

Histological findings of the internal inguinal ring in patients having indirect inguinal hernia

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Abstract

Background Aiming to deepen the understanding of the factors involved in the genesis of groin hernia, this study is focused on identifying the histological changes within the muscle fibers of the internal inguinal ring in patients having indirect inguinal hernia.

Methods In eight patients with primary or recurrent bilateral indirect inguinal hernia who underwent a Stoppa open posterior inguinal hernia repair, a tissue specimen from the edge of the internal inguinal ring was biopsied and histologically examined.

Results In all of the tissue samples, remarkable degenerative changes such as fibrohyaline degeneration of the muscle fibers, vascular congestion, and phlogistic infiltration through lymphohistiocytary elements was constantly detected. Also, in the patients with recurrent hernia, the key characteristic of the muscular change was that of fibrohyaline and, occasionally, myxoid degeneration of the myocytes. Nerve endings were frequently detected within the muscular structures of the internal inguinal ring.

Conclusion The degenerative fibrohyaline alteration, as well as the evidence of phlogistic elements within the examined structures, could represent a reason for a contrac-

tile incompetence of the internal inguinal ring. Consequently, the described findings lead the authors to depict this inflammatory degenerative structural weakness of the internal inguinal ring as a possible culprit of indirect inguinal hernia formation.

Keywords Inguinal hernia · Indirect etiology · Inguinal ring · Sphincter · Inflammation · Fibrosis

Introduction

Even today after over 100 years of scientific study, hernia genesis is still an exciting issue. Many scientists are constantly engaged with studies on this topic, looking to establish all of the factors involved in the development of the disease. Regardless of progress with surgical technique and implants, problems such as recurrence, postoperative complications, chronic pain, and patient discomfort are still a reality for the surgical community. The existence of all these problems may probably lie in the so far not fully disclosed reasons why a hernia appears. Recent scientific articles related to biochemical investigations have suggested that modification in collagen structure may play a role in inguinal hernia formation [1–3]. Nevertheless, even in the latest published literature, no report deals with the histological changes of the muscular structures involved in hernia development. Aiming to fill a lack of knowledge on this subject, we have focused our attempts in detecting structural modification of the tissues involved in the hernia protrusion. To achieve our goal, we have focused on performing tissue biopsy from crucial anatomical structures of the hernia complex, choosing patients which have the most frequent hernia type, the indirect inguinal hernia, representing 2/3rds of all hernias in the human [4].

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Methods

From March 2008 to June 2008, in eight patients who underwent open posterior hernia repair according to Stoppa, biopsies were taken from the internal inguinal ring for histological study. All of the patients had bilateral indirect inguinal hernia. Among these, two patients had recurrent inguinal hernia on one side of the groin. In the entire cohort, we excised a strip of muscular tissue from the upper border of the internal inguinal ring having a length of circa 1 cm, as well as 0.5 cm height and thickness. All of the tissue specimens were fixed in 10% neutral buffered formalin for at least 1 h. After routine tissue processing, sections were cut at 46 μ m and stained with hematoxylin and eosin. The sections were microscopically studied.

Results

The histological outcomes have demonstrated several remarkable changes concerning the structure of the internal inguinal ring. This was seen in all biopsy samples. In our opinion, the most significant commonality was the occurrence of diffuse hyaline or fibrohyaline degeneration of the muscle fibers (Figs. 1 and 2). Also, in the two patients with recurrent hernia, the muscular structures were affected by fibrohyaline degeneration (Figs. 3 and 4) and, in addition to these findings, a myxoid degeneration of the muscle structure was also present (Fig. 4). In many samples, the muscular structures showed clear evidence of an inflammatory response shaped by phlogistic infiltrate and distinguished by lymphohistiocytary elements (Figs. 2 and 3). Venous congestion was constantly detected (Figs. 1–4). Also in line with previous anatomical literature [5], we noted nerve endings in the tissue samples (Figs. 4 and 5).

Discussion

We have performed this study aiming to define if and which alterations are eventually evident in the surrounding structures close to the hernia sac. In performing the described experience, we decided to deepen the histological knowledge of the most common hernia type, the indirect inguinal. To achieve this, in eight patients who underwent a standard Stoppa open posterior procedure for bilateral inguinal hernia, for histological examination, we sampled tissue biopsies from the internal inguinal ring. The results were identical for all of the patients: the muscular frame of the internal inguinal ring revealed massive changes in its muscular substrate, resulting in degenerative alteration of the muscle fibers, as well as venous congestion. We could also see evidence of a considerable inflammatory reaction

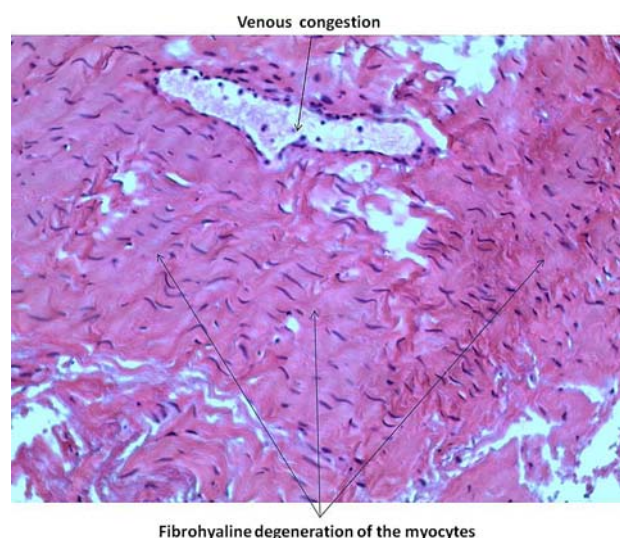


Fig. 1 Primary indirect inguinal hernia: diffuse fibrohyaline degeneration within the muscle fibers of the internal inguinal ring. Venous congestion (H&E \times 18)

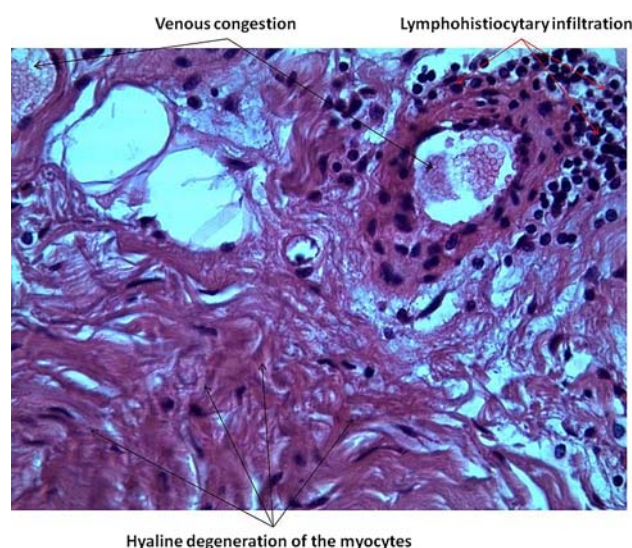


Fig. 2 Primary indirect inguinal hernia: hyaline muscle degeneration, venous congestion, and lymphohistiocytary infiltration in the internal inguinal ring (H&E \times 18)

confirmed by the occurrence of lymphohistiocytary infiltration. Moreover, in addition to these outcomes, in the patients with recurrent hernia, the hyaline degeneration was accompanied by marked fibrotic and myxoid modification of the muscular fibers. Not fully unexpected was the histological finding of nerve endings in the small tissue strips of this peripheral, although highly specific, muscular formation. The demonstration of nerve endings in this area supports the suggestions and corresponds to the statements of numerous scientists engaged with the subject of hernia. As imagined by several authors, the internal inguinal ring moves like a shutter mechanism in order to impede the

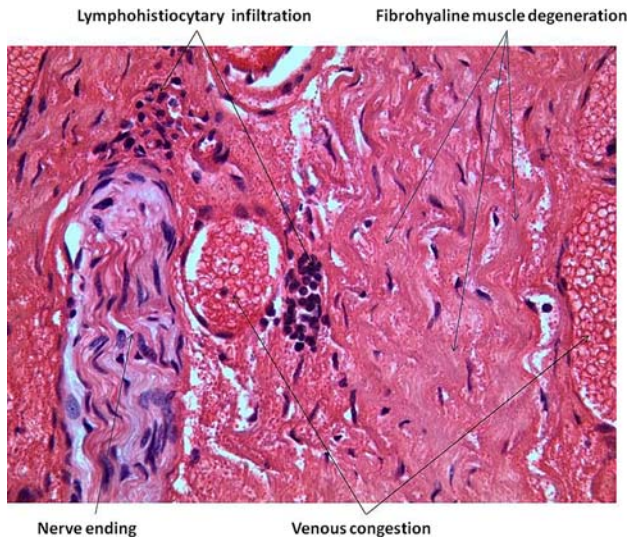


Fig. 3 Recurrent indirect inguinal hernia: inflammatory infiltration, vascular congestion, and fibrohyaline muscle degeneration in the internal inguinal ring. Presence of nerve ending (H&E $\times 18$)

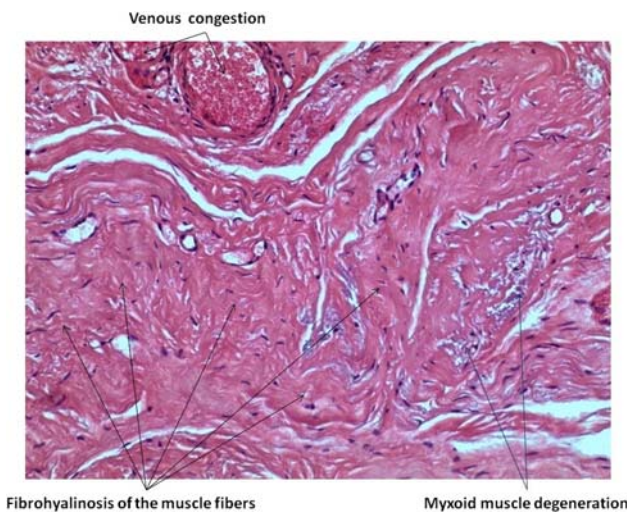


Fig. 4 Recurrent indirect inguinal hernia: lymphohistiocytary infiltration and venous congestion. Myxoid and fibrohyaline degeneration of the muscle fibers in the internal inguinal ring (H&E $\times 18$)

passage of the abdominal content through the inguinal canal if the abdominal pressure rises [6–9]. This closure movement, as depicted in a previous scientific report, is clearly consequent to a nervous impulse [10]. In our opinion, the presence of nerve endings within the muscle fibers of the internal inguinal ring has to be considered as obvious. However, if a nerve stimulates a muscular frame with such significant structural changes like fibrohyaline and myxoid degeneration, we must ask “is it able to adequately contract?”

With respect to the internal inguinal ring, can such as muscular structure, if dramatically altered, effectively close off the passage of the viscera, impeding the protrusion of

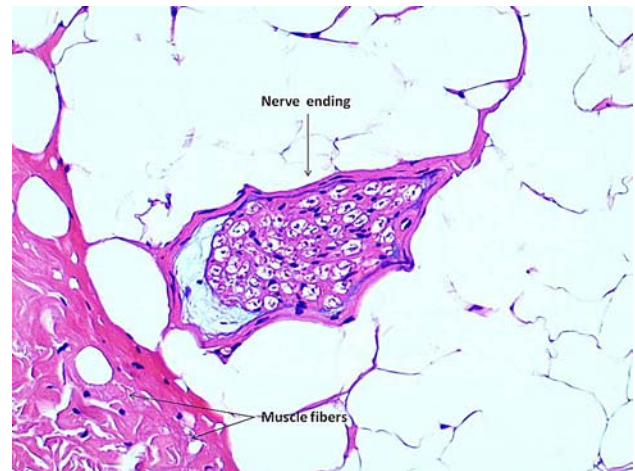


Fig. 5 Primary indirect inguinal hernia: nerve ending close to the muscle fibers of the internal inguinal ring (H&E $\times 18$)

the abdominal content? And, if the structural insufficiency of the shuttering motion of the internal ring increases over time, could this represent a reason for a permanent protrusion of the abdominal viscera and hernia formation? Why should this inflammatory damage happen and what causes the presence of the phlogistic elements and inflammatory changes in the muscle? All these elements together probably represent a key to a possible explanation as to why hernia occurs: somehow, an inflammation involving the structures of the groin occurs. The delicate function of the internal inguinal ring becomes impaired because of its weakness following the muscle fiber degeneration. Extending this line of thought, we think that with every increase in abdominal pressure, the inguinal ring remains progressively more patent. Thus, a hernia can gradually protrude. These suggestions also help to explain why an inguinal hernia is infrequent in the case of patent processus vaginalis [11]. In fact, despite the patency of the inguinal canal, if the shuttering motion of the internal inguinal ring is effective, the protrusion of abdominal viscera through the inguinal ring will be avoided thanks to the efficient contraction of this muscular frame. Otherwise, if the internal inguinal ring, due to impaired muscular function, has been damaged, the occurrence of an indirect inguinal hernia is possible. Is this explanation too simple? Maybe. Many aspects have still to be verified and discussed with regard to this theory. We hope that the initial findings could represent a useful starting point to unify theories around hernia genesis.

Conclusion

This report describes and discusses the findings of histological examination in tissues excised from the internal inguinal ring in eight patients who underwent open posterior

hernia repair for bilateral indirect hernia. The main histological findings demonstrate the presence of massive fibrohyaline degeneration of the muscle fibers of the internal inguinal ring as well as a phlogistic infiltrate, represented by lymphohistiocytary elements, and venous congestion within the muscular frame. A reflection is made on the above mentioned results, considering the relationship between the pathological changes within the structure of the internal inguinal ring and the impairment of the shuttering action of this muscular formation due to these pathological changes. The authors believe that these structural changes to the inguinal ring could be significant enough to impair the important shuttering action and lead to inguinal hernia.

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