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37th Edition

Dr. Sherbaz Bichu

CEO & Specialist Anaesthetist
Aster Hospitals & Clinics,
UAE, Oman, and Bahrain



On behalf of Aster's leadership, I am delighted to welcome you to the 37th edition of HealthNews and share an important development. Aster DM Healthcare has announced the expansion of Aster Hospital, Al Qusais, with more operational beds. This initiative reaffirms our commitment to strengthening our clinical capabilities, expanding capacity, and improving access to comprehensive healthcare.

This month also included World Cancer Day, serving as a reminder to advance cancer prevention, early detection, and treatment. I commend our oncology teams for their unwavering dedication to delivering integrated, patient-centric care to improve clinical excellence and treatment outcomes.

As we enter the holy month of Ramadan, I extend my sincere appreciation to all our doctors and healthcare professionals who continue to serve with empathy and resilience. Your commitment during this sacred time exemplifies the values that define our care, kindness, and respect for the communities we serve.

Dr. Ramanathan V

Medical Director
Aster Hospitals & Clinics,
UAE



As the Group Medical Director of Aster Hospitals and Clinics, I am pleased to address you in the 37th edition of HealthNews, as we continue to celebrate the exceptional clinical and surgical excellence demonstrated by our doctors across Aster Hospitals.

This edition highlights several exclusive and first-time successful surgical procedures performed at Aster, achievements that not only reflect advanced surgical capability but also the strength of our multidisciplinary collaboration, decision-making, and unwavering focus on patient safety. These procedures represent significant milestones toward expanding specialised services and strengthening our capability to treat complex and high-risk cases through quality, compassion, and excellence.

Each case featured in this newsletter stands as a testament to your precision, resilience, and commitment to pushing clinical boundaries through innovation and expertise. Thank you for your continued contributions that elevate Aster's standards of care and clinical reputation.



Dr. Hariharan Chelladurai Pandian
Neurosurgery (Specialist)



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Novel Approach: Intervertebral Disc Disease treated effectively with Laser Discectomy at Aster Hospital, Al Qusais, Dubai

PRESENTATION

- 32-year-old male
- Medical history of a failed epidural steroid injection
- No significant family history
- Admitted with complaints of:
 - Persistent chronic lower back pain and radicular symptoms extending into the lower extremities
 - Numbness and tingling sensations (paresthesia) in the legs
 - A feeling of heaviness

FINDINGS

During examination:

- **Provocative Testing:** Positive Straight Leg Raise (SLR) test
- **Neurological Status:** Muscle weakness in the lower limbs and diminished deep tendon reflexes
- **Sensory Evaluation:** Confirmed sensory loss and tingling in specific dermatomal distributions
- **Palpation:** Localised tenderness over the affected spinal segments

Radiographic Assessment (MRI Lumbar Spine) showed:

- Progressive narrowing of the spinal canal and thecal sac indentation across multiple levels

Spinal Level	Findings	AP Diameter (mm)
L1-L2	Normal range	14.3
L2-L3	Normal range	12.8
L3-L4	Annular bulge; thecal sac indentation	12.1
L4-L5	Left posterolateral protrusion; canal narrowing	9.3
L5-S1	Diffuse bulge; bilateral recess narrowing	8.5

SURGICAL PROCEDURE

The patient underwent **L4L5 and L5S1 Laser Discectomy**:

- **Preparation:** The procedure was performed under local anaesthesia with the patient in a prone position.
- **Access:** Under fluoroscopic guidance, a fine-gauge needle was inserted into the nucleus pulposus, utilising **Kambin's Triangle** as the anatomical window.
- **Ablation:** A fibre-optic laser probe was threaded through the needle to emit controlled energy pulses.
- **Mechanism of Action:** The laser vaporised a small volume of the nucleus pulposus (ablation), creating a vacuum effect that allowed the protruding disc material to recede from the nerve root. This reduced internal disc pressure and deactivated intradiscal pain receptors.



**Laser needle placement in the disc space,
confirmed by the radiopaque dye**

POST PROCEDURE

The patient tolerated the procedure well and was discharged in stable condition.

Notable recovery milestones included:

- **Return to Work:** Achieved within 2 days due to the absence of muscle stripping or laminectomy.
- **Peak Improvement:** Expected within 10–14 days as local inflammation subsides.
- **Restrictions:** Heavy lifting and strenuous activity were restricted for 4–6 weeks to facilitate annular healing.

DISCUSSION

This report details the successful application of Percutaneous Laser Disc Decompression (PLDD), also known as Laser Discectomy, for chronic lower back pain and radiculopathy. Despite prior interventional attempts, including an epidural steroid injection, the patient remained symptomatic. Following minimally invasive laser ablation of the L4–L5 and L5–S1 discs, the patient demonstrated rapid functional recovery and returned to work within 48 hours. Performed for the first time in Aster with a successful outcome.

Clinical Advantages of PLDD:

- **Minimal Morbidity:** Reduced blood loss and use of local anaesthesia.
- **Zero Fibrosis:** Unlike open surgery, PLDD avoids the formation of epidural scar tissue (fibrosis), a leading cause of failed back surgery syndrome.

-
- **Structural Integrity:** Preserves spinal architecture and maintains stability.

Potential Risks:

While rare, risks include discitis (infection), recurrence of the bulge, or a minor risk of nerve injury.

CONCLUSION

Laser discectomy is an effective, tissue-sparing intervention for patients with small, contained disc prolapses that are refractory to conservative management but do not yet necessitate invasive microdiscectomy. It provides high patient satisfaction through rapid recovery and the preservation of spinal stability.

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Understanding Ear Infections: From Pathophysiology to Clinical Care



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INTRODUCTION

Infectious conditions affecting the ear continue to represent a major driver of healthcare consultation globally, contributing substantially to outpatient consultations, antibiotic use, and referral to specialist services (1,2). Epidemiological analyses consistently demonstrate that inflammatory ear disease contributes significantly to morbidity across age groups, with a particularly high burden observed in early childhood (3). Specifically, acute otitis media has a global incidence rate of 10.85%, corresponding to approximately 709 million cases each year, with 51% of episodes occurring in children under five years of age (1).

Despite early-life predominance, this disease burden is not limited to paediatric populations, as recurrent and chronic ear infections persist into adolescence and adulthood, contributing to long-term sequelae such as hearing impairment and chronic middle-ear pathology (1). Clinically, ear infections encompass a wide spectrum, ranging from mild, self-limiting inflammatory episodes to recurrent and chronic disease characterised by persistent effusion, biofilm-associated infection, and progressive tissue involvement (4,5).

Importantly, progression beyond the middle ear can result in intratemporal and intracranial complications, which, although less common, are associated with significant morbidity and potential mortality (6). In this context, accurate diagnosis and precise localisation of infection are critical, as appropriate management and optimal clinical outcomes depend on correct identification of disease type, anatomical site, and severity at presentation (4,7).

This article focuses on the epidemiology and clinical burden of ear infections, examines their underlying pathophysiology, outlines principles of diagnostic evaluation and disease stratification, and discusses therapeutic decision-making aimed at optimising outcomes and reducing long-term morbidity.

TYPES OF EAR INFECTIONS AND UNDERLYING PATHOPHYSIOLOGY

Ear infections are classified according to anatomical site, involving disease of the external, middle, and inner ear, with clinical expression across acute, recurrent, and chronic phenotypes (8). Among these, middle-ear infections represent the most clinically significant spectrum (8). These conditions comprise a group of complex infectious and inflammatory disorders affecting the middle ear, manifesting in various subtypes that differ in presentation, associated complications, and treatment (8).

The clinical course varies significantly between acute, recurrent, and chronic forms (8). Acute disease typically reflects a self-limited inflammatory response, whereas recurrent and chronic forms indicate persistence of underlying pathological mechanisms (8). Progression from acute to chronic disease is driven by a combination of host and local factors, including:

- **Eustachian tube dysfunction (ETD)**, resulting in impaired ventilation and drainage and sustained negative middle-ear pressure (9).
- **Persistent inflammation**, leading to epithelial disruption, mucosal remodelling, and increased secretory activity (8).
- **Structural extension**, with ossicular erosion and mastoid involvement in advanced disease (10).

In chronic states, these processes are frequently accompanied by ongoing bacterial colonisation, which acts synergistically with host factors to sustain inflammation and disease activity (8,11).

Figure 1 demonstrates how ETD and inflammatory changes anatomically converge to maintain middle ear effusion and promote chronic disease.

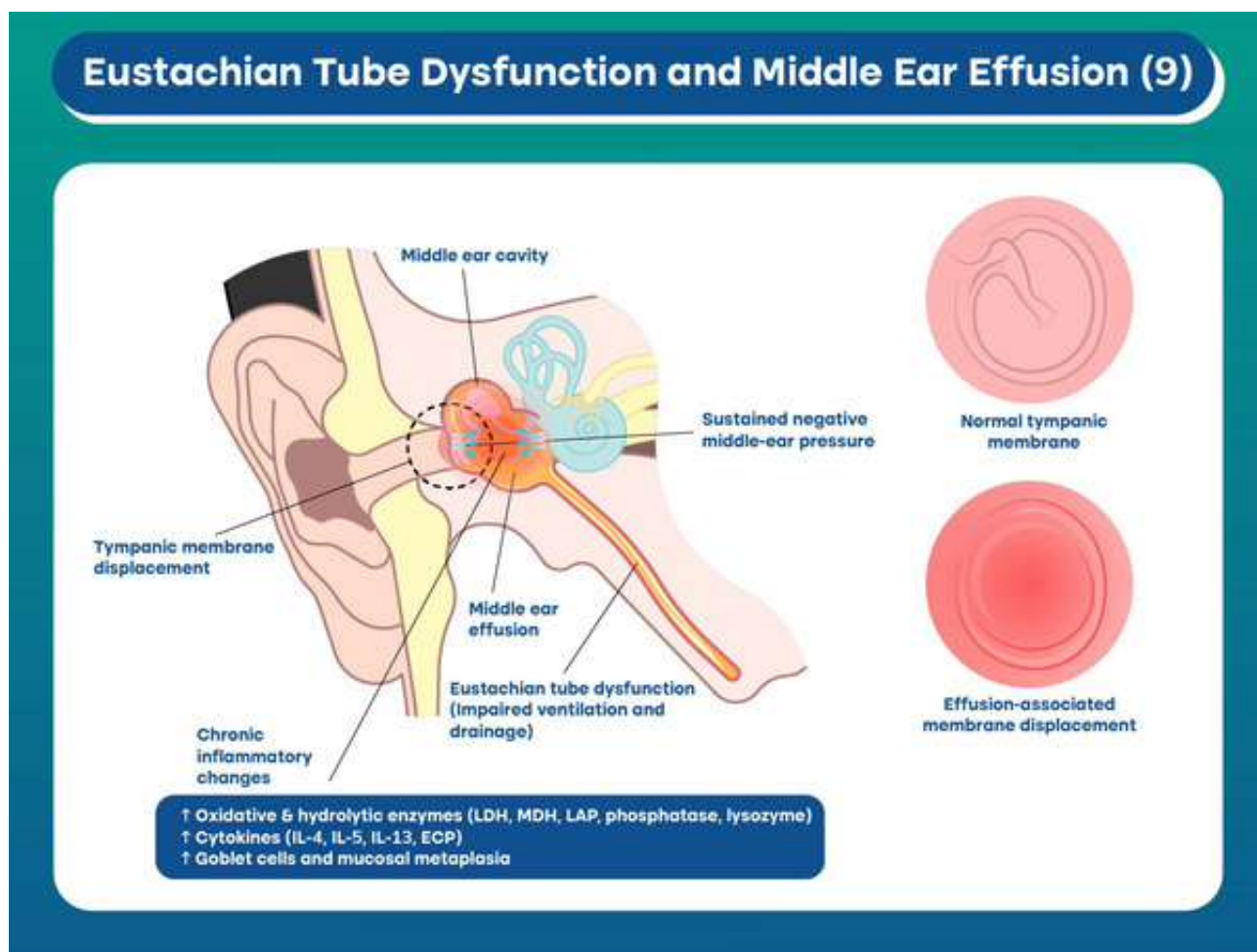


Figure 1. Eustachian tube dysfunction and inflammatory mechanisms underlying middle ear effusion (9)

(Abbreviations: LDH – Lactate dehydrogenase; MDH – Malate dehydrogenase; LAP – Leucine aminopeptidase; IL-4 – Interleukin-4; IL-5 – Interleukin-5; IL-13 – Interleukin-13; ECP – Eosinophil cationic protein)

DIAGNOSTIC EVALUATION AND DISEASE STRATIFICATION

Diagnostic evaluation in ear infections extends beyond confirming disease presence and is primarily directed toward accurate localisation, assessment of clinical significance, and stratification of risk to guide follow-up and escalation of care (12,13). This approach is essential because, while many ear infections resolve spontaneously, a proportion progress due to persistence, recurrence, or structural involvement, leading to long-term morbidity if not identified early (12,14,15).

A structured diagnostic approach begins with anatomical localisation and progressively incorporates disease behaviour and functional impact to enable meaningful risk stratification, as outlined below (12):

- Disease behaviour over time is assessed early, as acute inflammatory ear disease is expected to resolve within defined timelines, whereas persistence or recurrence indicates deviation from physiological resolution and raises concern for chronic mucosal disease or structural pathology (12,14).
- Recurrent disease should not be interpreted as repeated isolated acute episodes, as sustained inflammation reflects altered middle-ear biology that predisposes to effusion persistence and disease progression (8,12).
- Anatomical localisation provides essential diagnostic context by distinguishing external from middle ear disease and identifying features such as effusion, tympanic membrane abnormality, or early structural change that may signal persistence or progression (12,16,17).
- Functional impact, particularly hearing impairment, is integrated into assessment to determine clinical significance, as functional decline may occur even when otoscopic findings appear modest (16).
- Risk stratification emerges from the convergence of disease behaviour, anatomical context, and functional impact, enabling identification of patients at risk of progression or complications versus those suitable for conservative management with surveillance (12,14,15).

The contribution of individual diagnostic modalities to stratification is summarised below in [Figure 2](#)

Diagnostic Modalities and Their Role in Risk Stratification of Ear Infections (12,16,17)		
Diagnostic modality	Role in risk stratification	Role in risk stratification
Otoscopy/ microscopy	• Identifies site of disease and key structural features (e.g., effusion, perforation, retraction) • Differentiates self-limiting inflammation from persistent or progressive pathology	• Persistent symptoms • Recurrent disease • Suspicion of chronic otitis media
Audiological assessment	• Detects and quantifies hearing impairment • Serves as a marker of disease severity and progression beyond visible inflammation	• Persistent or recurrent disease • Functional decline disproportionate to otoscopic findings
Computed tomography	• Defines extent of bony involvement (ossicular erosion, mastoid changes) • Assesses advanced or complicated disease	• Suspected chronic disease • Failure of medical therapy • Concern for structural progression
Magnetic resonance imaging	• Defines extent of bony involvement (ossicular erosion, mastoid changes) • Assesses advanced or complicated disease	• Suspected chronic disease • Failure of medical therapy • Concern for structural progression

Figure 2. Therapeutic decision-making and escalation (12,16,17)

Consequently, imaging is not routinely indicated and is reserved for patients in whom integrated clinical assessment suggests elevated risk or atypical progression (13). Computed tomography (CT) delineates bony and mastoid involvement, while magnetic resonance imaging (MRI) evaluates soft-tissue and intracranial extension, refining the assessment of disease extent and informing the escalation of treatment rather than establishing a diagnosis (13,14).

Early identification of invasive or progressive ear disease is therefore critical, as delayed recognition increases the risk of irreversible morbidity, including permanent hearing loss and intracranial complications (14,15). Clinical concern should be heightened in the presence of persistence despite therapy, recurrent disease, progressive functional decline, or structural otoscopic features such as retraction pockets, granulation tissue, or keratin debris (12,14,15). Equally important is differentiation from non-infective conditions with overlapping presentations when disease behaviour deviates from expected inflammatory trajectories, prompting reassessment of diagnosis and management strategy (12,13).

THERAPEUTIC DECISION-MAKING AND ESCALATION IN COMPLEX EAR INFECTIONS

Therapeutic decision-making in ear infections is driven by the anticipated disease trajectory and the need to prevent irreversible morbidity, rather than by short-term symptom resolution alone (15). Management, therefore, follows a risk-responsive escalation framework, in which treatment intensity increases as evidence of treatment resistance, anatomical progression, and complication risk accumulates (18).

In acute otitis media, initial conservative management is justified by the high likelihood of spontaneous resolution; however, failure to improve or clinical deterioration within 24–48 hours represents deviation from the expected inflammatory course and warrants escalation to systemic antibiotic therapy (19). This threshold reflects evidence that delayed intervention in non-resolving acute disease is associated with prolonged illness and higher downstream risk (20).

The development of recurrent or persistent disease marks a critical shift in therapeutic reasoning, as repeated episodes or sustained symptoms indicate failure of episodic medical management, rather than inadequate treatment duration (20). At this stage, repeated courses of medical therapy alone are unlikely to alter the underlying disease biology, prompting reassessment of middle-ear ventilation, functional impact, and the need for procedural intervention (14,15).

Progression to chronic otitis media (COM) reflects established pathological change, particularly in the presence of persistent otorrhoea, tympanic membrane perforation, or structural middle-ear disease (15). Escalation in this context is driven not by treatment failure alone but by recognition of biological irreversibility, where definitive intervention is required to prevent cumulative tissue damage and functional decline (15).

The identification of cholesteatoma or progressive bony erosion constitutes a clear surgical threshold, as these entities are intrinsically destructive and associated with a high risk of extracranial and intracranial complications if left untreated (21). Accordingly, mastoidectomy remains the treatment of choice in chronic suppurative otitis media (CSOM) with cholesteatoma, aiming to eradicate disease and prevent extension beyond the middle ear (22).

In complicated chronic otitis media, including mastoiditis, facial nerve involvement, or intracranial extension, escalation becomes urgent and frequently multidisciplinary (15,22). Delay at this stage permits continued bone erosion and markedly increases the risk of permanent neurological sequelae, reinforcing the need for prompt surgical intervention alongside appropriate antimicrobial therapy (22).

Taken together, therapeutic escalation in ear infections follows a structured but flexible clinical logic, in which procedural and surgical thresholds progressively lower as treatment resistance persists, anatomical complexity increases, and the risk of irreversible complications accumulates (15,22).

Figure 3 visually organises therapeutic escalation across increasing disease complexity, aligning treatment thresholds with persistence, anatomical progression, and complication risk.

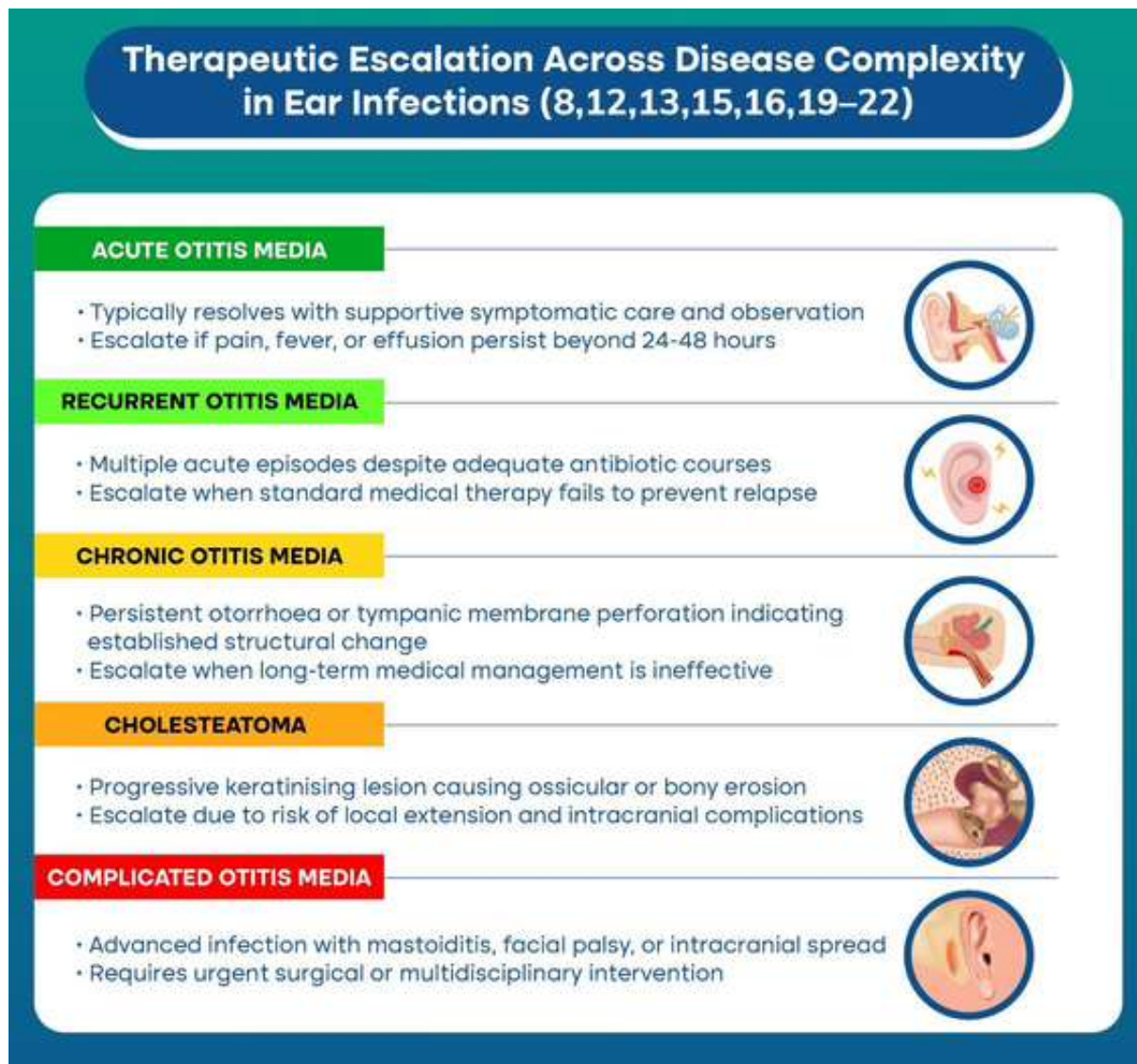


Figure 3. Therapeutic escalation across increasing disease complexity in ear infections (8,12,13,15,16,19–22)

LONG-TERM OUTCOMES, PREVENTION, AND FOLLOW-UP CONSIDERATIONS

Progressive ear infections are associated with sustained morbidity when disease extension is not adequately controlled, particularly in chronic or recurrent phenotypes where inflammatory activity and structural damage persist over time (17,22). Unlike acute presentations, where resolution is common, the long-term burden reflects the cumulative effects of persistent disease, which may continue to evolve even after apparent clinical control (14,22).

Importantly, long-term consequences extend beyond local pathology and involve neurological, functional, and quality-of-life domains, contributing to enduring impairment in communication, balance, and daily functioning

(14,17). These outcomes are shaped not only by disease severity but also by the timeliness of escalation, completeness of disease eradication, and the presence of residual pathology, explaining why chronic disease carries a disproportionate long-term burden (14).

Figure 4 visually organises these long-term consequences of chronic ear infections by clearly separating disease outcomes from surveillance and follow-up imperatives.

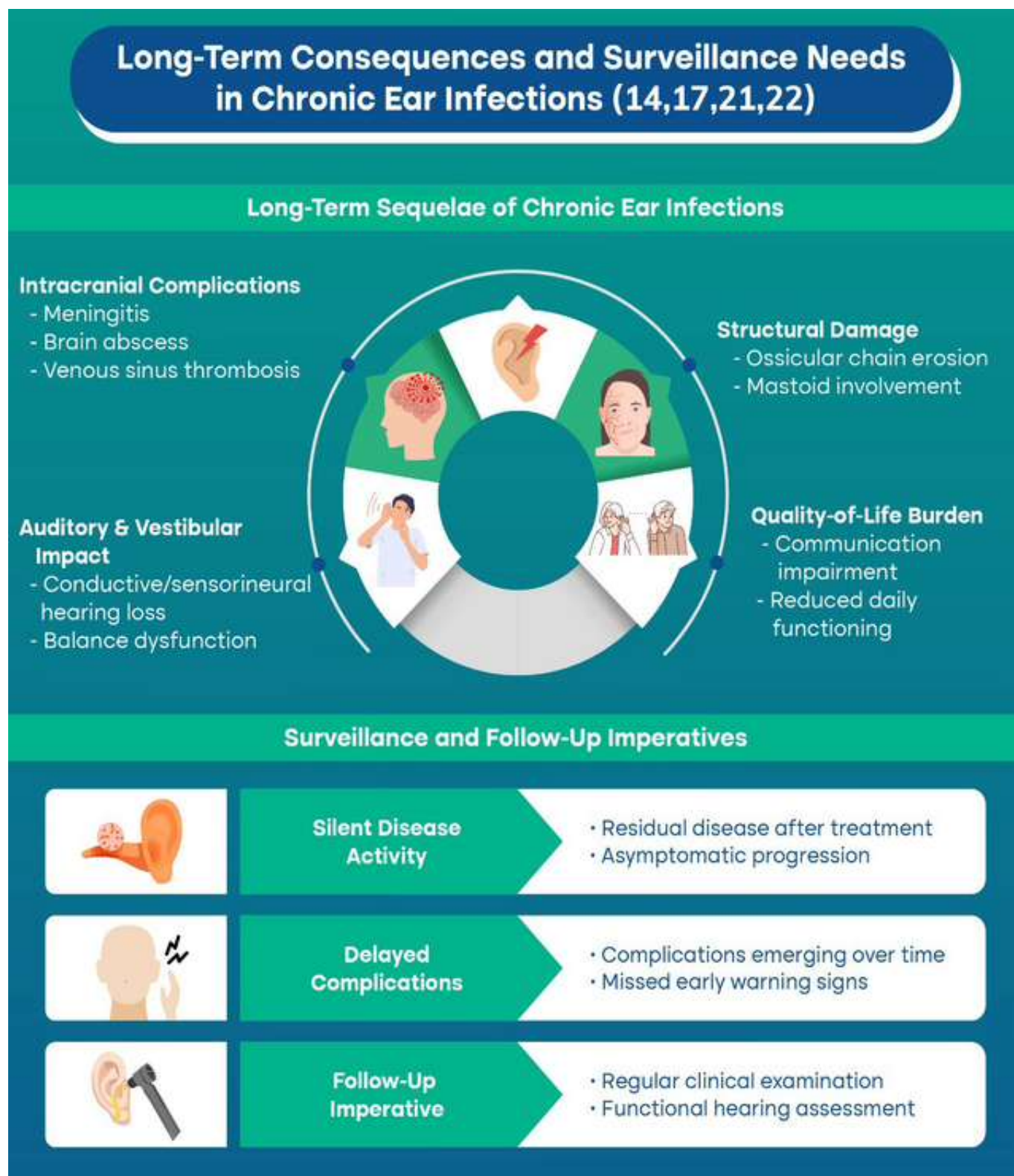


Figure 4. Long-Term outcomes and surveillance needs in chronic ear infections (14,17,21,22)

Following disease resolution or surgical management, prevention of recurrence and early detection of residual disease remain central to preserving long-term outcomes, extending beyond the immediate goal of resolving acute infection (15). Evidence indicates that residual or recurrent disease may remain clinically silent for prolonged periods, particularly after chronic infection or surgery, delaying recognition until irreversible functional decline has occurred (21,22).

Accordingly, structured long-term surveillance, incorporating regular clinical examination and functional assessment, is essential in patients with chronic disease, prior complications, or surgically managed ear infections (14). Such vigilance enables early identification of delayed complications, timely re-intervention, and sustained protection of auditory, vestibular, and neurological function (14).

Taken together, long-term outcomes in ear infections are determined not solely by the acute episode but by the trajectory of disease control over time (22). A proactive follow-up strategy that anticipates delayed complications and functional decline is therefore critical to reducing long-term morbidity and preserving quality of life in patients with chronic or progressive ear disease (14,22).

Key Highlights

- Ear infections represent a major clinical challenge, with acute otitis media affecting hundreds of millions annually and chronic forms causing substantial long-term morbidity (1–3).
- Disease progression is driven by Eustachian tube dysfunction and chronic inflammatory processes, rather than isolated infections (8–11).
- Improving outcomes requires integrating otoscopy, hearing assessments, and selective imaging in higher-risk cases to identify invasive disease early (12,13,15–17).
- Prompt escalation and structured surveillance are essential to prevent irreversible complications and maintain patient quality of life (14,15,21,22).

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A Case Report of Successful Laparoscopic Burch Colposuspension for Stress Urinary Incontinence (SUI) at Aster Hospital, Mankhool, Dubai



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Obstetrics and Gynaecology (Specialist)

PRESENTATION

- 49-year-old female, P3L3
- Obstetric history of last vaginal delivery 13 years back
- Medical history of bronchial asthma
- Admitted with complaints of:
 - Severe stress urinary incontinence
 - Heavy menstrual flow with passage of clots 3-5/28-30

DURING EXAMINATION

- Stable vitals
- Per Abdomen: Soft

A urology consultation was taken, which confirmed demonstrable stress incontinence. The ultrasonography showed a large fibroid abutting the endometrium.

DURING PROCEDURE

The preoperative evaluation included a comprehensive history and physical examination to confirm the diagnosis of stress urinary incontinence, and imaging studies to delineate the fibroid and assess pelvic organ anatomy.

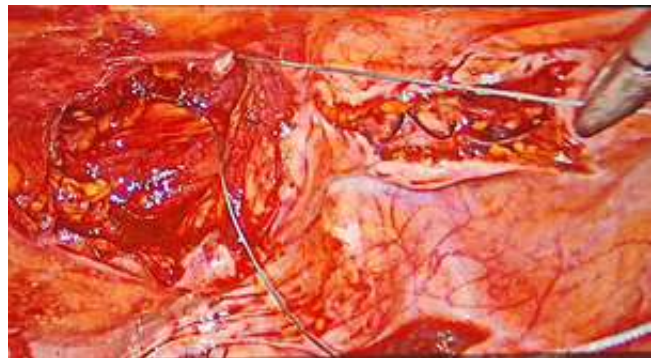
The patient underwent Laparoscopic Burch Colposuspension with concomitant myomectomy:

- Before the procedure, a Foley catheter was inserted, and the patient was positioned in dorsal lithotomy to facilitate both laparoscopic access and potential vaginal manipulation.
- After appropriate sterile preparation and draping, a primary supraumbilical port was established using a direct trocar entry, and subsequent accessory ports were strategically placed to ensure optimal instrument manoeuvrability and visualisation of the retropubic space.
- Pneumoperitoneum was established to 15 mmHg, allowing for clear visualisation of the pelvic structures, including the bladder neck and Cooper's ligaments, essential for the subsequent steps of the colposuspension.

- Initial dissection involved careful identification and mobilisation of the vesicovaginal space to adequately expose the endopelvic fascia, which is crucial for treating any concomitant cystocele and facilitating proper suspension.
- Once the space was adequately developed, permanent sutures were meticulously placed through the periurethral fascia on both sides of the urethra and then secured to Cooper's ligament, ensuring a tension-free elevation of the bladder neck and proximal urethra to achieve continence.
- Concomitantly, a myomectomy was performed to address the patient's symptomatic fibroid, utilising techniques designed to minimise blood loss and preserve uterine integrity.
- This combined approach aimed to address both stress urinary incontinence and the uterine fibroid in a single operative setting, optimising patient recovery and reducing the need for multiple surgical interventions.



Peritoneum was opened to enter Space of Retzius and bladder was gently mobilised



Non absorbable suture was passed through paraurethral fascia and fixed to Cooper's ligament

Benefits of Laparoscopic Burch Colposuspension:

- Elevates and suspends paraurethral and paravaginal tissues to Cooper's ligament, restoring bladder neck support and preventing leakage.
- Offers benefits over open surgery: smaller incisions, reduced blood loss, shorter hospital stays, faster recovery, and less post-operative pain.
- Preferred when synthetic mesh is contraindicated or combined procedures (e.g., paravaginal repair, sacrocolpopexy) are needed.
- Demonstrates comparable success rates to open methods and significant symptom improvement (UDI-6, IIQ-7 scores).

POST PROCEDURE

The patient tolerated the procedure well and had an uneventful post-procedure. Post-operative care focused on early mobilisation, pain management, and vigilant monitoring for potential complications, such as urinary retention or infection, to ensure smooth recovery. Special attention was given to monitoring bladder function post-catheter removal, with post-void residual volumes checked to confirm complete bladder emptying and rule out prolonged urinary retention.

The patient was discharged the next day with significant improvement in urinary continence and reduced menstrual bleeding, indicating a successful combined surgical outcome.

FOLLOW-UP AND LONG-TERM RESULTS

Regular follow-up appointments were scheduled for 2 weeks and 1 month post-surgery to assess continence, monitor uterine healing, and evaluate overall patient satisfaction. Subsequent follow-ups at 3, 6, and 12 months were planned to assess long-term efficacy and to rule out symptom recurrence or new complications.

The patient reported complete resolution of stress urinary incontinence and significantly improved quality of life at her 1-month follow-up, demonstrating the efficacy of this combined laparoscopic approach. This positive outcome aligns with findings from similar studies demonstrating high success rates for laparoscopic colposuspension in the treatment of stress urinary incontinence.

DISCUSSION

This case report meticulously details the first laparoscopic Burch colposuspension performed at Aster, addressing stress urinary incontinence, a prevalent condition affecting up to 25% of women and significantly impacting their quality of life. This surgical intervention offers a highly effective solution for patients whose stress urinary incontinence does not respond to conservative management, highlighting a critical advancement in gynaecological care within the institution.

Stress urinary incontinence, characterised by involuntary urine leakage during activities that increase intra-abdominal pressure, is a common and distressing condition impacting a significant proportion of the female population. Management of stress urinary incontinence typically starts with conservative approaches like pelvic floor exercises, lifestyle modifications, and medications for refractory cases, progressing to surgical interventions when necessary. While conservative treatments are often initially pursued, surgical interventions, such as Burch colposuspension, are frequently necessary for refractory cases and can be performed using either an open or a laparoscopic approach. The laparoscopic approach to Burch colposuspension offers several advantages over traditional open surgery, including smaller incisions, reduced hospital stays, faster recovery times and improved aesthetic outcomes.

Despite the emergence of tension-free midurethral slings, such as transobturator vaginal tape, as the most common surgical option with minimally invasive benefits, including shorter operative times and hospital stays, laparoscopic Burch colposuspension remains a viable alternative. It is preferred in scenarios involving concomitant procedures like paravaginal repair or sacrocolpopexy, or when synthetic mesh implantation is contraindicated.

Laparoscopic Burch colposuspension has long been a cornerstone in the surgical management of stress urinary incontinence, offering robust anatomical support and durable continence rates. Its efficacy is well-established, with studies comparing laparoscopic and open approaches demonstrating similar long-term success rates, though laparoscopic methods typically afford quicker recovery and less post-operative pain. Specifically, laparoscopic Burch colposuspension has demonstrated significant reductions in UDI-6 and IIQ-7 scores, indicating substantial improvement in urinary incontinence symptoms and overall quality of life at the 6-month follow-up. Moreover, advantages such as shorter hospital stays, reduced estimated blood loss, and decreased post-operative pain are consistently reported with the laparoscopic approach compared to traditional open methods.

Laparoscopic Burch Colposuspension + Myomectomy Integration:

The integration of myomectomy within the same operative session for a patient presenting with both stress urinary incontinence and a symptomatic fibroid further highlights the efficiency and

patient-centric benefits of a combined laparoscopic strategy. This approach not only addresses multiple gynaecological concerns concurrently but also leverages the inherent advantages of minimally invasive surgery, such as reduced recovery times and improved cosmetic outcomes, in line with patient preferences and current clinical advisories.

CONCLUSION

Therefore, for patients seeking to avoid potential mesh-related complications or when mesh use is contraindicated, laparoscopic Burch colposuspension offers a robust and effective surgical solution. This approach, while technically demanding, has demonstrated comparable long-term efficacy to open procedures and offers the added advantages of minimally invasive surgery, such as reduced post-operative pain and shorter recovery periods. Given these advantages and the desire to provide mesh-free surgical options, the successful performance of the first laparoscopic Burch colposuspension at Aster Hospital Mankhool represents a significant advancement in local urogynecological care.

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Fever in Cancer Patients: From Febrile Neutropenia to Risk-Adapted Care



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INTRODUCTION

Fever is commonly observed among patients with cancer and represents one of the leading reasons for urgent medical evaluation in oncology practice (1). These febrile episodes are defined by clinically significant elevations in body temperature that necessitate immediate assessment to determine the need for medical intervention (1). Such occurrences are prevalent across diverse cancer populations, with febrile illness developing in up to 80% of patients with haematological malignancies, with approximately 10–50% patients receiving cytotoxic chemotherapy for solid tumours (2,3). Ultimately, the frequent need for hospital admission, intensive diagnostic workups, and interruptions to scheduled anticancer therapy result in fever remaining a major source of morbidity and healthcare utilisation in cancer care (1,3).

Although considered a significant clinical feature, fever must be approached as a diagnostic sign rather than a disease entity, because it can be caused by a wide range of infectious and non-infectious causes that require distinct management strategies (1). Among these two, infections are the principal cause and must be managed as a priority in both neutropenic and non-neutropenic patients (1). In addition to this, pyrexia may also arise from iatrogenic or inflammatory mechanisms such as mucositis, specific medications, blood transfusions, radiotherapy, or surgical procedures (1).

Considering the different risks involved, timely determination of the underlying aetiology becomes essential to minimise mortality and ensure patient safety (1). In high-risk settings, especially febrile neutropenia (FN), delayed recognition and initiation of empirical antimicrobial therapy are often associated with sepsis and poor clinical outcomes (2,4). Conversely, failure to recognise non-infectious fever can lead to unnecessary antimicrobial exposure and avoidable disruption of anticancer treatment (1). A systematic, risk-adapted evaluation is therefore fundamental to optimising the patient outcomes while preserving the effective delivery of contemporary cancer therapy (4).

Read more for a deeper understanding of the differences between febrile neutropenia and non-neutropenic fever, an examination of infectious and non-infectious causes of fever in patients with cancer, followed by risk-adapted diagnostic and management strategies applicable to contemporary oncology practice.

KEY DIFFERENCES BETWEEN FEBRILE NEUTROPENIA AND NON-NEUTROPENIC FEVER

FN and non-neutropenic fever (NNF) represent distinct clinical fever syndromes in oncology, defined primarily by the presence or absence of profound neutrophil depletion and the resulting differences in host defence (5,6).

This distinction is clinically important because neutrophils are the hallmark of immunodeficiency in patients with cancer, and their quantitative and functional loss fundamentally alters susceptibility to bacterial and fungal invasion (7).

Fever in neutropenic and non-neutropenic patients arises through different biological pathways, producing divergent patterns of infection, inflammation, and early clinical deterioration (5,7). **Figure 1** summarises their biological, microbiological, and prognostic contrasts.

FN and NNF as Distinct Clinical Syndromes (5–10)		
	FN	NNF
Biological definition and immune state		
Definition	• Fever $>38.3^{\circ}\text{C}$ or $\geq 38.0^{\circ}\text{C}$ sustained for ≥ 2 hours • ANC $<0.5 \times 10^9/\text{L}$ or expected to fall	Fever in a patient with active cancer and ANC >1000
Innate immune status	Profound quantitative and functional neutrophil deficiency	Preserved neutrophil number and function
Primary mechanism	Uncontrolled microbial invasion with systemic inflammatory response	Cytokine-mediated inflammation from tumour, therapy, or focal infection
Infection risk and microbiology		
Bacteraemia frequency	~20% of episodes are complicated by bloodstream infection	Bacteraemia occurs, but at lower and more variable rates
Dominant pathogens	Gram-negative and Gram-positive organisms with increasing antimicrobial resistance	More stable epidemiologic pattern
Fungal infection risk	Substantial in prolonged or profound neutropenia	Uncommon unless additional immunosuppression is present
Clinical risk and management implications		
Short-term risk	20–30% develop complications; ~10% mortality, up to ~40% in high-risk patients	Severe illness in a substantial proportion; short-term mortality ~4%
Risk stratification	MASCC and CISNE classify low- vs high-risk patients	No validated risk tools
Impact on cancer therapy	May lead to chemotherapy delay, dose reduction, or discontinuation	Seen in solid and haematologic cancers, including CAR-T and transplant patients
High-risk populations	Hematologic malignancies, stem-cell transplant, CAR-T therapy	Occurs across solid and hematologic cancers, including CAR-T and transplant patients

Figure 1. Defining characteristics and infection-related features of febrile neutropenia and non-neutropenic fever (5–10)

(Abbreviations: ANC – absolute neutrophil count; CAR-T – chimeric antigen receptor T-cell; CISNE-Clinical Index of Stable Febrile Neutropenia; FN – febrile neutropenia; MASCC – Multinational Association for Supportive Care in Cancer; NNF – non-neutropenic fever)

FN develops due to impaired phagocytic activity, defective neutrophil migration (chemotaxis), and reduced intracellular pathogen clearance, allowing even minor infections to progress rapidly to severe sepsis (5,7). Early recognition and risk-adapted management are, therefore, critical for survival in these patients (5). In contrast, NNF occurs in patients with a relatively intact immune system, where tumour-related inflammation, therapy-induced cytokine release, or focal infection may coexist and evolve unpredictably (6).

These mechanistic differences explain why validated risk stratification tools, such as the Multinational Association for Supportive Care in Cancer (MASCC) and Clinical Index of Stable Febrile Neutropenia (CISNE) scores, apply to FN but not to NNF (11). Consequently, FN is universally treated as an oncological emergency, whereas NNF requires more individualised clinical assessment and diagnostic judgement (11).

The complexity of fever syndromes further increases in haematological malignancies, following stem cell transplantation, and in chimeric antigen receptor T-cell (CAR-T) therapy recipients (10). In these patients, immune dysregulation, prolonged cytopenias, and cytokine-driven inflammation often overlap with infectious processes, blurring the boundaries between infection-related and immune-mediated fever (10). This underscores the importance of integrating clinical, microbiological, and immunological assessment when evaluating fever in advanced oncological settings (10).

PATHOPHYSIOLOGICAL DRIVERS AND DIAGNOSTIC PITFALLS OF NON-INFECTIOUS FEVER

Fever in patients with cancer may be linked to a range of infectious and non-infectious causes, making an accurate differential diagnosis essential to avoid both delayed sepsis treatment and unnecessary antimicrobial exposure (1). The major non-infectious causes of fever in oncology fall into four biologically distinct categories (1):

A. Drug-related fever, often triggered by agents such as bisphosphonates, β -lactams, piperacillin, acyclovir, amphotericin B, trimethoprim/sulfamethoxazole, and vancomycin, causing hypersensitivity reactions and reflecting immune-mediated inflammation (12). Diagnostic challenges arise because the clinical presentation can mimic sepsis, with identification relying on recognising specific oncology-relevant triggers (1).

B. Tumour-related (neoplastic) fever occurs most often in Hodgkin and non-Hodgkin lymphoma, leukaemia, and renal cell carcinoma (RCC) (1). It is driven by tumour-derived cytokines, including tumour necrosis factor α , interleukins 1 and 6, and interferons, which promote prostaglandin E2 synthesis and result in sustained pyrexia (1). Because neoplastic fever shares many clinical features with infectious fever, differentiation between the two is often difficult (1,13). In contrast to infectious fever, neoplastic fever typically lacks significant tachycardia or rigours and shows a prompt resolution after naproxen administration, known as the naproxen test, which supports a neoplastic aetiology once infection has been reasonably excluded (1). Due to this overlap, careful assessment is required to prevent misinterpretation and to ensure appropriate management (13).

C. Immunotherapy-related fever is caused by the use of immune checkpoint inhibitors (ICIs), which can sometimes induce fever due to an activated immune system and elevated cytokine levels (14). In these cases, fever can appear as an isolated immune-related adverse event or as part of a broader systemic toxicity (14). Because ICIs trigger widespread immune activation, distinguishing between immune-mediated fever and infection can be difficult, particularly in patients on corticosteroids or concurrent immunosuppressants (14,15). A structured evaluation, including clinical context, timing of onset, and exclusion of infection, is essential to guide management and determine whether immunosuppressive therapy or antimicrobial treatment is required (10,16).

D. Following CAR-T cell therapy, fever is a characteristic manifestation of **cytokine release syndrome (CRS)** (13). It reflects rapid immune activation with the release of inflammatory cytokines, including IL-6 and IFN- γ , and is

commonly associated with early, frequently high-grade pyrexia (13).

Figure 2 highlights the major diagnostic pitfalls in cancer-related fever to demonstrate why these non-infectious fever syndromes are so frequently misinterpreted.

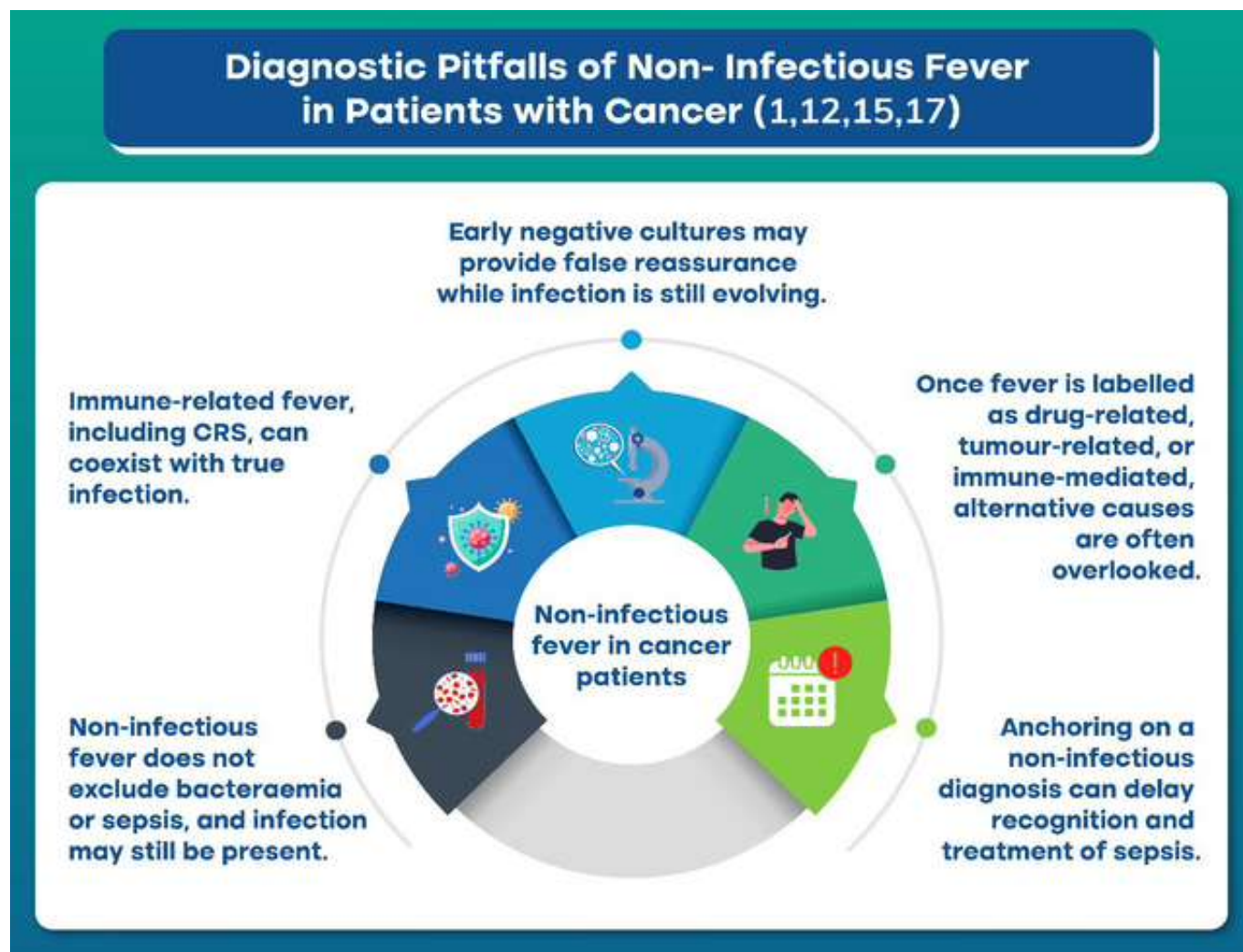


Figure 2. Diagnostic pitfalls contributing to delayed recognition of infection in febrile oncology patients (1,12,15,17)

(Abbreviations: CRS-cytokine release syndrome)

DIAGNOSTIC EVALUATION AND RISK-ADAPTED ANTIMICROBIAL STEWARDSHIP IN FN

FN constitutes an oncological emergency necessitating the prompt initiation of empiric broad-spectrum antimicrobial therapy (4). Clinical assessment, laboratory testing, and imaging should be undertaken in parallel with treatment initiation and should not postpone antibiotic administration, as early therapy is essential to lowering infection-related complications and mortality in neutropenic patients (4).

Regardless, these thorough evaluations are necessary to exclude non-infectious causes of fever, identify the clinical focus and responsible pathogens, and assess the severity of inflammation to guide further management (4):

- **Clinical examination** during the initial presentation and daily afterwards for hospitalised febrile patients helps determine a clinical focus and appropriate antimicrobial strategy for the causative pathogens (4).

- Minimum two sets of **blood culture samples**, typically drawn from separate puncture sites, before the initiation of antimicrobial therapy (4). In the case of indwelling central venous catheters, one additional pair should be drawn from the catheter to increase the diagnostic yield (4).
- **Imaging studies** guided by clinical symptoms are recommended (4). For example, a low-dose chest CT in cases with respiratory symptoms or an abdominal CT when an abdominal focus for fever is suspected (4).

These diagnostic findings and early treatment decisions are integrated with clinical risk assessment and stewardship principles, presented in **Figure 3**.

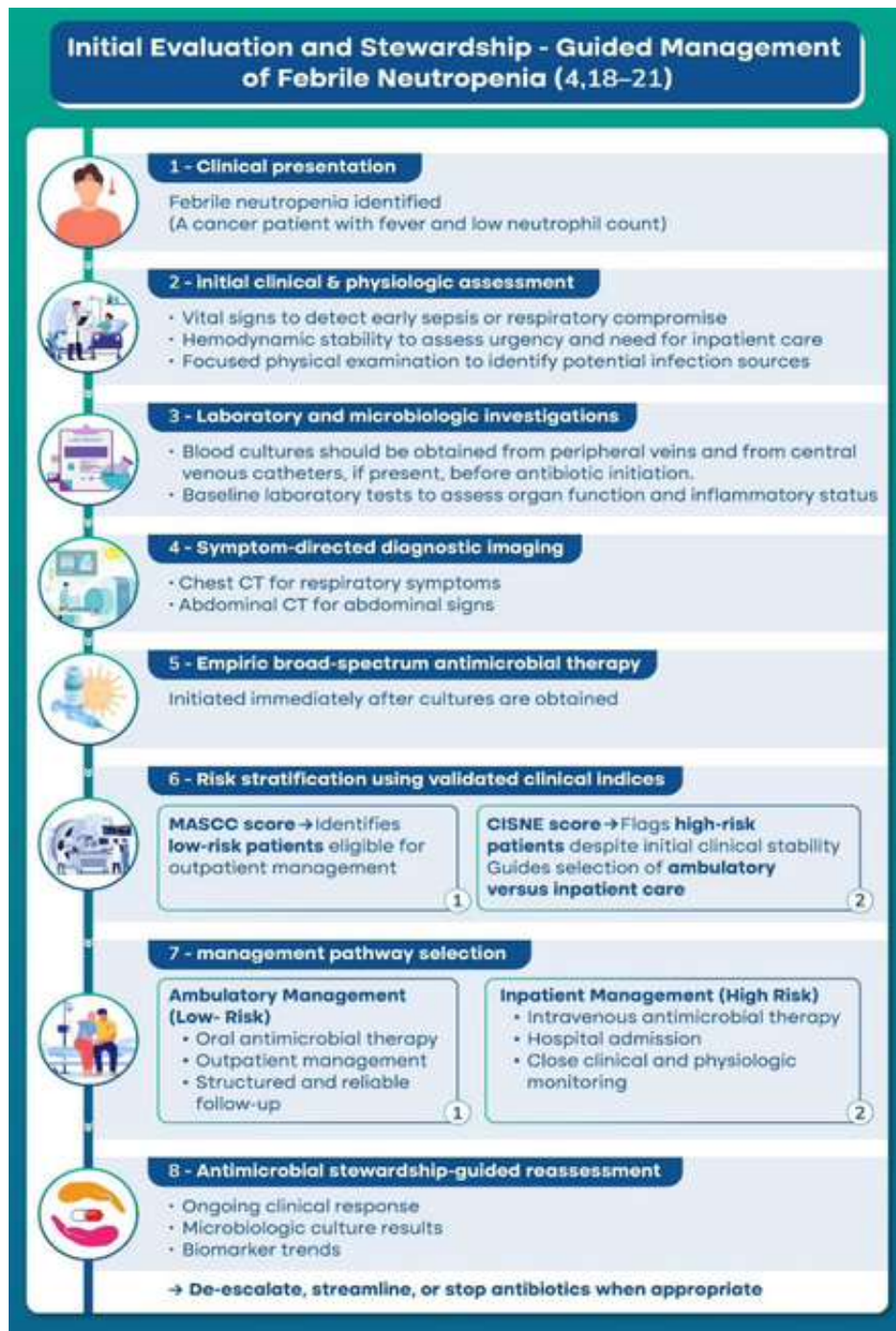


Figure 3. Initial diagnostic workup and antibiotic stewardship pathway in febrile neutropenia (4,18-21)

(Abbreviations: CT – Computed Tomography; MASCC – Multinational Association for Supportive Care in Cancer; CISNE – Clinical Index of Stable Febrile Neutropenia)

While early empiric therapy is essential, only a proportion of patients with FN develop severe complications (20). This variability underscores the importance of risk stratification, which allows clinicians to tailor management intensity to individual patient risk (20). Validated clinical indices such as the MASCC score and the Clinical Index of Stable Febrile Neutropenia (CISNE) score are routinely used to differentiate low-risk from high-risk cases (20).

Building on this risk-based evaluation, febrile neutropenia management incorporates three core antimicrobial-stewardship principles recommended across recent international guidelines (1,4).

- **Low-risk patients may receive simplified oral monotherapy** and immediate or early discharge following an observation period (20).
- **Antimicrobial stewardship** programs play a key role in promoting judicious antibiotic use without compromising patient safety (18). Although broad-spectrum agents are essential initially, prolonged and indiscriminate use is associated with the emergence of antimicrobial resistance, alongside drug-related toxicities, and rising healthcare costs (18).
- Accordingly, experts and guidelines advocate **antimicrobial de-escalation strategies** in clinically stable patients to limit unnecessary broad-spectrum antibiotic exposure (18).

This approach ensures that the intensity of care matches the patient's individual risk profile, allowing for treatment of high-risk sepsis while preventing the complications associated with over-treatment in stable populations (1,18).

Therapeutic Escalation and De-escalation in Febrile Cancer Care

Fever in cancer patients reflects a dynamic clinical state, influenced by infection, immune activation, and marrow suppression, rather than a static diagnosis (4). Clinical management, therefore, requires ongoing reassessment to determine when to escalate or de-escalate antimicrobial or immunomodulatory therapy in response to evolving risk (4).

The principal factors influencing these decisions are summarised in **Figure 4**, which outlines how clinicians balance infection risk, immune toxicity, and recovery signals throughout the course of febrile illness.

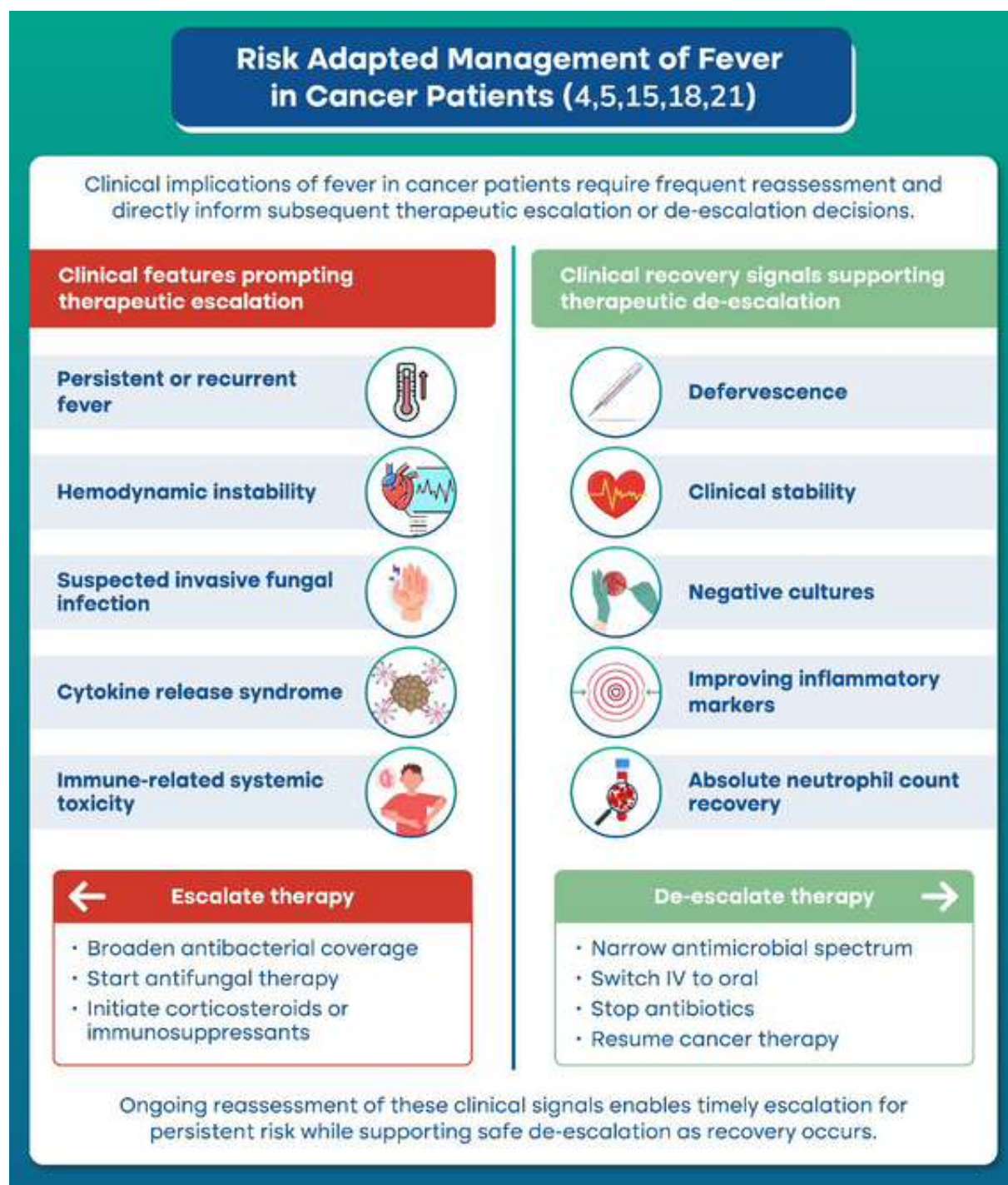


Figure 4. Clinical signals informing therapeutic escalation and de-escalation in febrile cancer care (4,5,15,18,21)

INDICATIONS FOR IMMUNOMODULATORY THERAPEUTIC ESCALATION

Persistent or recurrent fever in high-risk neutropenic patients warrants prompt reassessment because the guideline group recommends empiric therapy with a mould-active antifungal agent in cases of fever persisting beyond 72–96 hours or recurring despite appropriate antibacterial treatment (4). International guidelines, including the European Society for Medical Oncology (ESMO), similarly recommend empirical initiation of antifungal therapy when fever doesn't respond to broad-spectrum antibiotics after 3–7 days of adequate treatment (5).

Recommended agents include:

- Liposomal amphotericin B
- Echinocandins such as caspofungin or micafungin (1).

The selection is guided by prior azole exposure and epidemiology (4,5). Furthermore, cytokine release syndrome and immune-related toxicities generate systemic inflammation that requires corticosteroids or other immunosuppressive therapy rather than antimicrobial intensification, making accurate differentiation critical to patient safety (15).

PRINCIPLES OF THERAPEUTIC DE-ESCALATION

De-escalation becomes appropriate once clinical recovery and defervescence are achieved (4). Evidence from recent guidelines supports that empirical antibiotics can be safely discontinued after 72 hours of apyrexia and clinical stability, irrespective of neutrophil count (4). European guidelines further specify that discontinuation is appropriate after 72 hours in haemodynamically stable patients who have remained afebrile for a minimum of 48 hours (4).

This stewardship- driven approach reduces unnecessary exposure, limits the development of antimicrobial resistance, and minimises treatment-related toxicities (21). Additionally, fever resolution also directly influences cancer therapy decisions, because treatment resumption is typically considered once patients are afebrile and have achieved an absolute neutrophil count $\geq 0.5 \times 10^9/L$ for 24 hours (4,5).

The management of fever oncology is a precision-based exercise in risk assessment. While delayed escalation in persistent fever increases the risk of invasive fungal infections and poor outcomes, over-treatment through prolonged broad-spectrum exposure drives resistance and healthcare costs (4,18). By integrating timely escalation for high-risk patients with structured, guideline-directed de-escalation for stable populations, clinicians can protect patients from acute sepsis while preserving the continuity and safety of contemporary cancer therapy (5,21).

Key Highlights

- Fever in cancer reflects both infection and immune dysregulation, requiring structured diagnostic evaluation rather than empirical treatment alone (1,4).
- It can present as febrile neutropenia or non-neutropenic fever, which are biologically distinct syndromes with different risks, microbiology, and management priorities (5–7,12).
- Diagnostic evaluation is frequently complicated by overlapping inflammatory processes, increasing the risk of misclassification and delayed recognition of infection (1,11,12,14).
- Risk-adapted escalation, antimicrobial stewardship, and timely de-escalation are essential to protect patient safety while preserving cancer treatment delivery (4,7,18,21).

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An Emergency Case of Ruptured Globe Repaired in One Sitting at Aster Hospital, Al Qusais, Dubai



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Ophthalmology (Specialist)

PRESENTATION

- 36-year-old male
- Work site injury led to complete loss of vision in the left eye
- Medical history of hypertension for 2 years and scrotal and perineal abscess managed with drainage

FINDINGS

During examination:

- Vision with glasses:
 - Right eye: 6/6
 - Left eye: ?Perception of light
- IOP:
 - Right eye: 24mmHg
 - Left eye: Not recorded

On slit-lamp examination:

- Right eye: Normal
- Left eye: Corneal full-thickness laceration extending from limbus to limbus with clotted blood incarcerated in the wound and ?vitreous prolapse
- Anterior chamber: Entirely filled with hyphema
- No other structures visible
- Fundus: B-scan showed signs of vitreous haemorrhage and retinal detachment

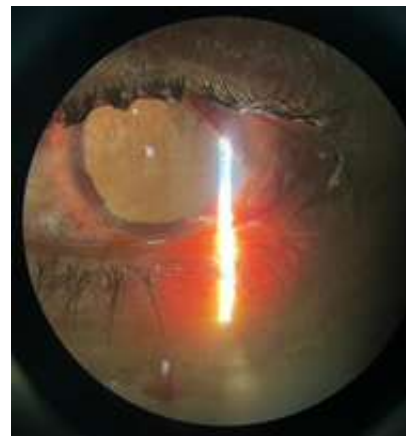
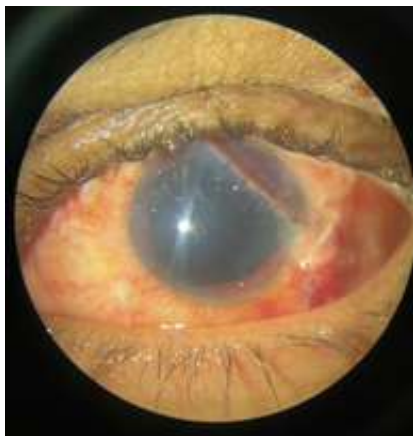


Pre-op image

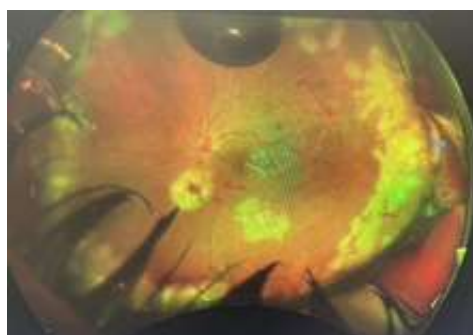
DURING PROCEDURE

The patient underwent Primary Wound Repair of the Ruptured Globe and Retinal Detachment Surgery in the same sitting under general anaesthesia:

- Primary wound repair of the cornea and sclera was done.
- Hyphema in the anterior chamber cleared, and the iris tissue was found to be totally absent.
- Lens material was also not found, with only signs of capsular remnants.
- Three scleral ports were made, and the retina was visualised.
- Complete nasal and inferior bullous retinal detachment identified.
- PFCL injection-assisted retinal re-attachment was done, followed by 360-degree endolaser.
- Silicone oil-PFCL exchange was performed.
- Scleral ports were removed and sutured with 8-0 Vicryl.
- Antibiotic eyedrops were applied, and the eye was patched.



Post-op images on slit-lamp examination



Optos of Retina

POST PROCEDURE

The patient tolerated the procedure well and was in a stable condition on the next day. The globe was found intact with no leaks, and the retina was found to be attached.

On follow-up at 1 week, the eye was found to be stable, and vision had improved to counting fingers at 2 meters. He was advised to have regular follow-ups.

DISCUSSION

The management of a ruptured globe complicated by vitreous haemorrhage (VH) and retinal detachment (RD) remains one of the most challenging scenarios in ophthalmic trauma. Traditionally, the "staged approach" primary closure followed by pars plana vitrectomy (PPV) 7 to 14 days later has been the gold standard. However, this case demonstrates that **immediate, single-setting vitreoretinal surgery** can achieve anatomical success and rapid visual rehabilitation even in the presence of severe trauma, including hyphema, aphakia, and aniridia.

The classical delay in vitrectomy was designed to allow for corneal clearing, spontaneous posterior vitreous detachment (PVD) induction, and reduced uveal congestion. In this patient, however, the severity of the injury, characterised by a limbus-to-limbus laceration and complete loss of iris and lens, necessitated a proactive approach.

The primary advantage observed here was the **early removal of the inflammatory component**. By promptly clearing the hyphema and vitreous haemorrhage, we eliminated the scaffolding for fibrovascular proliferation, thereby significantly reducing the long-term risk of **Proliferative Vitreoretinopathy (PVR)**.

The success of a single-setting repair hinges on intraoperative stability. Once the corneal and scleral lacerations were sutured to create a water-tight seal, the eye could withstand the infusion pressure required for vitrectomy surgery.

Key surgical manoeuvres in this case included:

- **Anterior Segment Washout:** Vital for visualizing the posterior pole.
- **Use of Perfluorocarbon Liquid (PFCL):** As noted in the procedure, PFCL was instrumental in unfolding the nasal and inferior bullous detachment and stabilising the retina for endolaser.
- **Silicone Oil Tamponade:** Given the high-risk nature of the injury and the absence of the iris/lens diaphragm, silicone oil provided the necessary long-term internal tamponade to prevent phthisis bulbi.

The total absence of iris tissue and lens material (traumatic aniridia and aphakia) suggests a massive expulsion of intraocular contents at the time of impact. Despite this empty anterior segment, the patient's vision improved from Bare Light Perception to **Counting Fingers at 2** meters within just one week. This rapid gain validates the decision to reattach the retina immediately, as it prevented the secondary neuroretinal degradation that often occurs during the waiting period of staged repairs.

Features	Staged Approach (Traditional)	Single-Setting (Our Case)
Risk of PVR	Higher due to retained blood/fibrin	Lowered by early evacuation
Surgical Load	Two separate anaesthetic events	Single anaesthetic event
Visualisation	Easier (after cornea clears)	Challenging (requires washout)
Recovery	Delayed visual rehabilitation	Faster anatomical stabilisation

CONCLUSION

This case highlights that with modern wide-angle viewing systems and PFCL, the primary goal of trauma surgery should always consider **comprehensive globe reconstruction** as an alternative to mere wound closure and staged repairs whenever possible. In cases where the wound can be securely closed, immediate PPV and RD repair offer the best chance at salvaging functional vision and preventing the devastating cycle of PVR and hypotony.

In conclusion, when the wound can be securely sealed and visualisation is adequate, primary comprehensive repair should be considered the preferred strategy to maximise visual salvage and prevent the progression toward phthisis bulbi.

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