Trans-scrotal approach (Bianchi) for management of patent processus vaginalis(hernia and hydrocele) versus traditional approach.

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Abstract

The classic inguinal approach for treatment of inguinal hernia has proven its efficacy in all aspects of treatment adequate access to the hernia sac and its meek minimal damage to the gonads, low recurrence rat and an acceptable scar.
Inguinal hernia repair is one of the most common general surgical operations performed by pediatric surgeons and evolution of hernia repair throughout history has led to the conclusion hat a simple removal of the hernia sac herniotomy its sufficient for treatment of pediatric inguinal hermits.

Key Word:

Trans scrotal approach for management of patient processs vaginalis (Hernia and Hydrocele) Versus

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Introduction and aim of the work

A persistent patent processus vaginalis and underlying abnormality lead to the development of the congenital inguinal hernia or hydrocele. The processus vaginalis develops during the third month of gestation as an outpouching of the peritoneal cavity, and extends through the internal and external rings to reach the scrotum during the seventh month and in most males obliterates spontaneously from the internal ring to the scrotum after testicular descend is complete. A patent processus vaginalis is a potential hernia [1,2].

Inguinal hernia repair is one of the most common general pediatric surgical operations performed by pediatric surgeons and evolution of hernia repair throughout history has led to the conclusion that a simple removal of the hernia sac "herniotomy" is sufficient for treatment of pediatric inguinal hernias. The inguinal herniotomy has proven its efficacy in all aspects of treatment: adequate access to the hernia sac and its neck, minimal damage to the gonads, low recurrence rate and an acceptable scar [3].

The true incidence of inguinal hernia is not known, although fairly accurate estimation is available, based on different surveys. The incidence of inguinal hernias in children ranges from 0.8 % to 4.4 % [3], and is higher in infants; commensurate with the higher rate of patent processus vaginalis [4].The incidence of inguinal hernia is highest during the first year of life, with a peak during the first few months. Approximately one third of children with hernia are less than 6 months of age at operation [3]. Incidence is highest in premature infants, with reports ranging from 16% to 25% [5].

Congenital indirect inguinal hernia occur more frequently on the right side, an incidence consistent with the observation that descent of the right testicle proceeds more slowly and, therefore, obliteration of the processus vaginalis occurs somewhat later [6] overall, 55% to 70% of inguinal hernias are diagnosed on the right side roughly 10% are bilateral, and approximately 35% are solitary on the left [7].

Boys are affected approximately six times more often than girls [8]. Reported sex ratios range from 3:1 to 4:1 in favor of males. The relative risk for inguinal hernia is higher in premature infants, but there is no clear evidence of a difference between

genders in these infants. Although **Rajput, Gauderer, and Hack,** [5] reported that in very low birth weight infants 26% of boys developed an inguinal hernia compared to 7% of girls. Others have not found a significant difference [9].

Despite a variety of exposure techniques performed by experienced pediatric surgeons, the conventional ‘‘inguinal’’ approach has been most popular. Other inguinoscrotal surgical procedures, such as orchidopexies and the management of hydroceles and varicoceles have usually been carried out by the same exposure, resulting in a ‘‘comparably good scar,’’ hidden underneath the child’s diapers or underpants.

Bianchi and his colleagues reported their high trans-scrotal approach to the palpable undescended testis and later for inguinal hernias and hydroceles. [10,11,12,13,14,15,16,17,18].The scrotal approach to repairing inguinal hernias and hydroceles in boys is a relatively new approach, which offers the pediatric surgeon the choice of leaving a more cosmetically acceptable scar.

Aim of the work:

In our study patient (boys) with patent processus vaginalis pathologies (hernia or hydrocele) in different age groups will be operated upon through the scrotal approach to evaluate the accessibility of this approach to perform the high ligation of the sac and to compare the result with the traditional suprapubic approach as regard the cosmetic result and the post operative complications (recurrence, testicular atrophy, scrotal hematomas, and local wound complications).

Embryology

Indirect inguinal herniotomy is one of the most frequently performed surgical procedures in children. The overall incidence of inguinal hernias in childhood ranges from 0.8% to 4.4% [19, 20].The incidence is up to 10 times higher in boys than in girls [21].The incidence is much higher in premature infants; inguinal hernias develop in 13% of infants born before 32 weeks gestation and in 30% of infants weighing less than 1000 g [22].

Indirect inguinal hernias in children are basically an arrest of embryologic development rather than an acquired weakness, which explains the increased incidence in premature infants. The formation of inguinal hernias in children is directly linked to descent of the developing gonads [22].

The gubernaculum

The gubernaculum is a condensation of mesenchymal cells occupying the core of the inguinal fold (peritoneal fold) which condense as a cord. This extends from the epidermal ectoderm will later form the scrotum, through the inguinal fold and the mesorchium to the caudal pole of the testis. It traverses the site of the future inguinal canal, which is formed around it by the muscles of the abdominal wall as they differentiate. The gubernaculums precedes the testis both spatially and in rate of growth, forming a tapering column of soft tissue with the diminutive testis at its cranial pole. It continues to grow until the seventh month, by which time its caudal part has filled the future inguinal canal and has begun to expand the developing scrotum [22].

Descent of the testis

The descent of the testis from the embryologic retro-peritoneum begins early in gestation. As the mesonephros (developing kidney) ascends into its usual position in the retro-peritoneum, the testis remain at the level of the internal rings, these processes may be initiated and controlled by the calcitonin gene-related peptide (CGRP) released by the genitofemoral nerve under the influence of fetal androgens.

The final descent of the testis into the scrotum occurs late in gestation between weeks 28 and 36 [23].

Hutson, [24] has proposed a two stage model for testicular descent. During the first stage of descent, known as trans-abdominal descent, the testis is anchored to the inguinal region by enlargement of the gubernaculum. Inguino-scrotal descent occurs between 26 and 40 weeks of gestation, and involves active gubernacular migration, because it has been shown that prior to this phase the gubernaculum ends at the abdominal wall and is not found in the future scrotum. The genitofemoral nerve hypothesis was later proposed to explain inguinoscrotal descent. This hypothesis proposes that androgens released from the developing testis act primarily on the genitofemoral nerve via the central nervous system, the genitofemoral nerve then acts to stimulate gubernacular migration. Subsequently, calcitonin gene-related peptide has been identified as the neurotransmitter present in both the genitofemoral nucleus and genitofemoral branches of the rodent, and is known to cause rhythmic contractions of the whole gubernaculum [25].

The testis are preceded in this descent by the gubernaculum and a ‘‘finger’’ of peritoneum, which ultimately forms the processus vaginalis. This finger or ‘‘diverticulum’’ of peritoneum is first visible around the 12th week of gestation [22].

The processus vaginalis:

At the end of the second month, the caudal part of the ventral abdominal wall is horizontal; but, after the return of the intestine to the peritoneal cavity, it grows in length and progressively become vertical. As a result the umbilical artery pulls up a falciform peritoneal fold, as it runs ventrally from the dorsal to the ventral wall, and this forms the medial boundary of a peritoneal fossa into which the testis project. This fossa is the succus vaginalis or lateral inguinal fossa. Its lower end protrudes down the inguinal canal along the ventroposterior aspect of the gubernaculums, as the processus vaginalis. The distal end of the processus vaginalis, into which the testis projects, forms the tunica vaginalis, but the portion associated with the spermatic cord in the scrotum and in the inguinal canal normally becomes obliterated, usually leaving a fibrous remnant. Sometimes the remnant atrophies completely. Alternatively, its original cavity may persist in whole or in part and in any location. These variations

may form the walls of hernial sacs or encysted fluid sites. Some researchers have suggested that formation of the processus vaginalis is a result of intra abdominal pressure whereas others believe that it is an active process. In normal development, the processus vaginalis closes, obliterating the peritoneal opening of the internal ring between the 36th and 40th week of gestation [26].The distal portion of the processus vaginalis obliterates, except for the part that becomes the tunica vaginalis. This process is often incomplete, leaving a small patent processus in many newborns. However, closure continues postnatally, and the rate of patency is inversely proportional to the age of the child [1, 2, 27].

Although the data are somewhat variable, approximately 40% of patent processus vaginalis close during the first months of life and an additional 20% close by 2 years of age [20].This closure is asymmetric; the left testis descends before the testis on the right. The closure of the patent processus vaginalis on the left also precedes closure on the right; therefore, it is not surprising that 60% of indirect inguinal hernias occur on the right side [22].

Much of the confusion about indirect inguinal hernias in children stems from the assumption that a patent processus vaginalis is the same as an inguinal hernia. The presence of a patent processus vaginalis is a necessary but not sufficient variable in developing a congenital indirect inguinal hernia. In other words, all congenital indirect inguinal hernias are preceded by a patent processus vaginalis, but not all patent processus vaginalis go on to become inguinal hernias. The classic teaching has been that approximately 20% of boys have a patent processus vaginalis at 2 years of age [2]. In other words ,a hernia is not inevitable when the processus vaginalis remains patent, and factors involved in the development of a clinically apparent inguinal hernia have been reported as urogenital anomalies (undescended testis, exstrophy of bladder), the presence of raised peritoneal fluid (ascites, ventriculoperitoneal shunt, peritoneal dialysis), the presence of increased intra-abdominal pressure (repair of exomphalos/gastroschisis, severe ascites, meconium peritonitis), chronic respiratory disease (cystic fibrosis), and connective tissue disorders (Ehlers-Danlos syndrome, Hunter-Hurler syndrome, Marfan syndrome, mucopolysaccaridosis) [8].

It is assumed that closure will continue during childhood for some but not all

patients. **Van Veen** and colleagues [28] studied over 300 adults undergoing unilateral hernia repair. These patients had laparoscopic exploration of the contralateral side; 12% of these patients had a patent processus vaginalis. With a 5.5-year average follow-up, inguinal hernias developed in 12% of adult patients with a patent processus vaginalis, a rate four times greater than in the adults in the study who had a closed ring. An incidence of 12% to 14% has been confirmed in other studies of adults as well [29].

Because the overall incidence of indirect inguinal hernias in the population is approximately 1% to 2% and the incidence of a patent processus vaginalis is approximately 12% to 14%, clinically appreciable inguinal hernias should develop in approximately 8% to 12% of patients with a patent processus vaginalis [29].

Although the embryology is well described, the molecular basis for closure of the patent processus vaginalis is not known. **Tanyel** suggested that failure of regression of smooth muscle (present to provide the force for testicular descent) may have a role in the development of indirect inguinal hernias [30,31], smooth muscle is present in inguinal hernia sacs in children but absent in the wall of hydroceles and hernia sacs associated with undescended testis [30,32]. The mechanism for disappearance of the smooth muscle is not yet elucidated, although mediators of autonomic tone have been suggested to have a role [31,33,34]. Recent studies reported that incomplete obliteration of the processus vaginalis is related to the smooth muscle persistence and the clinical outcome as an inguinal hernia or hydrocele mainly depends on the amount of the persistent muscle [35], moreover, the presence of myofibroblasts, which were suggested to reflect attempted apoptosis by smooth muscle through dedifferentiation into an earlier stage, as an essential step for the obliteration of the processus vaginalis , was found within sacs associated with inguinal hernia and hydrocele [30].

Several studies have investigated genes involved in the control of testicular descent for their role in closure of the patent processus vaginalis, for example, hepatocyte growth factor [34,36] and calcitonin gene-related peptide [34,37,38].

The genetics of inguinal hernias, like the molecular biology, are also poorly understood. There is some genetic risk incurred for siblings of patients with inguinal

hernias; the sisters of affected girls are at the highest risk with a relative risk of 17.8% [39]. In general, the risk for brothers of a sibling is around 4 to 5 times, as is the risk for a sister of an affected brother [39].

Both a multifactorial threshold model and autosomal dominance with incomplete penetrance and sex influence have been suggested as an explanation for this pattern of inheritance [40,41].

Anatomy of the inguinal canal

The inguinal canal is not really a canal in the strict sense of the word. It should be thought of as an area, completely occupied by easily recognizable structures, that begins at the internal inguinal ring, which lies at a point approximately mid-way between the anterior superior iliac spine and the pubic tubercle, and the external inguinal ring, which is located just above and lateral to the pubic tubercle [6]**.**

- ***The superficial inguinal ring:***

The superficial inguinal ring is an aponeurotic hiatus superolateral to the pubic crest, and is somewhat triangular with its long axis parallel to the deep aponeurotic fibers. Its size varies enormously depending on the individual situations. In a simple congenital hernia it is small, whereas in a large direct hernia the ring may be greatly enlarged. Its base is the pubic crest. Lateral to the ring a variable number of fibers running almost transversely between the crura, these are the intercrural fibers. The superficial inguinal ring transmits the spermatic cord in males, uterine round ligament in females, and ilioinguinal nerve in both. From its margins the external oblique aponeurosis continues as tenuous investment around the spermatic cord and testis, the external spermatic fascia. The ring is visible only when this fascia is cut around [42].

- ***Anterior wall of the inguinal canal:***

The fibers of the external oblique run parallel to their lower border, the inguinal (Poupart's) ligament. Above its medial end they diverge from each other to make a V-Shaped opening, the superficial inguinal ring. The lateral crus of this opening is attached to the pubic tubercle, the medial crus to the pubic crest near the symphysis pubis. The intervening part of the pubic crest receives no attachment from the external oblique aponeurosis [43]. At the point of junction of the crura are shining fibers, running at right angles across the external oblique aponeurosis, they bind the crura together, and serve as a visible landmark to the superficial ring at operation, they are the intercrural fibers [43].

The anterior wall of the inguinal canal is reinforced laterally by the lowest muscle fibers of the internal oblique. The deep inguinal ring lies above the midpoint of the inguinal ligament; the internal oblique fibers extend medial to this, for they arise from the lateral two-thirds of the ligament. So the anterior wall of the canal is formed from the skin, superficial fascia, external oblique aponeurosis and in its lateral third, muscular fibers of the internal oblique [42].

- ***The floor of the canal:***

Inferiorly the free lower border of the aponeurosis of the external oblique muscle is folded back upon itself to form the inguinal (Poupart's) ligament. It must be emphasized that this ligament is not thicker than the external oblique aponeurosis.

The medial lowermost margin of the inguinal ligament is free. It is attached firmly at the anterior superior iliac spine and it is also attached to the fascia covering the iliopsoas muscle and the fascia lata of the thigh [44].

The medial extent of Poupart's ligament lies over the femoral vessels. It is attached to the pubic tubercle; those fibers of the external oblique that insert into Cooper's ligament form the lacunar ligament of Gimbernat. The medial half of the inguinal ligament has tenuous attachment to the transversalis fascia, which it overlies. A portion of the ligament continues onto the lower portion of the rectus abdominis sheath, as the reflected inguinal ligament [6].

- ***The roof of the canal:***

This is formed by the arched lower borders of the internal oblique and transversus abdominis muscles. The internal oblique muscle arises by fleshy fibers from the lateral two thirds of the inguinal ligament, the fibers arch medially and downwards emerging into a flat aponeurosis which inserts into the symphysis pubis, pubic crest, pubic tubercle and the pectineal line. Medially the fibers of the internal oblique aponeurosis fuse with those of transversus abdominis to form the "Conjoint tendon" [43]. However, ***Skandalakis,*** stated that, in about 10%of subjects, the termination of the aponeurosis of the internal oblique muscle and the transversus

abdominis muscle meet to form the conjoint tendon. In most individuals, the tendon is absent [45].

The transversus abdominis arises from the lateral half of the inguinal ligament by fleshy fibers deep to those of the internal oblique, they arch downwards to make an aponeurosis that is attached along the pubic crest and extends out along the pectineal line; fusing with the aponeurosis of the internal oblique. The lowermost fibers of the internal oblique and transversus abdominis are supplied by the ilioinguinal nerve.

- ***Posterior wall of the canal:***

Medially this consists of the reflected inguinal ligament and the strong conjoint tendon if present. Lateral to the conjoint tendon there is only a thin posterior wall, the gap between the arched roof (internal oblique and transversus abdominis muscles) and the floor (the inguinal ligament) being covered by the weak alveolar tissue of the transversalis fascia and peritoneum [43].

- ***Transversalis fascia:***

Is a thin areolar stratum between the transversus abdominis and extraperitoneal fat. In the inguinal region it is thick, dense and augmented by the attenuated aponeurosis of transversus abdominis, but it thins as it ascends to blend with the inferior diaphragmatic fascia. Behind, it fuses with the anterior lamina of the thoracolumbar fascia. Below it is attached along the whole iliac crest between the transversus abdominis and iliacus and to the posterior margin of the inguinal ligament from the anterior superior iliac spine to the femoral vessels, where it is continuous with the iliac fascia. Medial to these vessels it is thin and fused to the pecten pubis behind the conjoint tendon, with which it blends [42].

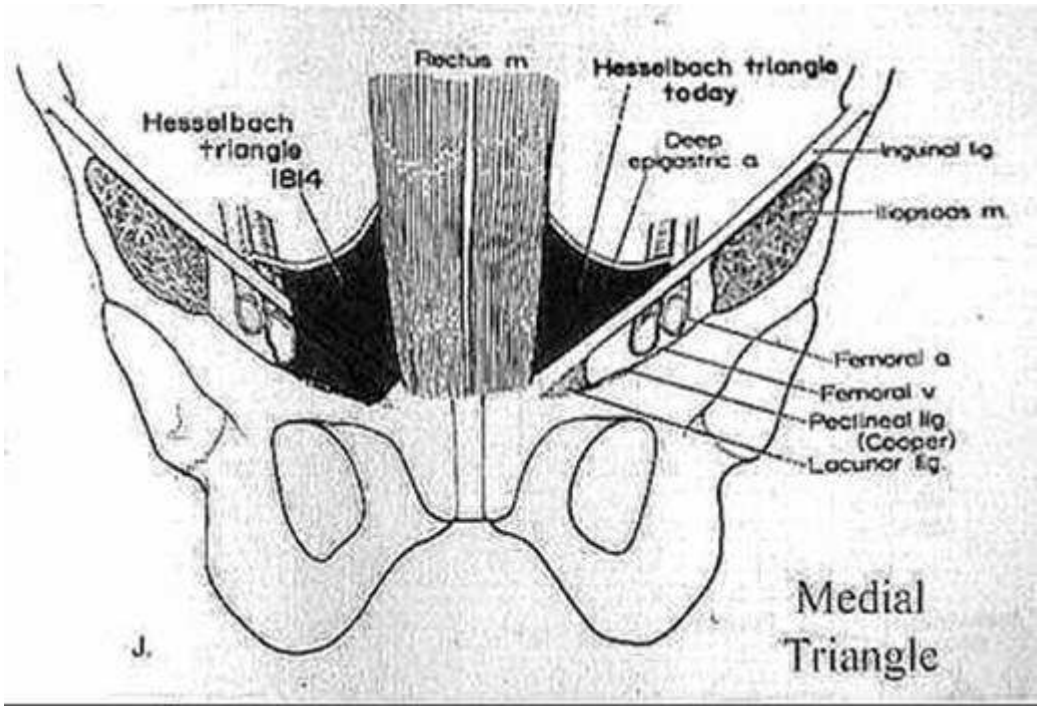

Figure no.1 (Hasselbach triangle) [43].

A number of terms are used to designate structures that are derivatives of the transveralis fascia. These include the transversalis fascial sling, a portion of the femoral sheath, the iliopubic tract (Thomson's ligament), the internal spermatic fascia, and the internal inguinal ring.

The transversalis fascial sling is located at the internal abdominal ring; it may be considered a semicircular or curved condensation of the transversalis fascia on the medial aspect of the spermatic cord. The superior prolongation of this sling is known as the superior crus, the inferior crus of the sling lies just above the iliopubic tract. The iliopubic tract fibers run parallel to the inguinal ligament and become continuous with the anterior femoral sheath [6].

- ***The deep (internal) inguinal ring:***

It lies in the transversalis fascia midway between the anterior superior iliac spine and the symphysis pubis; it is about 1.25 cm above the inguinal ligament; oval, its long axis is vertical; it varies in size, being larger in males. It is related above to the lower margin of transversus abdominis, medially to the inferior epigastric vessels and