

Damage to the vascular structures in inguinal hernia specimens

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Abstract

Background Few scientific reports to date describe the histological modification of structures outlining a hernia opening. This article is focused on the identification of the pathological changes in vascular structures in tissues excised from cadavers with inguinal hernia. A deeper comprehension of this topic could lead to essential improvements in the detection of hernia genesis.

Materials and methods Different kinds of hernia, including indirect, direct and mixed, were identified in 30 autopsied subjects. Tissue samples were resected for histological study from abdominal wall structures close to the hernia opening. Histological examination focused on the detection of structural changes in arteries and veins. The results were compared with tissue specimens excised from equivalent sites of the inguinal area in a control group of 15 fresh cadavers without hernia.

Results Significant modification of vascular structures were identified in the tissue specimens examined. The veins demonstrated parietal fibrosis, perivascular edema and vascular dilation due to congestion and stasis. The arterial structures detected showed thickening of the media due to medial hyperplasia, ranging from luminal sub-occlusion to a manifest artery occlusion. These findings are present independent of hernia type in cadavers with

inguinal hernia. These pathological changes were lacking in the control group of cadavers without hernia.

Conclusions The notable changes in vascular structures described in the report could be the result of a steady compressive effect exerted by the abdominal viscera in the inguinal area. These pathological changes could represent one of the factors involved in the weakening of the inguinal region leading to hernia protrusion.

Keywords Inguinal hernia · Etiology · Blood vessel · Venous congestion · Artery obstruction

Introduction

Groin hernia embodies one of the most frequent surgical diseases, resulting in over 800,000 hernia repairs carried out each year in the United States alone [1]. Despite all the efforts made to research its underlying origin, the pathogenesis of inguinal hernia remains poorly understood. Conversely, much effort and many resources have been devoted to developing new materials and devices for hernia repair. At this point an essential question should be asked: how can we manage a disease without understanding the effective basis of its origin? Moreover: does the reason for the current (still high) rate of complications and recurrences lie in this unanswered question [2–6]? This provides, in our opinion, valuable motivation in the search for a new etiological model aimed at disclosing how and why inguinal hernia occurs. We believe there is a need for improved knowledge of tissue modifications in the area of hernia protrusions. Continuing a series of clinical investigations carried out on biopsy specimens excised from the structures surrounding the hernia opening [7], we describe histological changes detected in the groin structures of

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cadavers with inguinal hernia. In this report, we focused our interest on the occurrence of alterations of the vascular system in the area of the herniated muscular wall.

Materials and methods

Primary groin hernias were recognized in 30 fresh male autopsied subjects. In these cadavers we identified: 11 indirect inguinal hernia type 1 according to Nyhus; 8 indirect inguinal hernia type 2; 6 direct inguinal hernia type 3a; 5 other cadavers with direct inguinal hernia type 3b. The autopsies were performed 24–48 h after death. The mean age was 68 years (range 45–82 years). In all these cadavers, tissue samples for histological examination were excised from the structures close to and surrounding the hernia opening. In order to generate properly controlled data for comparison between subjects with and without hernia, we developed a standardized procedure for specimen excision. This concerned taking biopsies from tissue corresponding to the same zones in cadavers with and without hernia, despite the presence of different anatomical landmarks. In cadavers with hernia we utilized the hernia opening as the key landmark (Fig. 1). Bisecting the hernia defect with a vertical line, we used the central point of the lower border as the initial point. From here a line was drawn at 45° to the right of vertical, out until the upper border of the defect was reached. Distances along this line of 0.5, 1.5 and 2.5 cm away from the border were measured and a full thickness biopsy of 0.5 × 0.5 cm was taken at each point.

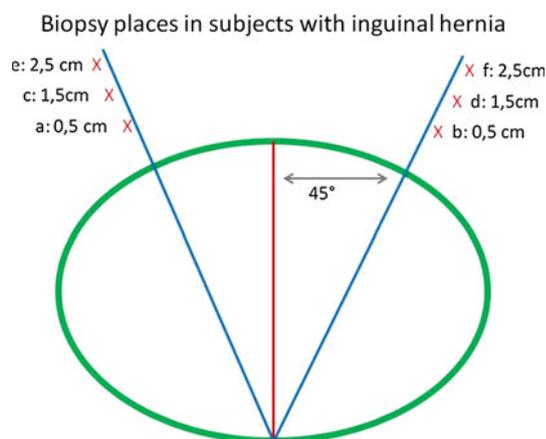


Fig. 1 Green ring Internal inguinal ring in case of indirect inguinal hernia, or hernia opening in case of direct inguinal hernia. A red line connects the lowest and the highest point of the hernia opening. Starting from the lowest point, a 45° inclined blue line transverses the figure on the right and left. X Excisions: a, b 0.5 cm above the junction of both 45° angled lines; c, d 1.5 cm above the junction of both 45° angled lines; e, f 2.5 cm above the junction of both 45° angled lines

The practice was repeated at 45° to the left of the vertical; again a line was drawn along this angle, then measurements taken along this line above the hernia border of 0.5, 1.5 and 2.5 cm, at which point biopsies were taken. This procedure was carried out in both direct and indirect hernias. In order to achieve an adequate control, biopsies were taken from the inguinal region in 15 male cadavers without hernia. The control autopsied subjects had a mean age of 65 years (range 48–79 years). To take control specimens from cadavers without hernia, two measurement sets were created. One to control against direct hernia and one to control against indirect hernia. For indirect hernia, the exact procedure described above was imitated using the internal ring as the landmark instead of an actual indirect hernia. However, for direct hernia we had to modify the procedure to account for the hernia defects of the direct hernia being displaced from the inguinal ring. To achieve a control sample for direct hernia we used the inguinal ligament as the key landmark. Parallel to this ligament and above it we drew three lines at a distance of 0.5, 1.5 and 2.5 cm. We then excised biopsy samples every 0.5 cm along these three lines, starting 0.5 cm medial from the epigastric vessels until 0.5 cm from the lateral border of the rectus muscle. In this way we realized an acceptable histological mapping of the entire fossa inguinalis media. Although not a perfect solution, we felt that this repeatable measurement method of direct and indirect hernia, and direct and indirect controls gave us a significant comparison of tissue samples from the same anatomical areas in cadavers with hernia and controls without hernia. All tissue specimens were fixed in 10% neutral buffered formalin for at least 12 h. After routine tissue processing, sections were cut at 4–6 mm and stained with Azan-Mallory, Weigert van Gieson, leucocyte common antigen (LCA) and hematoxylin eosine (H&E).

Results

Several pathological changes were observed in cadaver tissue specimens excised from the structures surrounding the hernia edge. Among these, a constant histological finding was the presence of inflammatory infiltrate, characterized by plasmocytic and lymphocytic elements. In the venous structures present in the tissues close to the hernia opening, we noted various alterations such as parietal fibrosis, perivascular edema and vascular dilation due to congestion and stasis (Figs. 2, 3, 4). Still greater histological changes can be reported in relation to the arterial structures. These occasionally showed parietal fibrosis (Fig. 4), but the main and recurrent evidence was thickening of the media due to hyperplasia of the

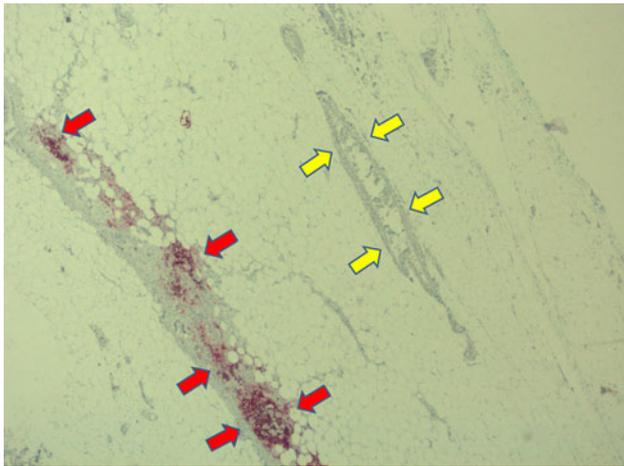


Fig. 2 Biopsy specimen excised 0.5 cm from indirect inguinal hernia border (Nyhus type 1). Note inflammatory infiltrate composed of lymphocytic elements (*red arrows*). Broad venous structure showing sclerosis of the wall (*yellow arrows*) surrounded by a fatty substitution of the muscle cells (*white circular elements*). Staining: leucocyte common antigen (LCA); magnification $\times 2.5$

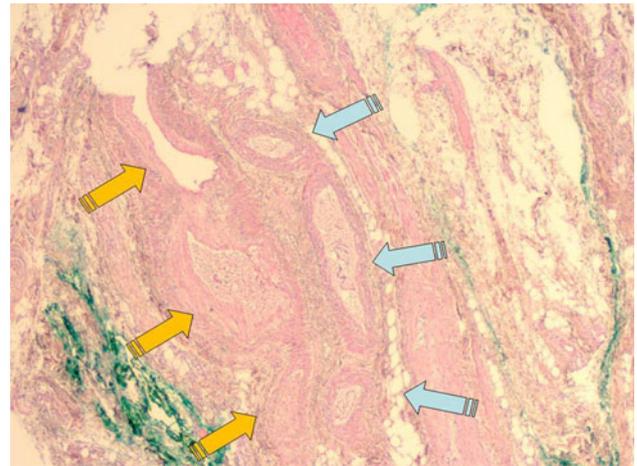


Fig. 4 Biopsy specimen excised 0.5 cm from mixed inguinal hernia border (Nyhus type 3b). Evident stasis and ectasia of the arteries (*yellow arrows*) and veins (*blue arrows*). All the vascular structures reveal signs of parietal fibrosis, more evident in the vein. These features could represent the result of chronic compressive damage. Staining: H&E, magnification $\times 2.5$

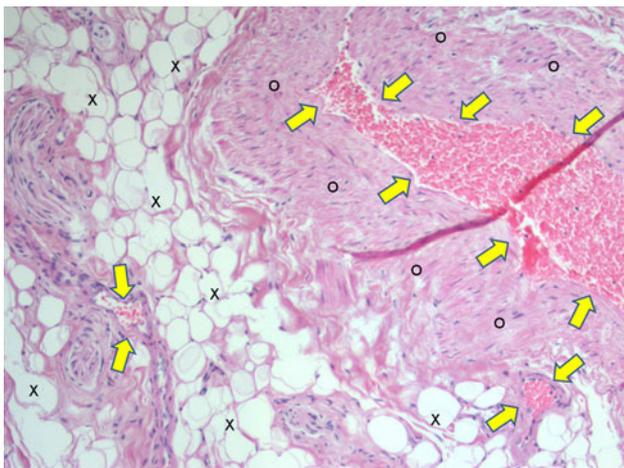


Fig. 3 Biopsy specimen excised 2.5 cm from indirect inguinal hernia border (Nyhus type 2). Evident vascular congestion (*yellow arrows*), fatty dystrophy (X) and fibrotic degeneration of the muscle fibers (O). Staining: hematoxylin eosine (H&E), magnification $\times 10$

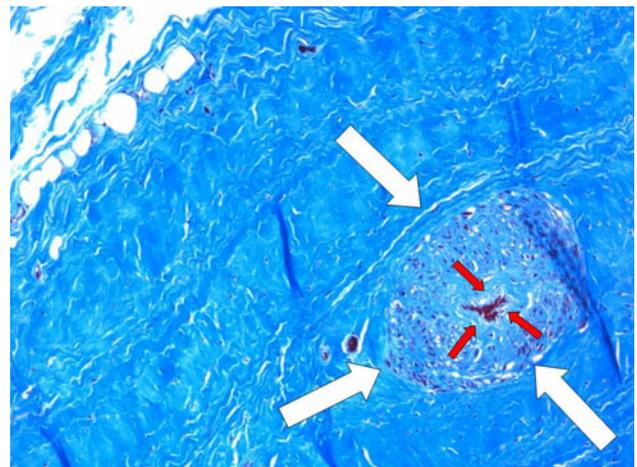


Fig. 5 Biopsy specimen excised 2.5 cm from mixed inguinal hernia border (Nyhus type 3b). Artery media hypertrophy with sub-occlusion (*red arrows*) due to medial thickening resulting from muscular hyperplasia (*white arrows*). Hyaline and fibrotic degeneration of the neighboring muscle fibers. Staining: Azan-Mallory, magnification $\times 10$

muscularis tunica. These structural alterations of the artery were so evident that many vessels having a luminal sub-occlusion or even a manifest occlusion were identified (Fig. 5). The muscle fibers close to the hernia border were consistently altered, showing various degrees of dystrophic degeneration with a variety of stages and types of lesions from fibrohyaline degeneration to fatty substitution (Figs. 2, 3, 5). Even the commonly found nerve trunks revealed clear signs of progressive degenerative changes in nerve axons. These findings were seen regardless of the hernia type and site of excision. Conversely, in the control specimens, no comparable degree

of structural change to vascular structures was seen. In the majority of the control samples, the vessels demonstrated a regular structure without significant alterations (Fig. 6). Only in a few middle-aged cadavers could a mild parietal fibrosis of the veins be detected. No vascular congestion or dilation was seen. In some control cadavers, the artery showed age-related subintimal fibrosis of the arterial media; however, without thickening of the artery wall or sub-occlusion (Fig. 7).

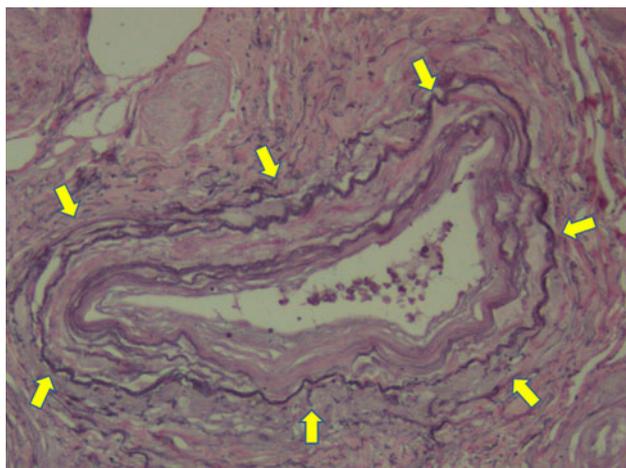


Fig. 6 Control cadaver aged 40 years. Specimen excised 1.5 cm lateral from internal ring. Healthy arterial structures (*yellow arrows*). The elastic fibers of the artery media are normal. Evident patency of the artery lumen. Staining: Weigert van Gieson, magnification $\times 20$

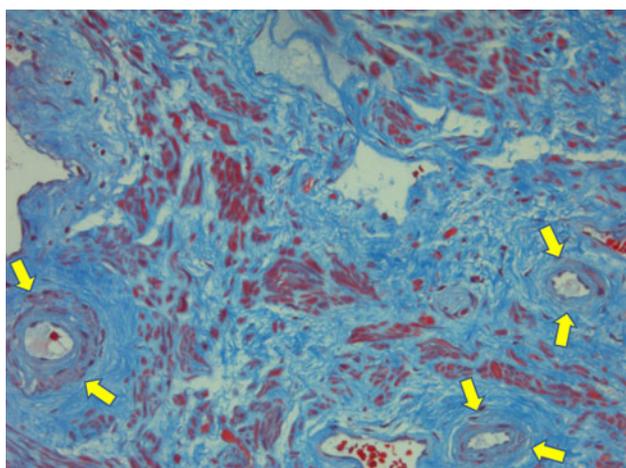


Fig. 7 Control cadaver aged 42 years. Specimen excised 0.5 cm lateral from internal ring. Patent arterial structures with mild fibrosis of the artery media (*yellow arrows*) in a context of spotty stromal fibrosis (colored *blue* among muscle bundles in *red*). Staining: Azan-Mallory, magnification $\times 20$

Discussion

The genesis of inguinal hernia has been studied by groups of scientists for many years [8–11]. To date there is no consensus in the scientific literature regarding the factors involved in hernia genesis [11]. Recent interesting theories on collagen tissue changes have provided insight into the associated molecular changes but do not address all the histological alterations [12–14]. The outcomes we highlight in this report are a part of a wider study into the histological alterations of all tissue structures close to the herniated groin in cadavers. The large amount of data collected within the frame of this study has been broken up

to into several pieces to make the work more accessible to the reader. Consequently, some of the histological findings have been discussed in previous articles [7, 15, 16].

Tissue sampling was extended up to 2.5 cm away from the hernia border in order to exclude any highly localized compressive impact exerted by the hernia content. This report deals specifically with damage to the vascular structures caused by hernia disease. The reported evidence indicates that there are pathological changes to the vascular structures in cadavers with hernia that are not seen in the controls. Both venous congestion and arterial occlusion clearly imply a diminished blood flow to the inguinal structures. Beyond the vascular alterations in these specimens we also noted the presence of plasmocytic, as well as lymphocytic, infiltrate. This is often associated with pressure on the muscular tissue resulting in fibrohyaline degeneration and fatty dystrophy. These combined results could be the consequence of a steady compression of abdominal wall structures over a long time. Long-term funneling of the viscera to the inguinal region causes a very high pressure area. This high pressure impacts several anatomical and physiologic mechanisms that normally retain an intact barrier in the inguinal region. There are direct consequences of this compression on morphological cell changes of the muscles (e.g., fatty dystrophy). There is also evidence of direct nerve compression [7]. This would eliminate efferent nerve firings, which are part of the short spinal reflex that creates a contraction of the inguinal area (tightening, shortening and contraction) during high pressure events. This is often thought of a shuttering mechanism to close off the inguinal ring—but it would also work to shorten the muscles of the inguinal area and thus create a thicker barrier. The nerve damage observed would greatly reduce this physiological mechanism. However, a secondary consequence of nerve damage is the reduced ability of the muscles to contract, which will cause an amount of atrophy—and thus weakening.

With specific reference to the impact on vascular structures, we have seen what is well recognized as chronic compressive injury to the vascular structures. This manifests as arterial sub-occlusion and venous congestion. In fact, the presence of venous congestion is a standard feature in the case of tissue damage due to chronic compression [17, 18]. The direct impact of such compression on the localized tissue would be quite large, and would manifest as reduced blood flow. The arterial sub-occlusion or occlusion following the thickening of the media could be explained by a long-acting compression applied to these areas. This would explain the concentric hyperplasia of the muscular structure of the artery. Furthermore, the obstructing medial hyperplasia of the artery is a known consequence of chronic inflammation, resulting in the withdrawal of the arteriolar capillary bed and the sclerosis of microvascular structures

[19]. Reduced blood flow would have major anatomical and physiologic impacts upon the tissue. The reduced oxygen supply would have considerable metabolic impacts. This would manifest itself as long-term morphological changes such as fatty dystrophy, reduced muscle thickness, and stress-induced cellular injury, all of which weaken the anatomical defense mechanisms in the area. These three factors—inflammation, venous congestion and arterial obstruction—could represent an effective source of structural weakening of the abdominal wall in the inguinal area involved in the hernia protrusion. The resulting effects are: decrease of blood supply as a result of artery obstruction, hindered blood outflow and congestion in the venous system.

The tissue alterations detected in autopsy specimens of cadaver with hernia, are consistently present independent of the location from which the specimens were excised. On the other hand, these pathological changes are clearly absent in control cadaver samples. We feel this is not an artifact of cadavers with hernia, as we have previously reported similar findings in living patients [15, 16]. The sum effect of all of these anatomical and physiological changes would be a weakened inguinal area that can no longer physiologically defend itself and thus a hernia would start to protrude in the area.

We conclude that these histological alterations could be the result of a steady compressive effect exerted by the abdominal viscera in the inguinal region. A weak inguinal area may be at higher risk of hernia protrusion. We do not feel the vascular changes are the sole culprit, but they could be a contributing factor to the complex multifactorial events that lead to inguinal hernia.

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