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Histopathological features and tissue remodeling in chronic abdominal wall mesh infection – a clinicopathological study

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Abstract

Background: Chronic mesh infection is a severe implant-associated complication characterized by persistent inflammation and progressive tissue remodeling. This condition is closely associated with foreign body reaction (FBR), fibrosis, and biofilm-related chronic inflammatory processes, which significantly alter the structural integrity of the abdominal wall. *Patients, Materials and Methods*: We performed a retrospective clinicopathological study of patients treated for chronic abdominal wall mesh infection between January 2021 and December 2024. Explanted mesh specimens together with adjacent periprosthetic tissues were collected and processed using standard histological techniques, including formalin fixation, paraffin embedding, and Hematoxylin–Eosin staining. Histopathological (HP) evaluation focused on inflammatory infiltrates, FBR, fibrotic response, and tissue remodeling. *Results*: HP examination consistently demonstrated foreign body granulomatous reaction characterized by multinucleated giant cells surrounding mesh filaments, associated with chronic inflammatory infiltrates composed predominantly of lymphocytes and plasma cells. Extensive fibrosis and dense collagen deposition with fibrotic encapsulation of the prosthetic material were observed in all cases. These findings were associated with marked structural remodeling of the surrounding soft tissues. *Conclusions*: Chronic mesh infection is associated with characteristic HP features, including foreign body granulomatous reaction, chronic inflammation, and extensive fibrosis. These morphological alterations reflect persistent implant-associated inflammatory processes and contribute to progressive tissue remodeling. HP evaluation provides essential insights into the biological mechanisms underlying chronic mesh infection and its long-term tissue effects.

Keywords: chronic mesh infection, histopathology, foreign body reaction, fibrosis, tissue remodeling.

Introduction

Abdominal wall hernias, particularly incisional hernias, represent a frequent pathological condition that reflects structural weakness and progressive remodeling of the abdominal wall connective tissues. They occur in approximately 10–25% of patients following midline laparotomy, one of the most commonly performed surgical procedures [1]. The widespread use of synthetic prosthetic meshes has significantly improved mechanical stability and reduced recurrence rates compared with primary suture repair, decreasing recurrence from 8.2% to 2.7% [2, 3]. However, implantation of synthetic material introduces a permanent foreign body into host tissues, triggering a complex cascade of biological responses, including inflammation, fibrosis, and tissue remodeling.

From a morphological perspective, prosthetic mesh implantation induces a foreign body reaction (FBR) characterized by activation of macrophages, formation of multinucleated giant cells (MGCs), and progressive deposition of fibrous connective tissue around mesh filaments. This biological response plays a critical role in mesh integration but may also contribute to pathological processes when dysregulated. Among mesh-related complications, chronic mesh infection represents one of the most severe implant-associated pathological conditions, characterized by persistent inflammatory activity, tissue destruction, and progressive structural alterations of the abdominal wall [4, 5]. Chronic infection is associated with prolonged inflammatory stimulation, excessive fibrosis, impaired tissue vascularization, and formation of fibrotic encapsulation, all of which significantly alter tissue architecture and function.

Histopathologically, chronic mesh infection is characterized by a combination of foreign body granulomatous reaction, chronic inflammatory infiltrates, and extensive extracellular matrix (ECM) remodeling. Persistent inflammatory stimulation promotes fibroblast activation and excessive collagen deposition, leading to the formation of dense fibrotic tissue that encapsulates prosthetic material. These morphological changes compromise tissue elasticity, alter vascular supply, and contribute to progressive tissue dysfunction. In addition, biofilm formation on the

prosthetic surface plays a crucial role in maintaining chronic inflammation by protecting microorganisms from immune clearance and antimicrobial therapy, thereby sustaining a persistent inflammatory microenvironment [4].

Chronic inflammatory and fibrotic changes associated with prosthetic mesh implantation may also produce complex clinicopathological manifestations. Extensive fibrosis, chronic inflammatory infiltrates, and tissue remodeling may mimic other pathological conditions, including malignant infiltration of the abdominal wall, emphasizing the importance of histopathological (HP) examination in establishing an accurate diagnosis and understanding the underlying biological processes [6]. The morphological evaluation of explanted mesh specimens provides essential insights into the mechanisms of FBR, chronic inflammation, and tissue remodeling, contributing to a better understanding of implant–tissue interactions.

Clinicopathological correlation plays a fundamental role in elucidating the structural and biological consequences of chronic mesh infection. HP analysis allows characterization of the cellular inflammatory response, evaluation of fibrotic remodeling, and identification of tissue alterations induced by persistent implant-associated inflammation. These morphological changes reflect the dynamic interaction between prosthetic material and host tissues and represent key pathological features underlying the progression of chronic implant-associated inflammatory processes [7, 8].

Aim

The aim of this study was to investigate the HP characteristics and patterns of tissue remodeling in chronic abdominal wall mesh infection, focusing on FBR, inflammatory response, fibrosis, and structural alterations at the mesh–tissue interface, and to assess their clinicopathological significance.

Patients, Materials and Methods

Study design and patient selection

This retrospective clinicopathological study included all consecutive patients diagnosed with chronic abdominal wall mesh infection who underwent mesh explantation between April 2023 and December 2025 at Dr. Constantin Andreoiu Emergency County Hospital, Ploieşti, Romania. Chronic mesh infection was defined as a persistent implant-associated inflammatory process occurring months or years after prosthetic mesh implantation, characterized by chronic suppuration, sinus tract formation, abscess, or fistula, consistent with long-standing implant-associated pathology.

Inclusion criteria were: (*i*) history of ventral or incisional hernia repair with synthetic mesh implantation; (*ii*) clinical and/or imaging findings consistent with chronic mesh-associated inflammatory pathology; and (*iii*) availability of explanted mesh material and adjacent periprosthetic tissue suitable for HP examination.

Patients with early postoperative infections lacking features of chronic inflammatory remodeling were excluded.

Tissue collection and processing

Explanted mesh specimens together with adjacent periprosthetic soft tissues were collected and submitted for HP analysis. The collected specimens consisted of fragments of synthetic mesh associated with surrounding fibrous tissue, granulation tissue, and inflammatory infiltrates.

Immediately after removal, tissue specimens were fixed in 10% neutral buffered formalin for adequate preservation of tissue architecture. Following fixation, specimens were processed using standard histological protocols, including dehydration, paraffin embedding, and sectioning at 3–5 μm thickness. Tissue sections were subsequently stained with Hematoxylin–Eosin (HE) for routine microscopic evaluation.

Histopathological evaluation

HP evaluation was performed using light microscopy to assess the morphological features associated with chronic implant-related tissue response.

Microscopic examination focused on the following parameters: (*i*) FBR, defined by the presence of macrophages and multinucleated foreign body giant cells surrounding prosthetic mesh filaments; (*ii*) chronic inflammatory infiltrate, consisting predominantly of lymphocytes, plasma cells, and macrophages within periprosthetic connective tissue; (*iii*) acute inflammatory changes, including neutrophilic infiltrates and microabscess formation; (*iv*) fibrotic response, characterized by increased collagen deposition and fibrotic encapsulation of prosthetic material; (*v*) tissue remodeling, including structural alterations of connective tissue architecture and reactive epithelial changes; and (*vi*) vascular alterations, including reduced vascular density in chronically inflamed and fibrotic areas.

All specimens were independently evaluated by two experienced pathologists.

Ethical approval

The study was conducted in accordance with the Declaration of Helsinki and was approved by the Ethics Committee of Dr. Constantin Andreoiu Emergency County Hospital, Ploieşti (Approval No. 13741/24.03.2023). All data were analyzed anonymously.

Results

HP analysis was performed in all cases. A total of 10 patients were included, with a mean age of 67.5 years (range 58–78 years) (Table 1). The cohort was predominantly male (80%) and presented a high mean body mass index (45.1 kg/m^2, range 36–60 kg/m^2), with morbid obesity observed in 80% of cases. Relevant comorbidities included hypertension (50%), atrial fibrillation (40%), diabetes mellitus (10%), and ankylosing spondylitis (20%).

Table 1 – ***Demographic and clinical characteristics of the patients***

Parameter	Value
n	10
Age, mean (range) [years]	67.5 (58–78)
Sex (Male/Female)	8/2
BMI, mean (range) [kg/m^2]	45.1 (36–60)
Morbid obesity (BMI ≥40 kg/m^2), *n* (%)	8 (80%)
Diabetes mellitus, *n* (%)	1 (10%)
Hypertension, *n* (%)	5 (50%)
Atrial fibrillation, *n* (%)	4 (40%)
Ankylosing spondylitis, *n* (%)	2 (20%)

BMI: Body mass index; *n*: No. of cases.

All patients had a history of incisional ventral hernia repair with polypropylene mesh implantation. The onlay technique was used in 70% of cases, while sublay placement was used in 30% (Table 2).

Table 2 – ***Characteristics of the initial hernia repair***

Parameter	*n* (%)
Incisional ventral hernia	10 (100%)
Open surgical approach	10 (100%)
Onlay mesh placement	7 (70%)
Sublay mesh placement	3 (30%)
Polypropylene mesh	10 (100%)

n: No. of cases.

The clinical course was dominated by severe mesh-related septic complications (Table 3). Chronic suppuration and persistent sinus tracts were present in all patients. Periprosthetic abscess formation occurred in more than half of the cohort (60%), while enterocutaneous fistulas developed in 40% of cases. Structural mesh failure, including migration or rupture, was documented in 20% of patients.

Table 3 – ***Mesh-related complications***

Complication	*n* (%)
Chronic mesh suppuration	10 (100%)
Cutaneous sinus tract	10 (100%)
Enterocutaneous fistula	4 (40%)
Periprosthetic abscess	6 (60%)
Mesh migration/rupture	2 (20%)

n: No. of cases.

Histopathological findings

HP examination of explanted mesh specimens and adjacent periprosthetic tissues revealed consistent morphological features of chronic implant-associated inflammatory response. A pronounced foreign body granulomatous reaction was observed in all cases, characterized by multinucleated foreign body giant cells surrounding refractile prosthetic mesh filaments, associated with macrophages and chronic inflammatory infiltrates composed predominantly of lymphocytes and plasma cells (Figure 1).

Low-power microscopic examination demonstrated significant tissue remodeling, including dermal fibrosis, chronic inflammatory infiltrates, and reactive epithelial changes. Vascular alterations, including reduced vascular density, were also observed in areas of advanced fibrosis (Figure 2).

Extensive fibrotic remodeling was also identified, with dense collagen deposition and formation of fibrotic connective tissue encapsulating prosthetic material. These changes resulted in marked structural alteration of the surrounding soft tissues. At the mesh–tissue interface, fibrovascular granulation tissue and chronic inflammatory infiltrates were observed surrounding empty spaces corresponding to removed prosthetic filaments, further illustrating active periprosthetic remodeling (Figure 3).

In deeper tissue planes, skeletal muscle bundles were separated by fibrotic septa and chronic inflammatory changes, reflecting advanced architectural disruption and confirming the extent of structural remodeling induced by persistent implant-associated inflammation (Figure 4).

The foreign body granulomatous reaction and fibrotic encapsulation were consistently observed in all examined specimens (10/10; 100%), confirming the reproducibility of these morphological features.

Discussions

Chronic mesh infection represents a complex implant-associated pathological condition characterized by persistent inflammation and progressive tissue remodeling at the prosthesis–host tissue interface. Unlike superficial surgical site infections, chronic mesh infection is driven by biofilm-associated microbial colonization, which promotes sustained inflammatory stimulation and alters the normal biological response to implanted material [4, 9, 10]. The presence of bacterial biofilm on prosthetic surfaces contributes to persistent immune activation, impaired bacterial clearance, and maintenance of a chronic inflammatory microenvironment, explaining the prolonged and refractory nature of implant-associated infections [10–13].

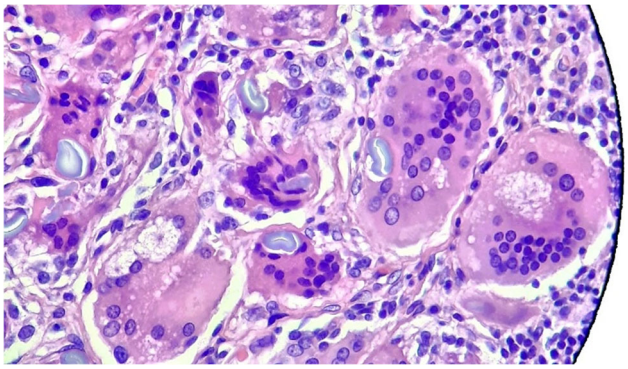

Figure 1 – ***Histological cross-section demonstrating a pronounced foreign body granulomatous response to prosthetic mesh material. Multinucleated giant cells encircle refractile foreign material, associated with chronic inflammatory infiltrate and extensive fibrotic stromal reaction. These findings are characteristic of chronic mesh-induced inflammation and support the presence of a persistent foreign body reaction. Hematoxylin–Eosin (HE) staining, ×400.***

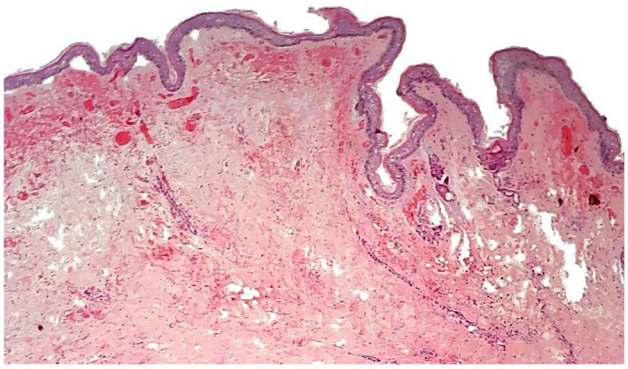

Figure 2 – ***Low-power microscopic view revealing chronic inflammatory remodeling of the abdominal wall. The epidermis displays reactive epithelial changes, while the dermis is expanded by dense fibrous tissue, chronic inflammatory infiltrate, and vascular alterations. These features are characteristic of prolonged inflammatory response in the context of chronic mesh-related infection. HE staining, ×100.***

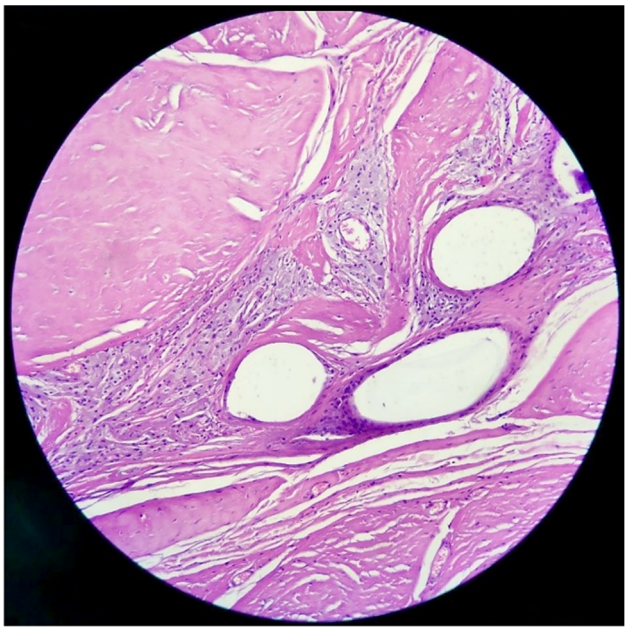

Figure 3 – ***Periprosthetic chronic inflammatory response with granulation tissue at the mesh–tissue interface. Multiple empty spaces correspond to removed mesh filaments, surrounded by fibrovascular granulation tissue and chronic inflammatory infiltrate. HE staining, ×200.***

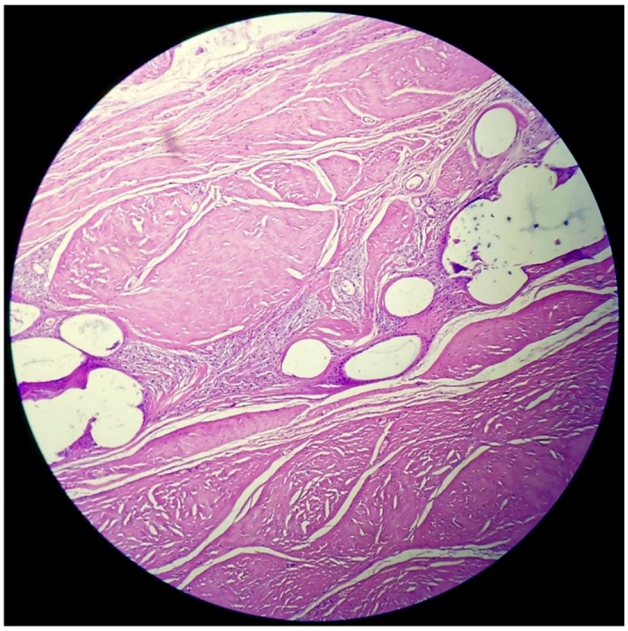

Figure 4 – ***Abdominal wall soft tissue remodeling adjacent to the infected mesh, showing skeletal muscle bundles separated by fibrotic septa and periprosthetic chronic inflammatory changes; empty spaces indicate mesh filament footprints. HE staining, ×100.***

From an HP perspective, the defining feature of chronic mesh infection is the FBR. This process is mediated by macrophage activation and the formation of multinucleated foreign body giant cells surrounding prosthetic material, as observed consistently in our specimens. These cellular responses represent a hallmark of host reaction to non-degradable synthetic material and reflect an attempt to isolate and contain the foreign body [14]. Previous experimental and clinical studies have demonstrated that polypropylene mesh implantation induces a sustained granulomatous inflammatory response, characterized by macrophage recruitment, MGCs formation, and progressive ECM deposition [15–23].

In addition to foreign body granulomatous reaction, chronic inflammatory infiltrates predominantly composed of lymphocytes and plasma cells were observed in all cases. This pattern reflects persistent antigenic stimulation and ongoing immune response, which plays a central role in maintaining chronic inflammation. Similar HP findings have been reported in previous studies evaluating explanted mesh specimens, confirming that chronic inflammatory infiltrates represent a key morphological component of implant-associated pathological processes [20–23]. Persistent inflammatory activity promotes fibroblast activation and ECM production, leading to progressive fibrotic remodeling.

Fibrosis represents a critical morphological consequence of chronic implant-associated inflammation. In our study, extensive collagen deposition and dense fibrotic encapsulation of prosthetic material were consistently observed. This fibrotic response results in the formation of rigid connective tissue structures that encapsulate mesh filaments and alter normal tissue architecture. Experimental studies have demonstrated that mesh implantation induces progressive collagen deposition and connective tissue remodeling, reflecting a dynamic interaction between prosthetic material and host tissues [18, 21]. Excessive fibrosis contributes to structural alterations of the abdominal wall and represents a characteristic feature of chronic implant-associated tissue response.

Chronic inflammation and fibrosis are closely associated with vascular alterations. Reduced vascular density and morphological features suggestive of chronic ischemic remodeling were observed in fibrotic regions, reflecting impaired tissue perfusion. Previous studies have shown that fibrotic encapsulation of prosthetic material may compromise vascular supply and impair immune surveillance, thereby perpetuating chronic inflammatory processes and favoring persistence of implant-associated infection [24–26]. These vascular alterations further contribute to tissue remodeling and structural dysfunction.

HP changes observed in our study, including foreign body granulomatous reaction, chronic inflammatory infiltrates, and extensive fibrosis, reflect the biological mechanisms underlying chronic mesh infection. Persistent inflammatory stimulation promotes progressive connective tissue remodeling, resulting in structural alterations of the abdominal wall. HP examination also plays an important diagnostic role by differentiating chronic implant-associated inflammatory processes from other pathological conditions, including neoplastic infiltration or nonspecific chronic inflammatory lesions. The identification of foreign body granulomatous reaction and characteristic fibrotic remodeling represents a key diagnostic feature of implant-associated pathology. These morphological changes represent the pathological substrate responsible for clinical manifestations such as chronic suppuration, sinus tract formation, and fistulization, which have been reported in association with advanced implant-related pathological processes [27–31].

Fibrotic remodeling and chronic inflammation may also affect tissue biomechanics and functional integrity. Excessive collagen deposition and structural reorganization alter the mechanical properties of connective tissue and impair

normal abdominal wall function. Previous studies have demonstrated that chronic implant-associated inflammation and fibrosis may contribute to persistent tissue dysfunction and structural instability [32–40]. These findings support the concept that HP alterations play a fundamental role in determining the structural and functional consequences of chronic mesh infection.

The morphological findings observed in our study are consistent with previous HP investigations demonstrating that synthetic mesh implantation induces a complex and prolonged biological response involving inflammatory activation, FBR, and fibrotic remodeling [18, 20–23]. The persistence of MGCs, chronic inflammatory infiltrates, and dense fibrosis confirms the presence of ongoing implant-associated inflammatory processes and reflects the inability of host tissues to fully integrate or eliminate synthetic material.

These observations highlight the importance of HP evaluation in understanding the biological mechanisms underlying chronic mesh infection [41]. Microscopic examination provides essential information regarding the nature and extent of inflammatory and fibrotic changes and allows characterization of implant–tissue interactions. HP analysis contributes to the identification of structural alterations associated with chronic inflammation and provides insight into the pathological processes responsible for progressive tissue remodeling [42–45].

Limitations

The present study is limited by its retrospective design and relatively small number of cases. However, the consistency of HP findings across all examined specimens supports the reproducibility of these morphological patterns and confirms the characteristic features of chronic implant-associated inflammatory response.

Overall, chronic mesh infection is characterized by a distinct HP profile consisting of foreign body granulomatous reaction, persistent chronic inflammatory infiltrates, and extensive fibrotic tissue remodeling. These morphological alterations reflect the complex biological interaction between synthetic prosthetic material and host tissues and represent the structural basis of chronic implant-associated pathological processes.

Conclusions

Chronic abdominal wall mesh infection is characterized by a distinct HP pattern including foreign body granulomatous reaction, persistent chronic inflammatory infiltrates, and extensive fibrotic encapsulation at the mesh–tissue interface. These findings reflect a sustained implant-associated inflammatory process and progressive connective tissue remodeling.

This study highlights the consistent coexistence of FBR, chronic inflammation, and fibrosis in explanted mesh specimens, confirming their central role in the pathogenesis of chronic implant-associated pathology. HP evaluation provides essential insight into implant–tissue interactions and the morphological mechanisms underlying chronic mesh infection.

Conflict of interests

The authors declare that they have no conflict of interests.

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