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LAPAROSCOPIC MESH EXPLANTATION FOR DELAYED INTRAPERITONEAL ONLAY MESH (IPOM) INFECTION: A CASE REPORT

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ABSTRACT

Background: Intraperitoneal onlay mesh (IPOM) repair is a widely used minimal access technique for the repair of primary and incisional ventral hernias, valued for its low recurrence rates and short hospital stay.[1] Mesh infection is an uncommon but serious complication of IPOM and can present acutely, within weeks of surgery, or as a delayed, chronic infection presenting months to years later. Here we present a case of a 30-year-old female who had delayed mesh infection four years after IPOM surgery done for umbilical hernia, presenting with pain and swelling for a short duration of one week. After investigations, she underwent laparoscopic IPOM mesh explantation.

Conclusion: Delayed mesh infection, although rare, should be considered in any patient presenting with peri-umbilical swelling even years after IPOM repair. Unlike acute mesh infection, in which the mesh typically lies loose within a purulent collection and is retrieved with relative ease, chronic infection is characterised by dense incorporation of the mesh into the abdominal wall, making laparoscopic explantation technically demanding, though still feasible and safe in experienced hands.

KEYWORDS: Intraperitoneal onlay mesh; IPOM; mesh infection; mesh explantation; laparoscopic hernia repair; delayed infection; biofilm

INTRODUCTION

Intraperitoneal onlay mesh (IPOM) repair, in which a composite mesh is placed within the peritoneal cavity and fixed against the underside of the abdominal wall, is among the most frequently performed laparoscopic techniques for primary and incisional ventral hernias. Its popularity stems from reduced postoperative pain, shorter hospital stay and low recurrence when compared with open repair.[1]

Mesh infection remains one of the most feared complications of hernia mesh surgery. Although the overall incidence is low, it carries substantial morbidity, often necessitating prolonged antibiotic therapy, repeated imaging and, frequently, surgical removal of the mesh.[5] Mesh infection is conventionally divided into acute and chronic (or early- and late-onset) categories, most series using a cut-off of approximately 90 days from the index operation, although some authors use six months to a year.[2,7] This distinction is not merely temporal; it reflects two different underlying mechanisms. Acute infection typically results from intraoperative or perioperative wound contamination, superimposed infection of a haematoma or seroma, or a breach in aseptic technique, and manifests while the mesh is still poorly incorporated into the surrounding tissue. Chronic or delayed infection, by contrast, is increasingly attributed to low-grade bacterial biofilm formation on the mesh surface, most often by coagulase-negative staphylococci or *Staphylococcus aureus*, which can remain clinically silent for years before an inciting event — such as a persistent seroma, minor trauma, or haematogenous seeding from a remote focus — triggers overt suppuration.[3,4]

We report the case of a young woman who developed a mesh infection four years after laparoscopic IPOM repair of an umbilical hernia, initially masquerading as a simple seroma, and discuss the mechanisms, distinguishing features and preventive strategies relevant to delayed mesh infection as against its acute counterpart.

Case Presentation

History and Examination

A 30-year-old woman presented with peri-umbilical pain of one week's duration and swelling for five days. The pain was insidious in onset, gradually progressive and pricking in character; the swelling was likewise gradually progressive and was associated with a change in the overlying skin colour. There was no history of trauma, dental caries, chronic respiratory infection, pelvic inflammatory disease, otitis media or vaginal discharge. She had no history of diabetes mellitus or hypertension and reported decreased appetite for the preceding week, with normal bowel and bladder habits.

Four years earlier, she had undergone laparoscopic umbilical hernia repair with IPOM at another hospital in

Chennai, performed ten months after her second delivery (she is P2L1D1, both normal vaginal deliveries, with puerperal sterilisation done at the time of her second delivery). She remained entirely asymptomatic for the following four years before the onset of the present complaints. She works as a lawyer.
On examination, her body mass index was 25.5 kg/m² and vital signs were stable. Inspection revealed a diffuse, 5 × 4 cm peri-umbilical swelling with overlying erythema; there were no dilated veins, ulceration, discharge or cough impulse. On palpation, the swelling was mildly warm and tender, without a cough impulse or any other swelling. The head-raising test accentuated the swelling, confirming its parietal (abdominal wall) location rather than an intra-abdominal or hernial origin. A provisional diagnosis of an inflammatory parietal wall swelling was made.

Investigations and Initial Management
Ultrasonography of the abdomen demonstrated a 50 mL fluid collection in the retro-umbilical region, and contrast-enhanced computed tomography confirmed a 50–60 mL collection beneath the mesh in the peri-umbilical plane. A diagnosis of seroma was made, and the patient was managed conservatively for one week with antibiotics and analgesics.
As the collection persisted despite conservative management, she underwent ultrasound-guided aspiration of the collection under sterile conditions in the operating theatre. Pale-coloured fluid was aspirated and sent for culture and sensitivity, which showed no growth of organisms. Following aspiration, the swelling resolved completely and the patient was relieved of her symptoms.
The swelling reappeared after ten days. A repeat ultrasound scan at this stage showed an increase in the collection to approximately 150 mL, and a revised diagnosis of delayed mesh infection was made. The patient was accordingly planned for laparoscopic exploration and mesh explantation.

Operative Findings
Under general anaesthesia, diagnostic laparoscopy revealed omental and bowel adhesions to the anterior abdominal wall from the previous surgery, which were released by adhesiolysis (Figure 1). A 7 cm swelling was noted in the parietal wall. On incising the swelling, turbid fluid was aspirated and thoroughly suctioned. The IPOM mesh was found firmly adherent to the parietal wall within a flap of tissue, rather than lying loose within the collection (Figure 2). Owing to dense adhesions, the mesh could be removed only in strips and with considerable difficulty (Figure 3); the excised mesh fragments are shown in Figure 4. The area was irrigated thoroughly with normal saline, and a drain was placed before closure. Both the explanted mesh and the intraoperatively aspirated fluid were sent for culture and sensitivity, which, similar to the earlier aspirate, showed no growth of organisms.

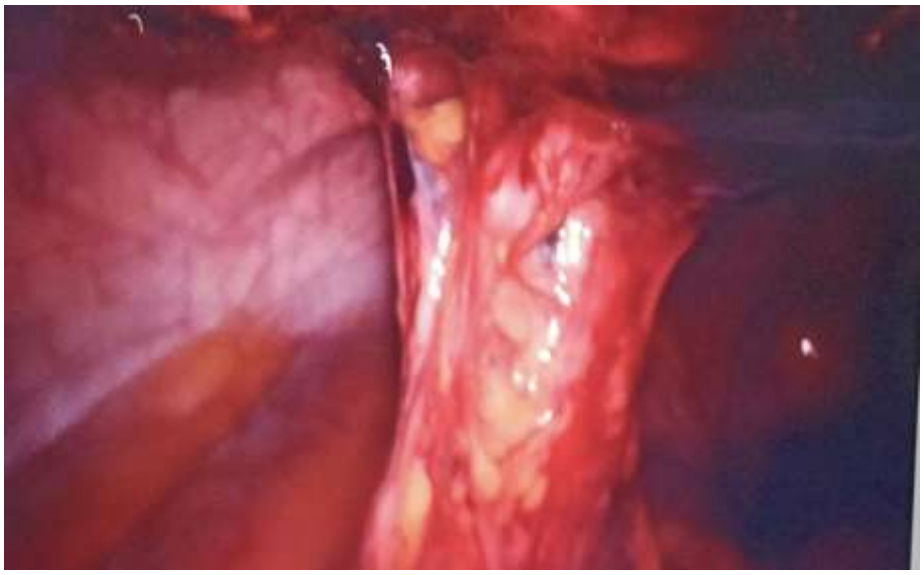
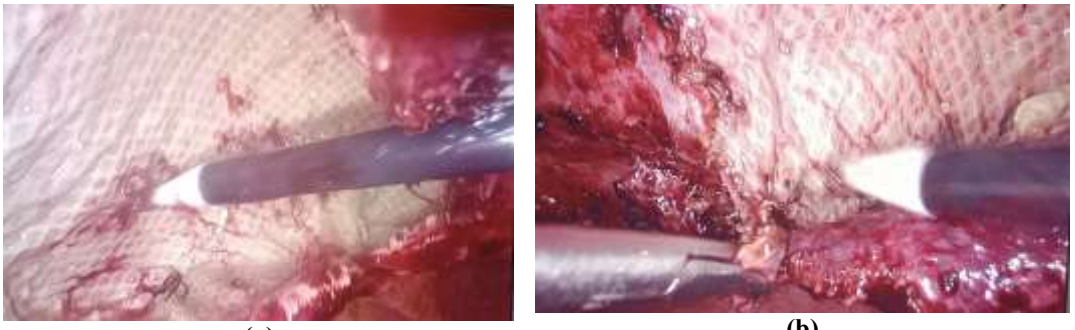


Figure 1. Laparoscopic adhesiolysis showing dense omental adhesions to the anterior abdominal wall from the previous surgery.



(a) (b)

Figure 2. Intraoperative view after evacuation of the infected collection, showing the IPOM mesh densely incorporated into the parietal wall (a, b).

(a) (b) (c)

Figure 3. The densely adherent mesh being grasped and removed in strips owing to dense fibrous adhesions (a–c).

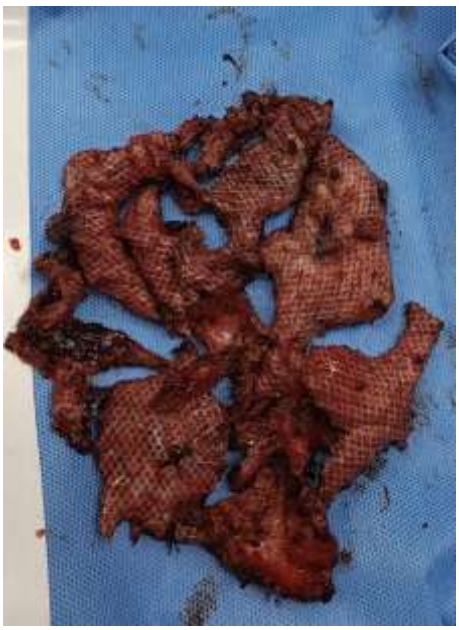


Figure 4. Gross photograph of the explanted mesh, retrieved in multiple fragments owing to dense fibrous incorporation into the abdominal wall.

Postoperative Course and Follow-up

The postoperative period was uneventful, and the patient was discharged on the second postoperative day. She returned to work ten days after surgery. She has since been followed up for one year, remaining entirely asymptomatic, without pain, swelling, recurrent hernia or recurrence of infection.

DISCUSSION

IPOM is now firmly established as a standard approach to laparoscopic ventral and umbilical hernia repair, combining the advantages of minimal access surgery with acceptably low rates of recurrence and wound complication.[1] Mesh infection, while uncommon after laparoscopic repair, remains one of its most serious complications and is a leading reason for reoperation and mesh removal.[5]

Acute Versus Chronic Mesh Infection

Most contemporary series define acute mesh infection as one occurring within 90 days of the index operation, and chronic (or late-onset) infection as one occurring beyond this point; some authors instead use a six-month or one-

year threshold.[2,7] This temporal division corresponds to a genuine difference in pathogenesis. Acute infection generally arises from direct bacterial contamination at the time of surgery, an infected haematoma or seroma, or a wound-healing complication that tracks down to the mesh, and tends to present with the classical features of an abscess — fever, discharge and a rapidly evolving collection — within the early postoperative weeks.[5,7] At this early stage, the mesh has not yet been incorporated by fibrous tissue and typically lies loose or 'floating' within the purulent collection, so that it can usually be retrieved intact and with relative ease.[7]

Chronic or delayed mesh infection, as seen in the present case, is now understood to be closely linked to bacterial biofilm formation on the mesh surface. Low-virulence organisms such as coagulase-negative staphylococci or *S. aureus* can adhere to the mesh at the time of implantation or shortly thereafter, encase themselves in an extracellular polymeric matrix, and persist in a dormant, clinically silent state for months to years.[4] In one study of explanted hernia meshes removed for chronic pain, recurrence or mesh shrinkage without overt infection, staphylococcal biofilms were demonstrated on the majority of specimens, suggesting that subclinical colonisation may be considerably more common than clinically apparent infection.[4] A subsequent stimulus — most often a persistent or enlarging seroma, as in our patient, but also minor local trauma or haematogenous seeding from a distant focus such as dental, urinary or skin infection — can convert this dormant colonisation into overt suppuration years after the index operation.[3,6] Our patient had no identifiable dental, urinary, respiratory or gynaecological focus of infection, making mesh-surface biofilm activation the most plausible explanation for her delayed presentation.

Why Chronic Infection is Harder to Explant

By the time chronic mesh infection becomes clinically evident, the mesh has usually been present for years and has undergone extensive fibrous incorporation into the abdominal wall, with dense adhesions to adjacent omentum and, occasionally, bowel. Series comparing early- and late-onset infection have specifically noted that late-presenting mesh is frequently found crumpled and toughly adherent to surrounding scar tissue, in contrast to the loosely floating mesh typical of acute infection.[7] This was precisely our operative finding: the mesh was firmly fixed within a parietal flap and could only be removed in strips after laparoscopic adhesiolysis, reflecting the greater technical difficulty of chronic mesh explantation compared with the acute setting.

Prevention

Because acute and chronic infection arise through different mechanisms, their prevention strategies differ in emphasis, though several measures reduce the risk of both. Perioperative measures include strict aseptic technique, judicious use of prophylactic antibiotics, minimisation of intraoperative mesh handling and contamination, control of modifiable patient factors such as obesity, smoking and glycaemic status, and meticulous haemostasis to reduce the risk of haematoma or seroma formation, which can act as a nidus for later colonisation.[5,8] Mesh selection also plays a role: macroporous, monofilament meshes permit better tissue in-growth, immune cell access and vascularisation than microporous or multifilament alternatives, and are therefore less prone to sustaining biofilm.[5]

Specific to the prevention of delayed infection, prompt recognition and drainage of a persistent or enlarging postoperative seroma — rather than prolonged conservative observation — may reduce the risk of secondary bacterial colonisation of an initially sterile collection.[8] Adequate mesh fixation to prevent folding, migration or erosion, and patient education about reporting new swelling or discharge even years after an apparently uneventful repair, are additional practical measures. In our patient, the initial collection was appropriately treated as a seroma; the persistence of the collection despite conservative management, its transient resolution after sterile aspiration, and its recurrence within ten days in a patient who was otherwise four years out from an uncomplicated repair, were the key clinical clues that ultimately prompted surgical exploration.

Management Principles

Once chronic mesh infection is confirmed or strongly suspected, particularly when conservative management fails to control an enlarging collection, complete mesh removal remains the definitive treatment, since biofilm-laden mesh does not respond reliably to antibiotics alone.[4,5] A laparoscopic approach to exploration and explantation, as employed in this case, allows thorough adhesiolysis, direct visualisation of the extent of mesh involvement and any bowel adherence, and complete evacuation of the collection, while preserving the benefits of minimal access surgery even in a reoperative field.[3] Thorough irrigation and drain placement after mesh removal, as performed here, help to reduce residual bacterial load and postoperative fluid collection. Our patient's uncomplicated recovery, early discharge and sustained one-year symptom-free follow-up support the feasibility of this approach even when dense adhesions are encountered.

Notably, both the initially aspirated fluid and, subsequently, the explanted mesh and intraoperative fluid in our patient were sterile on standard culture, despite unequivocal clinical, radiological and operative evidence of infection. This culture-negative pattern is well recognised in chronic prosthetic infections: biofilm-encased organisms are shielded within a protective extracellular matrix and are frequently missed by conventional culture techniques, which are optimised for detecting free-floating (planktonic) bacteria rather than sessile biofilm communities.[9,10] A similar disconnect between clinical infection and culture positivity has been reported in delayed-onset mesh complications at other anatomical sites, reinforcing biofilm-related pathogenesis, rather than acute exogenous contamination, as the more likely explanation in our patient.[9] As with any single case report,

these inferences cannot be generalised, and larger series using molecular or culture-independent diagnostic methods are needed to confirm the role of biofilm in delayed IPOM infection.

CONCLUSION

Delayed mesh infection after IPOM repair is uncommon but should remain a differential diagnosis in any patient presenting with a peri-umbilical or parietal wall collection, even years after an apparently uneventful hernia repair, particularly when a presumed seroma fails to resolve or recurs after aspiration. Unlike acute mesh infection, where the mesh typically floats freely within a purulent collection and is easily retrieved, chronic infection is marked by dense fibrous incorporation of the mesh into the abdominal wall, making explantation technically more demanding. Nonetheless, as illustrated by this case, laparoscopic exploration and mesh explantation is a safe and effective approach even in the presence of dense adhesions, and awareness of the distinct pathophysiology of delayed mesh infection can help guide earlier recognition and more timely intervention.

DECLARATIONS

Consent: Written informed consent was obtained from the patient for publication of this case report.

Conflict of Interest: The authors declare no conflict of interest.

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