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Review Article

Enigmatic aetiology of inguinal hernia

Ketan Vagholkar*, Suvarna Vagholkar

Department of Surgery, D. Y. Patil University School of Medicine, Navi Mumbai, Maharashtra, India

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***Correspondence:**
Dr. Ketan Vagholkar,
E-mail: kvagholkar@yahoo.com

ABSTRACT

The aetiology of inguinal hernia continues to be a topic for debate. A wide range of hypothesis have been postulated over a period of time. These are based on genetic factors, tissue configuration, biochemical enzymatic activity and anatomical structural weaknesses. None of these except the anatomical basis has helped in the evolution of hernia surgery. The article reviews the contemporary hypothesis postulated for the aetiology of inguinal hernias.

Keywords: Aetiology, Hernia, Inguinal, Groin, Pathology, Risk factors

INTRODUCTION

Inguinal hernia is one of the commonest abdominal wall hernias. Various theories were postulated for preventive mechanisms in normal adults. However surprisingly none of these mechanisms are taken into consideration while devising operations for treating inguinal hernias. So, the aetiology of inguinal hernia continues to be an enigma for surgical researchers. Review of literature has revealed variable factors and mechanisms which could possibly explain the formation of an inguinal hernia. The various mechanisms are discussed in this brief review on aetiology of inguinal hernias.

REVIEW OF LITERATURE

The life time risk of developing an inguinal hernia is 27% for men and 3% for women. The inguinal canal starts at the deep inguinal ring and ends at the superficial ring. It transmits the spermatic cord in males and round ligament in women. The integrity of the abdominal wall depends upon the orientation of the inguinal canal, transversalis fascia and the sphincter like function of the deep ring. Inguinal hernias can further be divided into two subtypes direct which emerges from the Hassel Bach's triangle and indirect type which emerges from the deep ring and traverses all along the cord or inguinal canal to emerge from the superficial ring to enter the scrotum.

Age is an important factor in the aetiology of inguinal hernia. It is closely related to the hormonal changes occurring with age.

Older men have altered sex hormone levels leading to weaker muscle tissue and scarring. Hong Zhao et al found a link between hormones and hernias accidentally while experimenting on a murine model for breast cancer research.[1] They had created humanized transgenic mice by genetically modifying them to carry the human gene for aromatase. Aromatase is a key enzyme for conversion of testosterone into oestrogen. The mice expressed human aromatase and had higher levels of muscle tissue oestrogen compared with the control group of mice. The investigators further noticed that male mice used for breeding could not walk well and had swollen abdomens. The lower abdomen showed tissue atrophy leading to weakening and extensive fibrous scarring comparable to that observed in human muscle tissue specimens from patients who had developed a hernia and had undergone inguinal hernia repair. Furthermore, treatment of such

animals with aromatase inhibitors entirely prevented the muscle cell atrophy, fibrosis and herniation thus supporting the central role of oestrogen in the aetiology of inguinal hernia seen in aging men. Hence a corollary which follows this observation is that therapy with aromatase inhibitors could possibly prevent the occurrence of a hernia or prevent recurrence.[1]

Body mass index (BMI) has been linked to the aetiology of inguinal hernia.[2,3] However the association at times seems to be confusing.[4] Low BMI predisposes to inguinal hernias while high BMI is associated with high recurrence rate after surgery. Therefore, a normal BMI would be protective in subjects.[4,5]

Smoking increases the risk of developing a hernia. The possible mechanism is decreased collagen synthesis with increased collagen degradation by the human fibroblasts.[6]

Increased exposure to high intraabdominal pressures increases the propensity to develop a hernia.[7] Straining at stools or while passing urine is a risk factor. Heavy weight lifting activity exceeding 6 hours a day may predispose to herniation as well.[7]

Connective tissue alterations have a significant role to play in the aetiology of inguinal herniation.[8] Changes in the tissue take place at different levels.[9] There is alteration of collagen fibres, tissue architecture and at three levels of enzymatic action in the connective tissue homeostatic process. Collagen is the most important fibre in connective tissue and the ratio and cross linkage between the thick type I and type III fibres largely determine the tensile strength and mechanical stability of the connective tissue. Patients suffering from hernias have a lower collagen I to III ratio in the abdominal wall as compared to controls.[9] Various other studies have revealed decrease in type I and increase in type III collagen in indirect hernias.[8,9] Effectively the collagen fibres are thinner thereby contributing to herniation. There is also significantly reduced collagen in the transversalis fascia and rectus sheath with advancing age thereby explaining the increased predisposition to herniation with age. Patients suffering from direct hernias have less collagen and more abundant and disorganised elastic fibres. Young men with thinner rectus sheaths have a higher incidence of direct hernias.

The enzymes which are potentially involved in inguinal hernia development are matrix metalloproteinases (MMP) which digest proteins of the extracellular matrix during the course of tissue homeostasis and lysyl oxidase which cross links collagen and elastase.[10,11] Increased MMP leads to altered collagen ratios seen in hernia patients by way of activation of cytokine transforming growth factor.[12,13] Increased MMP levels are seen in the transversalis fascia of hernia patients. MMP is a copper dependent enzyme. Therefore, decreased copper levels lead to decreased activity as seen in the transversalis fascia of patients suffering from groin hernias.[14,15] The net effect of connective tissue disarray is altered and weakened abdominal fascia architecture, larger collagen degradation and weakened transversalis fascia due to altered enzyme activity. This makes one think as to whether hernia is an end result of systemic alteration of connective tissue.[16,17]

Genetic predisposition was attributed to development of hernias.[18] Complex multifactorial inheritance clustered in families may perhaps predispose to hernias. Susceptible loci involved in connective tissue homeostasis seems to be the most plausible explanation.[19,20]

DISCUSSION

Though various theories to explain the aetiology of inguinal hernia have been postulated yet the traditional theories viz. patency of the processus vaginalis and a weakened or deficient local anatomy still hold true. This is evidenced by the fact that all repairs focus on these two anatomical issues. The status of the processus vaginalis cannot be overemphasized. A patent processus vaginalis is a risk factor for developing indirect inguinal hernias.[21] Asymptomatic patent processus has been reported in 20% of patients aged five months, 9% at 12 years and 6-19% of adults.[22] This suggests that other factors superimposed on a persistent processus vaginalis lead to the development of an indirect inguinal hernia. Persistent smooth muscle cells are found in unobliterated processus vaginalis.[23,24] Smooth muscle cells are proposed to aid in testicular descent by pushing the testis into the scrotum. Thereafter apoptosis of the cells may facilitate obliteration of the processus vaginalis. Smooth muscle cells have been found to be more frequent in sacs from inguinal hernias than from hydroceles and undescended testis in addition to insufficient apoptosis and absence of apoptotic nuclei in the smooth muscle cells from the processus vaginalis.[25] They have also been found as local thickenings around the internal ring of indirect hernias indicating inadequacy of obliteration of the processus vaginalis thereby predisposing to herniation.

Failed apoptosis could also be related to the sympathetic nervous system which enhances smooth muscle growth and survival of smooth muscle cells in vitro.[25]

Androgens indirectly regulate the descent of the testis by acting on the genitofemoral nerve possibly by releasing calcitonin gene related peptide.[26,27] In vitro studies have indicated induction of the obliteration of the processus vaginalis by transforming epithelial cells to mesenchymal cells.[28] However calcitonin gene related peptide was only associated with mesenchymal fibroblasts. Fibroblasts secrete hepatocyte growth factor.[29,30] The effect of calcitonin gene related peptide could be mediated by this growth factor acting directly on the epithelial cells.[31,32]

After considering a wide variety of factors the final factor which needs consideration is the transversalis fascia.[33] Dissection of normal groins for non-hernia diseases such

as lipoma of the cord or for varicocele ligations revealed two anatomically distinct layers. A weak transversalis fascia and a strong aponeurotic layer formed by the variable aponeurotic fibres sent by the arching transversus abdominis muscle. The contribution of the latter determines the strength of the posterior wall of the canal.[33] In normal inguinal canals there is a full cover of aponeurotic extension though the density of fibres varies from individual to individual with posterior wall being strong and dynamic. In patients with inguinal hernias there was either absence of this expansion or deficient length of these extensions. As a result, the posterior wall was weak, flabby and adynamic.[34,35] The posterior wall in these cases is extremely weak and cannot offer any protection against herniation. Transversalis fascia by itself is incapable of protecting against herniation. Muscular contraction of the transversus abdominis pulls the posterior wall and the aponeurotic extensions upward and laterally creating increased tension or tautness. This offers protection and prevents internal pressure from causing herniation. This is supposed to be the biggest protective factor. Therefore both the physical strength and dynamic nature of the posterior wall prevent herniation.[35,36] An adynamic and weakened posterior wall of the inguinal canal either due to absent or deficient aponeurotic extensions from the transversus musculoaponeurotic arch is the main anatomical factor leading to the formation of an inguinal hernia.[34,35] Hence in hernia repairs maximum stress is focussed on strengthening the posterior wall..

CONCLUSION

A wide spectrum of hypothesis has been postulated to explain the aetiology of an inguinal hernia. However, none of the hypothesis can emphatically justify the cause of a hernia. The effective end result still continues to be a weakened posterior wall which needs reinforcement. Continued research and better understanding will improve stratification and timing of preventive and therapeutic measures by using biological information and biomarkers on the level of molecular disease pathways, genetics, proteomics as well as metabolic processes. Innovative methods such as adding stem cells to meshes may in future revolutionize surgery for hernia repair.

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