

Histology of the internal inguinal ring: it is really a novelty?

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To the Editor,

“Without any question, as long as the internal ring remains intact, hernia cannot develop.”
Albert R. Dickson, M.D., 1931.

We have read with great interest the article published by Amato et al. regarding their histopathological findings of the internal inguinal ring in patients having indirect inguinal hernia [1]. Although we consider this article not only interesting but also important to comprehend the morphological changes of inguinal hernia, we have some considerations and comments regarding this subject and, particularly, this report.

After careful analysis and discussion over the microphotographs published in the article of Amato et al., we failed to identify the so called “diffuse fibrohyaline degeneration within the muscle fibers of the internal inguinal ring” [1]. Moreover, the microphotographs included in the article did not show any muscle fibers. The images corresponded to a fascial fibrous structure corresponding to the specialized connective tissue, characteristic of the transversalis fascia, which, at the level of the internal inguinal ring, becomes thicker [2, 3]. A marked attenuation

of the transversalis fascia and significant reduction in thickness of connective tissue in the area of the internal inguinal ring has been previously described in patients with indirect inguinal hernias [2], this abnormality being caused by collagen disorders [4].

Regardless that the article of Amato et al. is an original report on the histology of the internal inguinal ring, it is no less true that, for many years, it has been known that the internal inguinal ring is an opening in the transversalis fascia [2, 3, 5]. This opening is thick at the edges of the inguinal ring, which is surrounded by fibrous fascial fibers from the conjoint tendon and muscular fibers from the internal oblique muscle [3, 5]. The lower fibers of the internal oblique muscle have their origin at the Poupart’s ligament and curve upward, inward, and downward to form the conjoint tendon. Contraction of these fibers approximates the muscle edge and Poupart’s ligament, reinforcing the internal ring against increased intra-abdominal pressure [3]. Consequently, if Amato et al. claimed to have taken biopsy samples from the “muscular tissue from the upper border of the inguinal ring” [1], they meant that the tissue samples they aimed to take corresponded to muscular fibers from the internal oblique muscle, which originate, in part, from the Poupart’s ligament covering the internal inguinal ring [3, 5]. Nonetheless, they did not show any muscular fibers in their microphotographs. Regarding the inflammatory response they claimed to have observed, a careful examination of their microphotographs failed to show an important lymphohistiocytary infiltration. Moreover, the venous congestion they described corresponded to the normal tissue response to surgical injury, which is an expected phenomenon. Regarding the nerve endings found in tissue samples, they are also expected because they regulate the sphincter-like function of the conjoint tendon acting over the internal inguinal ring.

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In conclusion, although we value the efforts of Amato et al., we cannot accept their conclusions. Their work and conclusions are not supported by appropriate histopathological evidence.

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