td>
<td>1.2</td>
<td>1.5</td>
<td>1.8</td>
</tr>
<tr>
<td>ST-05</td>
<td></td>
<td>15</td>
<td>1</td>
<td>3</td>
<td>5.5</td>
<td>8.5</td>
</tr>
</tbody>
</table>

VD = Vertical Diameter HD = Horizontal Diameter

Table 2: Showing the Vertical diameters (VD) and Horizontal diameters (HD) of seminiferous tubules (ST) of “C, T₁, T₂, T₃ & T₄” in microns (µ).

Table 3: Showing Mean Tubular Diameter (MTD) of “C, T₁, T₂, T₃ & T₄” in microns (µ).

Table 4: Showing Histometric observations (HO).

Conclusion
The early genital systems in both sexes is similar hence the initial period of development has been termed as indifferent state of sexual development[15]. The gonads are derived from three sources: mesodermal epithelium lining the posterior abdominal wall, underlying mesenchyme, primordial germ cells [15]. Testicles usually develop in embryos with a normal Y chromosome but only the short arm of this chromosome is critical for sex determination[15]. The SRY gene for the testis-determining factor (TFD) on the short arm of the Y chromosome acts as a switch that directs the development of a gonad into a testis[15]. By 26 weeks the testes have descended retroperitoneally from posterior abdominal wall to the deep inguinal rings[16]. The process is controlled by androgens and takes 2 or 3 days[16]. More than 97% of full term new
born males have both testes in the scrotum and during the first three months most undescended testes descend into the scrotum[16]. Cryptorchidism refers to interruption of the normal descent of the testicle into the scrotum. The testicle may reside either in the retroperitoneum, inguinal canals or any of the rings. A distinction should be made between undescended and retractile testicle. A congenitally absent testicle results from failure of normal development or an intrauterine accident leading to loss of blood supply to the developing testicle. It is now established that cryptorchid testicle shows increased predisposition to malignant degeneration. In addition fertility is decreased when the testicle is not in the scrotum. Orchidopexy will never improve the fertility potential. The testicle will remain at risk to malignant change although its scrotal location will facilitate earlier detection of malignancy. Males with bilateral empty scrotum are often infertile. When a testicle is not in the scrotum it results in decreased spermatogenesis due to exposure to higher temperature. Mengel and coworkers demonstrated by histologic analysis that after two years of age there is reduction of spermatogonia. Incidence of infertility is twice as high in men who have undergone orchidopexy that in those men with normal testicular descent. In this study significant differences between apparently normal cadaveric and cryptorchid testes have been reported. The MTD is an excellent indicator of the development of the seminiferous epithelium[17]. In the prepubertal testes the tubular diameter depends principally on Sertoli cells and thus indicates whether they are adequately stimulated by FSH. Tubular diameter varies throughout, being smallest in the end of third year of life, slowly enlarging up to nine years of age and rapidly enlarging thereafter up to fifteen years by which the tubules reach their definitive diameter ranging from 160µ to 170µ. The most frequently abnormality in cryptorchid testes is a low MTD. These findings are supported by the fact that the MTD of a randomly selected seminiferous tubule of a healthy adult testis is approximately 200µ. This is in accordance with our findings where we have shown that the MTD of the normal testis obtained from cadaver is 208.35µ. None of the cryptorchid testes had an MTD remotely near the 100µ mark. Due to morphological distortions all the cryptorchid testes were smaller as well as lighter than the cadaveric testis. In the past, authors have emphasized the relationship between testicular deformity and malignant disease. The absence of spermatogenesis and identifiable spermatogonia, thickening of the basement membrane, vacuolated Leydig cells, extensive peritubular fibrosis and reduction in both, number as well as MTD of seminiferous tubules strongly supports this fact.

CONCLUSION
Cryptorchidism either unilateral or bilateral has deleterious effects on the testicles. Our study has demonstrated the same. Early orchidopexy in unilateral maldescent is strongly suggested to safeguard both ipsilateral and contralateral testicles.

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Conflicts of Interests: None

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