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SYNOPSIS/MINI-REVIEW

Measurement of Penile and Clitoral Sizes in Post-Neonatal Infants

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Introduction

When the appearance of a child's genitalia differs from the usual anatomical description, with difficulty in assigning gender, the genital is described as a disorder of sex differentiation (DSD). Late diagnosis of DSD in the developing world is frequently due to delayed presentation arising from non-recognition and inhibition from stigma. This is even worse when close to half of children are born in hospitals. This could be due to a lack of thorough external genitalia examination at birth that could have detected subtle aberrations in genital anatomy or a lack of knowledge about what genital sizes may constitute an abnormality. Early diagnosis of micropenis, clitoromegaly or other forms of DSD is important, since it may be a pointer to an underlying serious or life-threatening hormonal disorder. [1]

Embryology of sex determination

The sperm that fertilises the oocyte at fertilisation determines the chromosomal and genetic sex of the embryo. Fertilisation with a Y-bearing spermatozoon results in a male phenotype (XY), while the X-bearing one results in a female phenotype (XX).[2] After six to seven weeks post-fertilisation, the gonads in both sexes are known as the indifferent state of sexual development or the ambisexual genital tubercle. This structure gives rise to the penis in the male and the clitoris in the female.[3] Subsequently, after the differentiation of the bipotential gonadal ridge to the testis, testosterone production begins in the Leydig cells under the influence of human chorionic gonadotropin (hCG). This occurs at eight to twelve estimated weeks of gestation. Androgens, and in this context testosterone, act via the Androgen receptor (AR) found on the target organs or tissues. Both sexes express the Androgen receptor (AR), but due to low levels of testosterone in the genetic female, it results in the development of the genital tubercle into the clitoris.[4]

In target organs or tissues, testosterone is metabolised into the active 5α- dihydrotestosterone (DHT) by 5α-reductase, which then induces differentiation of the external genitalia in males into the penis and scrotum.[4] Foetal androgen production from the Leydig cells is high between estimated gestation weeks of eight to twenty-four weeks and peaks between fourteen to sixteen weeks, consequently leading to an increase in penile length from about 20mm to 38mm.[5] Therefore, micropenis occurs secondarily to a hormonal abnormality that occurs after the twelfth week of gestation, e.g. 5α-reductase deficiency or androgen insensitivity syndrome (AIS).[5]

Postnatally, with no clear physiological significance, the hypothalamic-pituitary-gonadal axis is activated in the first three months of life, leading to Gonadotrophin Releasing Hormone (GnRH), Luteinizing and Follicle-Stimulating Hormone (LH/FSH), and testosterone release. This activity leads to an increase in both testicular volume and penile length. [5,6]

The testes develop retroperitoneally in the lumbar region of the posterior abdominal wall.[2] They gradually descend into the scrotum, reaching the iliac fossa during the third month of intrauterine life and lie at the deep inguinal ring in the seventh month of intrauterine life. They pass through the inguinal canal during the seventh month and by the end of the eighth month of intrauterine life are in the scrotum.[2]

The descent is caused by:

- Differential growth of the body wall.

- Formation of the inguinal bursa from about the sixth month of intrauterine life. The bursa increases in size and depth, eventually reaching the scrotal sac. The testis then enters the bursa to reach the scrotum.
- The gubernaculum extends from the lower pole of the testis to the scrotum. It decreases in length as the embryo increases in size, and its shortening leads to caudal movement of the testis. It also dilates the inguinal bursa, providing a pathway for the descending testis.
- Hormones, especially testosterone, influence the migration of testes from the abdominal cavity to the scrotal sac.

The above events are known to have been influenced by testosterone, especially gubernaculum regression.
In a female foetus, intrauterine or post-natal exposure to excessive testosterone may result in genital anomaly, especially clitoromegaly on account of the trophic effect of testosterone on the clitoral tissue.[7]

Abnormalities of penile and clitoral development
The anatomic external and internal genital appearance is the product of complex interactions between genetic and endocrine processes during foetal development.[48] The external genitalia are considered DSD whenever there is difficulty in assigning gender to a child based on the appearance of the external genitalia.[4] The other common abnormalities of penile and clitoral development are webbed penis, buried penis, trapped penis, chordee, hypospadias and labial adhesions.[6,9] Since they are hidden by surrounding tissue, skin, or fat, webbed, buried, and trapped penis are classified together as inconspicuous penis.

The webbed penis has the skin from the scrotum tethered to the ventral surface of the penile shaft or the penoscrotal junction, which is close to the dorsum of the penile skin. In this condition, circumcision should be avoided until surgical correction is done. The two types of concealed penises are buried and trapped; buried penises are congenital, whereas trapped penises are acquired. The weak anchoring of the superficial fascia and penile skin to the deeper fascia at the penile base is responsible for the concealment. With somatic growth and a diminishing fat pad, the penile shaft is unburied, and if not, surgical correction is needed.[6,9]

Acquired, trapped penis occurs after poorly done circumcision, with the penile skin forming a circumferential distal scar, trapping the penis below it. Chordee is a penile curvature abnormality, and surgical correction is recommended in the first year of life. If not corrected, it may result in psychosocial problems, dyspareunia, and poor urinary stream while standing. Hypospadias occurs when the urethral meatus opens ventrally along the penile shaft. Corrective surgery is the treatment for the condition.
Labial adhesion is commonly diagnosed between three months and six years. Anterior fusion of the labia is often associated with the disappearance or fusion of the clitoral hood. [6,10] It is mostly benign and asymptomatic and often detected by the parents or during physical examination by a health worker. If not detected, it can cause skin rashes secondary to retained urine and urinary tract infection.[6,10] Therefore, painstaking physical examination of the genitalia, including separation of the labia minora, is important in any female infant or child with urinary tract infection. Labial adhesion is usually corrected by the application of topical oestrogen cream or by surgical lysis.

Mini puberty
Mini puberty refers to the post-natal activation of the hypothalamic-pituitary-gonadal (HPG) axis, which occurs mainly in the first six months after birth. During this time, high levels of gonadotropins and sex steroids are produced, leading to the maturation of sexual organs in both males and females. In males, the activation of gonads causes the levels of testosterone to rise, with peak levels being reached after 1-3 months. This results in the growth of the penis and testicles. In females, gonadal activation leads to follicular maturation and a fluctuating increase in oestrogen levels.[11]

Serum levels of LH and FSH are low immediately after delivery because of the high levels of placental hormones, which are gradually eliminated over about a week (except for FSH, which remains raised in females for up to three to four years). This releases the suppression, resulting in elevated gonadotropin levels until six to nine months of age.[11,12] Although there is no palpable increase in testicular volume, this hormonal surge during mini puberty in males is crucial for penile growth and somatic growth velocity.[12]

This hormonal surge is caused by the temporary activation of the HPG axis during mini puberty; when compared to females during the first six months of infancy, studies have shown a higher growth rate and a faster gain in weight related to somatic changes in males.[13]

During the first six months of life, boys had a significantly faster growth velocity than girls, according to studies, due to the hormonal surge of mini puberty. The maximum difference in growth velocity was observed at one month of age in males, reaching 4.1 cm per year, and coinciding with the peak of postnatal gonadal activation of the HPG axis.[14]

Penile size measurement

Penile size measurement was first studied in 1942 among male infants by Schonfeld and Beebe in New York City,[15] and the method employed in their study has been largely adopted for measuring penile length with some modifications. In the Schonfeld and Beebe method of measuring penile length, the dorsal length of the penis from the pubic-penile skin junction is measured with a ruler to the nearest 0.5cm; first for the flaccid penis, then for the fully stretched but flaccid penis. Three readings are recommended, and the median value is to be adopted for clinical use. [15] This method has been well documented. [16,17] Though simple and easily reproducible, the metric gradings on the ruler may introduce a visual bias, prompting some other investigators to modify the method.

The modification of the Schonfeld method may include the use of an unmarked wooden spatula instead of a marked wooden ruler. A disposable wooden spatula is applied firmly but gently against the pubic ramus with sustained traction along the length of the penis. The spatula is marked at the level of the tip of the glans penis. The distance between the end of the spatula and the marked point is then measured with a digital Vernier calliper.

A disposable syringe, modified by removing the needle-bearing end and introducing the piston into the cut end, was used in measuring penile length among Turkish children by Ozbey *et al.*[18] The flanged end of the syringe is placed over the penis to be measured, firmly pressed onto the pubis, and the piston is partially withdrawn, thereby causing suction which draws the penis into the injector, excluding the pubic fat. The length of the penis is then read from the graduation on the syringe when the suction is optimal. The syringe method may not be culturally acceptable in the Nigerian setting, with sentiments and bias on the reproductive potential of the male child, though it poses no possibility of injury to the child. This method has been found nonetheless to be less prone to inter-observer variation and very reproducible.

Schonfeld and Beebe, in their study, measured the penile width with graded sizes of rings that easily slip over the glans penis and easily fit the middle of the shaft. The graded sizes of the rings are 3- 12 cm. It is recommended that three readings should be made and the median adopted for clinical use. Nevertheless, this method is cumbersome and will not give exact measurements as the investigator will avoid the ring being too snug, constricting, or getting stuck on the penile shaft.

The Vernier callipers may also be used to measure the width or transverse diameter of the penile shaft at the middle of the shaft (point obtained as the half of median penile length) without applying pressure. A ruler may also be used to measure the transverse diameter in the middle of the shaft, and measurements of penis circumference may be obtained by measuring the middle of the loose penis with a paper ruler. [19] A plastic tube may also be placed around the mid penile shaft, the length of which is then measured to give the penile circumference.[20] These methods, though they appear simple and easily reproducible, may be inconsistent in how tight, firm or snug the plastic tube is to the penile width being measured, resulting in inconsistent measurements. The ruler also may introduce visual metric reading bias.

Testicular volume measurement

Measurement of testicular volume by Schonfeld and Beebe was done by the use of graded models of testicular volumes in a Prader orchidometer.[15] The Prader orchidometer consists of twelve ellipsoid models graded from one to twenty-five millimetres. The volumes in the orchidometer held in one hand were compared with a palpated testicular mass held in another hand. The model with the same volume is taken. Three readings are recommended for each testis, and the median should be adopted as the

estimated volume. Though the Prader orchidometer method offers a more practical, inexpensive method that requires little or no skill compared to ultrasonography, it has the potential to overestimate the testicular volume.[21] The overestimation may be ascribed to inexperience, thickness of the skin, scrotal subcutaneous tissues, and relatively large epididymis size, especially in small testes as found in infants.

Clitoral measurement

The earliest documented method of clitoral measurement in children from extensive searches was by Riley and Rosenbloom in 1980 among neonates in Florida, United States of America.[22] It is recommended that the child be placed in the frog-leg position; the investigator separates the labia majora using the thumb and the second finger of the left hand while an assistant holds the infant's legs. The actual measurement is done with the right hand using a calliper (for a right-handed investigator). The clitoral length is measured from the pubic ramus to the tip of the glans, while the clitoral width is measured by taking the transverse diameter at the middle of the clitoris using a calliper. [22]

Conclusion

Penile and clitoral dimensions with palpably estimated testicular volume are important in the assessment of the normal external genital structure development. Abnormalities in measured dimensions can connote underlying severe disease conditions. Therefore, healthcare providers should be familiar with simple methods of measuring these organs as part of routine postnatal examination and clinical evaluations.

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