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CONSIDERATIONS ON EMBRYOLOGY, ANATOMY AND CLASSIFICATION OF INGUINAL HERNIAS IN CHILDREN

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ABSTRACT

Background: Appropriate surgical treatment of inguinal hernia in children requires a comprehensive review of the embryologic, developmental, and anatomic aspects of the inguinal canal and hernia sac. The presented work represents an extensive survey of data from the literature, predominantly from recent years, and includes information on the knowledge of anatomy, embryology and classification of inguinal hernias, which have direct clinical implications in the surgery of hernia, hydrocele and undescended testis in children.

Keywords: inguinal hernias in children, embryology, vaginal process, classification of hernias.

The problem of approaching appropriate surgical treatment of inguinal hernia in children requires an extensive review of the embryological developmental and anatomical aspects of the inguinal canal and hernial sac [1, 2].

The inguinal canal is a structure of the anterolateral abdominal wall, occupied mainly by the spermatic cord in men and the round ligament in women [3]. This anatomical structure represents an area of complex embryology associated with gonadal development and descent. Embryological development of the inguinal canal occurs in both sexes as the body prepares to receive the migrating gonad, even though such migration occurs only in males [4]. The embryological hallmarks of the inguinal canal can be focused on the vaginal process and gubernaculum, two anatomically important structures for gonadal development [5]. In the male, the formation of the secondary external inguinal ring and spermatic cord duct is due to an evagination of the superficial fascia during the descent and passage of the testis through the inguinal canal into the primitive scrotum. While the testicle is still intra-abdominal, the peritoneum and fascial and muscular elements project and form a sac-like protrusion, the so-called inguinal bursa. The fascia of the muscle's projects into the bursa, not the aponeurosis. The secondary internal groin ring is formed from the membranous layer of the preperitoneal fascia. The classic internal inguinal ring is the edge of the prominent transverse fascia, i.e. the fascia covering the transverse muscle [6]. At the end of the second trimester of gestation, the descending testis passes through the inguinal canal and enters the abdominal subcutaneous space below the external inguinal ring. During this process, the genito-inguinal mesenchymal cord, known as the gubernaculum, connects the lower pole of the testicle to the inguinal bursa. The descending testicle and surrounding inguinal bursa exit through the classic external inguinal ring, which represents the splitting of the aponeurosis

of the external oblique muscle. To the fascia of the muscle is added the external spermatic fascia, as the outer covering. Next, the testicle and inguinal bursa project against the superficial fascia, which evaginates towards the scrotal swelling, a specialised pouch of skin from which the mature scrotum will develop. Most likely to accept the testicle and inguinal bursa, Scarpa's fascia detaches from the fascia wrapping the muscles, thus leaving posterior debris. This creates a structure called the secondary external groin ring. The evagination of Scarpa's fascia forms a tunnel-like projection that looks like a bottle. The mouth of the bottle is the secondary external inguinal ring; the neck is the canal of the spermatic cord, and the lower part covers the inner surface of the scrotal skin, which in the future will become the scrotal fascia Colles [6].

Congenital inguinal hernia in children is always due to failure of habitual closure of the vaginal process, its occurrence being closely related to testicular descent in males and round ligament attachment in females. In girls, the processes vaginalis is known as Nuck's duct and is smaller and more rudimentary than in boys. Hernia may be of the complete or incomplete funicular type [7].

Almost all researchers have found that the incidence of vaginal process permeability is highest in childhood. A persistent vaginal process has been found to be present in 80-95% of all male newborns, in 60% of 1-year-olds and decreasing to 40% in 2-year-olds, thereafter being seen in 15-37% [8]. According to some statistics, the incidence of asymptomatic patent vaginal process in Vietnamese adults is about 7.1% in men, and in women, this rate varies between 12.1% and 3.0%, being observed more frequently on the right side [9]. If the presence of a patent vaginal process invariably leads to hernia, it would be logical to assume that almost all children with unilateral inguinal hernia will develop a contralateral inguinal hernia at some point in their lives. Since this does not happen, we must assume that the process can also obliterate in some cases, or remain open without becoming a hernia.

The vaginal process begins to form in the 8th week of gestation as a caudally directed diverticulum of the peritoneum. It attaches to the ventral surface of the developing gubernaculum, which is a cord of fibromuscular tissue and develops in the fetus between 8 and 12 weeks. The gubernaculum, first named in the 1762 by the Scottish surgeon, John Hunter [10], is the most important structure involved in testicular descent, and there are several theories that explain this descent [11, 12]. The shortening and thickening of the gubernaculum allow the gonads to descend into the pelvis. In boys, under the influence of androgens, the distal gubernaculum continues to grow and enlarge to allow the gonads to descend through the inguinal canal into the scrotum. In girls, in the absence of androgens and anti-müllerian hormone, the gubernaculum interferes with the Müllerian duct and attaches, in its middle portion, to the horn of the uterus, which becomes the ovarian ligament above this attachment. The caudal section becomes the round ligament, attaching the horn of the uterus to the labia majora. This ligament helps to fix the normal anteflexed anteverted position of the uterus and prevent the descent of the ovary into the inguinal canal [13, 14].

According to some studies, in the first phase of intra-abdominal testicular descent, the testicle actively moves from the lower pole of the kidney to the bladder neck. Then, in the second phase, it continues to migrate in the process of development of the peritoneal vaginalis process, caused by the disappearance of the gubernaculum bulb of the testis [15].

Following the gubernaculum and probably the genitofemoral nerve, the vaginal process herniates through the abdominal wall, carrying with it several layers, which include the fascia transversalis, internal and external oblique muscles. The point of entry of the hernia through the abdominal wall,

created by the processus vaginalis, becomes the deep inguinal ring, while the muscle layers are worn with it, forming the inguinal canal. The vaginal process exits the abdominal wall through the aponeurosis of the external oblique muscle at the superficial inguinal ring [16].

Complete obliteration of the vaginal process occurs between 36 and 40 weeks of gestation, and the persistence of all or a part of the vaginal process results in a variety of inguinal abnormalities, such as scrotal hernia, distal obliteration of the process with a proximal hernial sac, communicating hydrocele, which is a hernia with a small communication with the peritoneal cavity, spermatic cord hydrocele, and distal hydrocele of the tunica vaginalis [17, 18]. Although obliteration of the vaginal process can occur prenatally, in about 80% of boys and 60% of girls, the vaginal process is still present at birth [19, 20]. By 8 weeks of age, 63% of boys will have persistent processus vaginalis, with obliteration occurring at any time up to two years. After this age, up to 40% of boys continue to have a persistent vaginal process, with about half remaining asymptomatic throughout their lives [7].

Obliteration of the vaginal process occurs in three stages in boys and in two stages in girls. The initial stage, common to both sexes, involves proximal closure of the deep inguinal ring, sealing the vaginal process from the peritoneum. In boys, this phase is followed by the distal closure of the vaginal process, i.e. the upper portion of the testicle. The tubular structure remaining between these two closures, the funicular process undergoes atresia during the final stage of obliteration, while the distal portion of the vaginal process will form the tunica vaginalis [21]. In girls, the last stage of obliteration is the atretia of the Nuck canal [22], described in 1691 by the German anatomist Anton Nuck [12].

The precise cause of obliteration of the vaginal process is questioned, with several hypotheses proposed, including loss of inhibitory control of the motor neuron of the cremaster muscle, persistence of smooth muscle due to failure of apoptosis, morphological changes in epithelial cells derived from the vaginal process due to growth factors, changes in the connective tissue of the hernial sacs, and defects in the structure of collagen fibers [23].

Some studies indicate that calcitonin gene-related peptide released from the genitofemoral nerve may play a role in fusion [24, 25, 26], and some histopathological research has highlighted the presence of smooth muscle in the pouches associated with inhibition of vaginal process obliteration. Myofibroblast degeneration induces apoptosis of smooth muscle fibres and mesothelium of the vaginal process, causing obliteration. Consequently, disturbances in this process cause disturbances in the normal obliteration of the vaginal process. At the same time, it is known that sympathetic nerves are among the factors that exert trophic changes in smooth muscle by increasing intracellular levels of cAMP via beta-adrenergic receptors, sympathetic innervation being necessary for maintaining smooth muscle [27, 28]. This apoptosis can occur due to decreased sympathetic innervation and increased parasympathetic innervation, which in turn is stimulated by a cascade of hormones. The neurotransmitter, calcitonin gene-related peptide, released from the genitofemoral nerve into the inguinal canal, appears to be the predominant hormone in stimulating closure and obliteration of the vaginal process [27, 29, 30, 31]. Given that the obliteration process involves programmed cell death of smooth muscle cells of the vaginal process, some studies have observed that in cases of patent vaginal process, vascular and mesothelial sac structures reveal evidence of apoptosis, while the smooth muscle component lacks the apoptotic process, concluding that failed smooth muscle apoptosis may play a role in the persistence of the vaginal process [32].

The inguinal canal is an oblique intermuscular passage extending from the inguinal rings, deep to superficial, through which the spermatic cord passes in men and the round ligament in women. The channel boundaries are as follows:

1. Posterior wall, formed laterally by the aponeurosis of the transversus abdominis muscle and the lateral transverse fascia, in three-quarters of subjects; in one-quarter of subjects, the posterior wall consists only of the transverse fascia. Medially, the posterior wall consists of the internal oblique aponeurosis or common tendon;
2. The anterior wall consists of the lateral internal oblique muscle and the aponeurosis of the external oblique muscle. There are no external oblique fibres in the groin area, only aponeurotic fibres;
3. The lining of the duct consists of the lower arcuate fibres of the internal and transverse oblique muscles of the abdomen.

The posterior wall of the canal consists of the fascia transversalis along its entire length. The inferior border of the canal consists of the rolled fibres of the medial inguinal ligament, then the pectineal fascia and the insertion of the lacunar ligament (Gimbernatum) [33].

In newborns and infants, the inguinal canal is not well developed and is very short, with the external and internal inguinal rings overlapping, the external ring being relatively large. Therefore, during opened herniotomy before the age of 1 year, it is not necessary to open the external oblique muscle [34, 35].

According to some reports, the average length of the inguinal canal is 1.0 cm (range, 0.7-1.1) in children under 2 years of age and an average of 1.1 cm (range, 0.7-2.3) in children over 4 years of age. Results from some studies show that the length of the groin is steadily increasing from an average of 0.7 cm at less than 2 months of age to 1.9 cm at over 6 years of age. The length of the inguinal canal does not increase linearly in the first 12 months of life. This finding suggests that the length of the canal is proportionally greater with age and measurement of height and weight in infants compared to older children. Its growth is therefore relatively delayed. The deep inguinal ring is located at the middle of the inguinal ligament as in adults, contrary to what has been reported by some authors, such as Parnis S.J. et al. (1997), who stated that the ring was located medial to the middle of the inguinal ligament throughout childhood [36].

Anatomically, inguinal hernia is the protrusion of the parietal sheet of the peritoneum and abdominal viscera through the opening of the abdominal cavity, which may be normal or abnormal. Based on this conclusion, two categories of inguinal hernia are described: direct and indirect (oblique) [37].

One of the first classifications, based on the principle of hernia location and stage of development, was proposed by Harkins (1959), who divided hernias into 4 grades:

- Grade I - oblique inguinal hernias in children;
- Grade II - simple oblique inguinal hernias;
- Grade III - oblique and direct intermediate inguinal hernias;
- Grade IV - 'complicated' hernias: femoral, recurrent and others [38].

Casten (1967) later divided inguinal hernias into 3 stages, based on the concept of the condition of the deep inguinal orifice of the inguinal canal:

- Stage I: oblique inguinal hernia with undilated deep inguinal orifice;
- Stage II: oblique inguinal hernia with dilated deep inguinal orifice;
- Stage III: direct inguinal and femoral hernia [38].

Indirect hernias, known as „congenital" hernias, are most common in children and are caused by the permeability of the vaginal process. In this type of hernia, the herniated contents pass from the lateral epigastric vessels to the inferior epigastric vessels and enter the deep inguinal ring. Direct hernias, also called „acquired" hernias, occur due to a weakening of the anterior abdominal wall in the inguinal triangle and are more common in adults, although this type can sometimes be seen in adolescents. In direct herniation, the herniated contents pass from the medial to the inferior epigastric vessels, pushing through the peritoneum and transverse fascia into the inguinal triangle („Hesselbach's triangle") to enter the inguinal canal. Both types of inguinal hernias can be unilateral (more often on the right side) and bilateral, clinically distinguished as symptomatic and asymptomatic hernias, and from a surgical point of view, it is important to determine whether the patient has a reducible or non-reducible inguinal hernia [37, 39]. Although direct hernia is predominantly found in adults, it can also be present in children, including newborns [40].

''Hernia en pantalon'' is a combination of indirect and direct inguinal hernias that occur quite rarely in children. Before the introduction of routine inguinal laparoscopy, this form of combined inguinal hernia was not known in children [41, 42].

Incarcerated inguinal hernia presents clinically as a non-fluctuating irreducible intumescence that cannot be reduced by manipulation. In incarcerated hernias, the sac may contain the intestinal loops, omentum or ovary with fallopian tube in girls and obstructive symptoms such as nausea, vomiting, lack of bowel function and abdominal distention may be present, with a tendency to cause serious complications in the absence of correct and timely treatment. In incarcerated hernia there is a risk of strangulation, which is vascular compromise of the contents of an incarcerated hernia, developing symptoms of peritonitis, bloody stools and hemodynamic instability [43, 44, 45].

A study by Sameh Shehata and co-authors of this research (2018) aimed to propose a classification of paediatric hernia and a tailored treatment based on the original Nyhus L.M. (1993) classification for adults [46], with a suggested tailored treatment for each subtype, comparing this approach to the control group of patients treated by classic herniotomy [47]. According to this classification, we distinguish the following types:

- Pediatric Nyhus Type 0 (PN0) - patented vaginal process detected by ultrasonography or during laparoscopy, without clinical herniation, requiring no treatment;
- Pediatric Nyhus Type 1 (PN1) - clinical situation in which hernia is not detected on examination, diagnosed by a reliable history and silk glove sign. Herniotomy alone is used, with or without opening the inguinal canal;
- Pediatric Nyhus Type II (PNII) - hernia occurs immediately after repeated straining, more than 30 seconds and requires manipulation to reduce. Irreducible hernia is also classified as type II. In this case, herniotomy and narrowing of the deep annulus is used and the inguinal canal must be opened;

- Pediatric Nyhus type III (PNIII) - the hernia occurs immediately, on minor or spontaneous exertion, and is very easily reduced with minimal manipulation. Herniotomy is used, plus complete repair of the posterior wall of the inguinal canal, iliopubic tract, up to the tendon part of the articular tendon.

In 1995, Toki A. et al. proposed an ultrasonographic classification for inguinal hernia in children, which included four types. Type I was characterized by the presence of bowel in the inguinal canal; type II included a cystic pattern at the level of the internal inguinal ring; in type III a patent enlarged vaginal process with increased abdominal pressure was determined; type IV was recognized in cases when the vaginal process contained mobile material without enlargement in size [48].

Subsequently, Toki A. (2003) proposed the classification of inguinal hernias into 6 types:

- I - intra-abdominal organ was observed in the inguinal canal;
- II - patent vaginal process was detected as a cyst at the inner ring of the inguinal canal, exceeding 20 mm along the major axis;
- III - in this type, the patent vaginal process is enlarged and shows an increase of abdominal pressure, and its length is equal to or greater than 20 mm;
- IV - the patent vaginal process contains moving fluid, without extension of the vaginal process;
- V - the patent vaginal process is enlarged and there is an increase of abdominal pressure, but the length is less than 20 mm;
- VI - other findings.

The authors concluded that in cases where hernias are sonographically included in types I - IV, it is necessary to perform a contralateral herniorrhaphy [49].

Tanaka et al. (2023) laparoscopically assessed hernia hole size and used the European Hernia Society classification for adolescent and adult patients, according to which hernia hole defect size of ≤1.5 cm was classified as L1; hernia hole defect size >1.5 cm - L2 or 3; hernia hole size greater than 3 cm - L3 [50].

Thus, knowledge of anatomy, embryology and the classification of inguinal hernias has direct clinical implications for surgery in hernia, hydrocele and undescended testis in children. Correct incision placement is necessary to minimise tissue trauma caused by excessive tissue tension, which can result from a misplaced incision. Therefore, knowledge of the detailed anatomy of this region has critical importance to the paediatric surgeon.

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