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HealthNews DIGEST

JANUARY 2026

4 Ship Technique

10 Renal Cell Carcinoma

18 Severe Panfacial Trauma

25 Bladder Cancer

37 Laser Cystolithotripsy via Mitrofanoff

36th Edition

Dr. Sherbaz Bichu

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

On behalf of Aster's leadership, I am pleased to welcome you to the 36th edition of HealthNews.

As we step into the New Year, I extend my warmest wishes to each of you for a healthy, fulfilling, and impactful year ahead. This new beginning also presents an opportunity to reaffirm our shared commitment to advancing the standards of care across Aster Hospitals and Clinics.

Every year, Aster sets new benchmarks in quality care through our unwavering focus on patient safety, clinical excellence, and empathy. Rooted in compassion and aligned with our Chairman's vision of prioritising lives above all else, we remain committed to delivering exceptional patient outcomes through a truly patient-centric approach.

The future of healthcare at Aster is also being shaped by rapid technological advancements. Innovations such as personalised medicine, telehealth expansion, AI and machine learning, wearable technologies, robotics and automation, and blockchain-enabled systems are transforming the delivery of care.

As clinicians, our expertise and commitment remain central to this transformation. Let's continue to push boundaries and set new standards of care and excellence this year as well.

Dr. Ramanathan V

Medical Director
Aster Hospitals & Clinics,
UAE

As the Group Medical Director of Aster Hospitals and Clinics, I am delighted to welcome you to the 36th edition of HealthNews.

This edition shows a strong reflection of the surgical excellence across Aster Hospitals, showcasing our most complex, challenging, and successful procedures. These cases are not only clinical milestones but powerful demonstrations of our expertise, precision, multidisciplinary collaboration, and unwavering commitment to patient care and outcomes.

Each featured case reinforces the depth of capability within our surgical teams. The advanced techniques and innovative approaches highlight the excellence that shows every success is built on teamwork and compassion.

Let us continue to raise the bar for clinical excellence as we step into the New Year. I thank you sincerely for your dedication and encourage you to take pride in the exceptional work you do every day and celebrate the successes and milestones we have achieved together as a team.

Wishing you and your families a prosperous and rewarding New Year.

Dr. Shipra Shrivastava
Cardiothoracic Surgery (Consultant)

Dr. Sandeep Shrivastava
Cardiothoracic Surgery (Consultant)

Ship Technique: Repair over Replacement

Preserving the Native Valve in Aggressive Rheumatic Mitral Valve Disease at Aster Hospital, Al Qusais, Dubai

PRESENTATION

Case 1:

- 31-year-old male
- History of **cerebrovascular disease** 3 months back
- Surgical history of **Closed Mitral Valvotomy (CMV)** for Mitral Stenosis
- Family history of Myocardial Infarction (MI) and hypertension
- Complaints of shortness of breath at rest, NYHA III-IV
- Admitted for:
  - **Redo** Mitral valve surgery

Case 2:

- 44-year-old male
- Complaints of dyspnea on mild exertion for 2 months, NYHA III-IV
- Admitted for:
  - Mitral valve surgery and aortic valve replacement **(double-valve surgery)**

Case 3:

- 35-year-old male
- Complaints of chest heaviness, shortness of breath, and dizziness for a month, NYHA IV
- Admitted for:
  - Mitral valve surgery with **severe pulmonary arterial hypertension (90 mmHg)**

FINDINGS

During Examination:

Case 1: Echo showed:

- Rheumatic heart disease
- Critical mitral stenosis, **peak gradient of 24 mmHg**
- **Mitral valve area: 0.54 cm^2**
- Severe pulmonary arterial (PA) hypertension, PA pressures of 45 mmHg
- Dilated left atrium
- Severe subvalvular fusion

Case 2: Echo showed:

- Rheumatic heart disease
- **Severe calcific mitral stenosis**
- **Moderate mitral regurgitation**
- **Severe aortic regurgitation**
- **Moderate calcific aortic stenosis**
- Normal sinus rhythm
- Severe pulmonary arterial (PA) hypertension, PA pressures of 43 mmHg
- Dilated left atrium
- Severe subvalvular fusion

Case 3: Echo showed:

- Rheumatic heart disease
- Critical mitral stenosis, **peak gradient of 35.86 mmHg**
- Moderate mitral regurgitation
- **Atrial fibrillation**
- Severe pulmonary arterial (PA) hypertension, **PA pressures of 90 mmHg**
- Dilated left atrium
- Severe subvalvular fusion

DURING PROCEDURE

All the patients underwent Mitral Valve Repair (Ship Technique) supported with Annuloplasty Band:

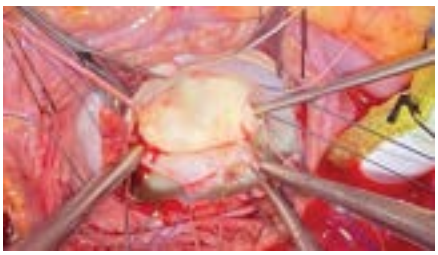
- A peripheral line, 16 G IJV, PA sheath and Swan Ganz catheter were inserted under general anaesthesia.
- Median Sternotomy was performed.
- Pericardium was harvested, cleaned and prepared to perform commissural reconstruction.
- The patient was heparinised.
- Cardio-pulmonary bypass (CPB) was instituted.
- LA approached trans-septal, and the LA appendage was closed using 4-0 proline sutures.
- Bilateral commissurotomy was done.
- The anterior and posterior mitral leaflets thinning was carried out.
- Postero-medial commissural reconstruction was done with the **Ship Technique.**

- One pair of commissural neo-chords was placed using CV-6 sutures.
- Papillary muscle splitting and chordal splitting were performed.
- In all the repairs, the annulus was always supported with Annuloplasty rings.
- A water test was performed. Good repair was achieved on the water test.
- Weaned off CPB.
- Post CPB Echo assessment revealed excellent Valve repair and normal left ventricular function.
- The patient was shifted to the ICU with stable haemodynamic, ventilatory support and mild inotropic support.

Intra-op Images

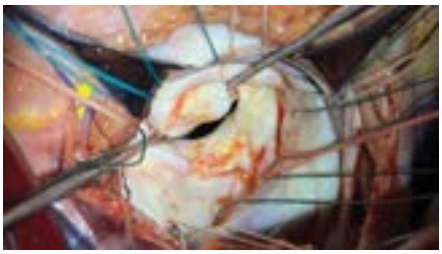
Case 1

Extensive thickening, fusion and calcification

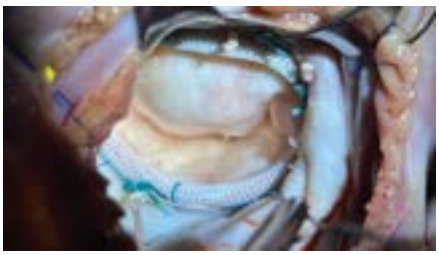


Leaflet appearance after aggressive peeling

Case 2

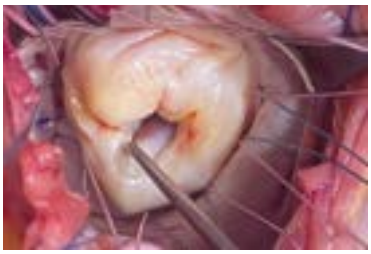


Mitral valve with severe calcification and fibrosis

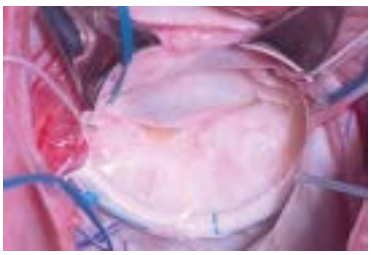


Face change with excellent function

Case 3



Advanced fibrosis and fusion



Native valve appearance after repair

SHIP TECHNIQUE: WHAT IS IT AND HOW IS IT USEFUL?

The commissures play a vital role in the hinge mechanism of mitral leaflets. They are an essential component of the mitral valve apparatus, which helps in the seamless functioning of the mitral leaflets during both systole and diastole.

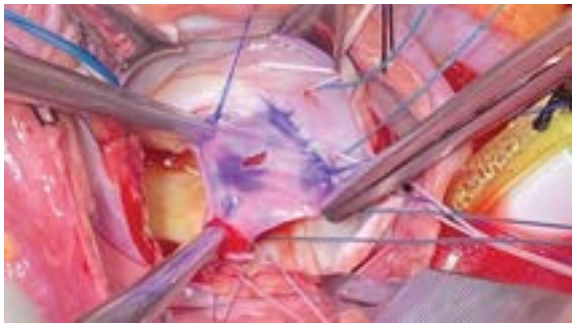
In Rheumatic Heart Disease (RHD), all the components of the mitral valve are involved. There is often extensive fusion, fibrosis, and even calcification of leaflets and commissures. In the standard repair, only a commissurotomy is performed, and surgeons often even change these valves.

Addressing the commissures and reconstructing them is mandatory for the mitral leaflets to function effortlessly. Hence, keeping this idea in the background after multiple modifications, we derived our technique of **Commissural Reconstruction.**

A kite-shaped pericardial patch is harvested and prepared. Bilateral extended commissurotomy is performed. The pericardial patch is marked with points for ease of repair. The pericardial patch is sutured to the edges of the commissurotomy with continuous sutures. One or two pairs of commissural neochords are then placed along the free edge of the patch to form the support system. A limited commissurotomy is then performed to ensure laminar blood flow across the mitral valve. Other standard procedures, such as leaflet thinning, papillary muscle splitting, and chordal splitting, are also performed. The mitral annulus is always supported with an annuloplasty ring.

This technique abolishes rheumatic rigidity and makes the leaflets more flexible, allowing them to function during the cardiac cycle. It achieves excellent diastolic opening, very good systolic closure, and gives normal haemodynamics. This significantly reduces stress on leaflets, enabling a durable, functional repair.

Our article on New Technique of Commissural Reconstruction: "Ship Technique" in Rheumatic Mitral Repairs has been published in The Annals of Thoracic Surgery Journal. It is now widely appreciated in the International Community of Cardiac Surgeons.



Ship Technique of Commissural Reconstruction

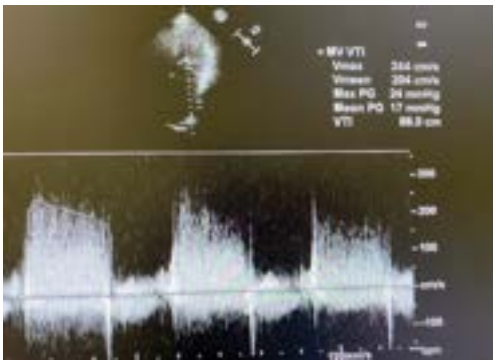
POST PROCEDURE

All the patients tolerated the procedure well and were in stable condition. They were extubated the next day. The post-operative period was smooth and uneventful.

FINAL RESULTS

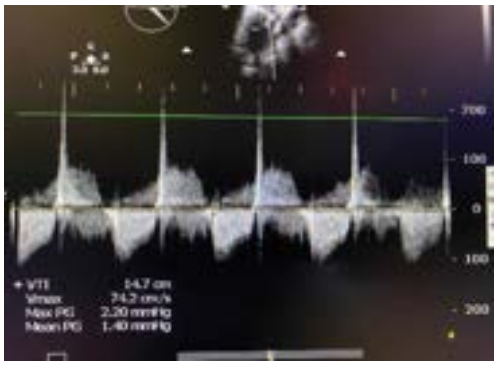
Case 1: The post-op echo revealed excellent results with a **mean mitral valve gradient of 1.4 mmHg. Normal PA pressures and no residual mitral regurgitation.** We achieved mitral valve opening distance and coaptation distance of 1.5 cm.

Pre-op Echo

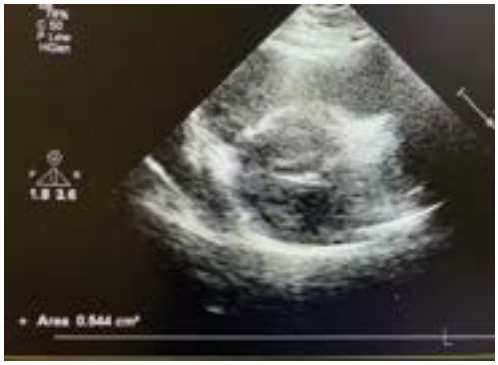


Peak Gradient of 24 mmHg

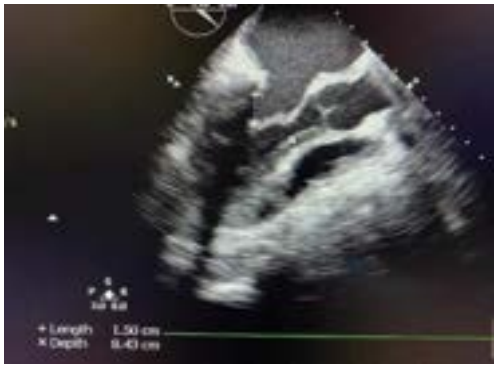
Post-op Echo



Mean Mitral Valve Gradient 1.4 mmHg



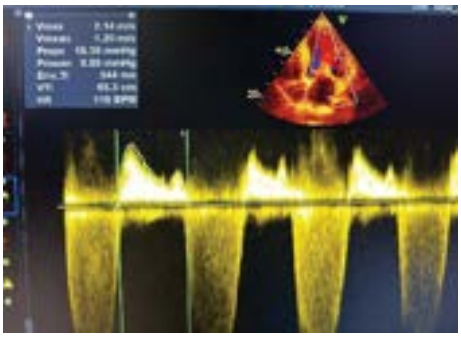
Mitral Valve Area of 0.5 cm^2



Mitral Valve opening distance 1.5 cm

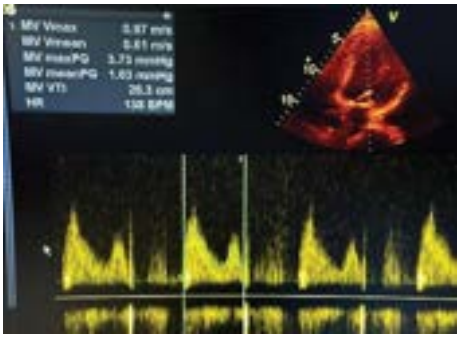
Case 2: The post-op echo revealed excellent results with a **mean mitral valve gradient of 1.6 mmHg, PA pressures of 16 mmHg, and no residual mitral regurgitation.**

Pre-op Echo

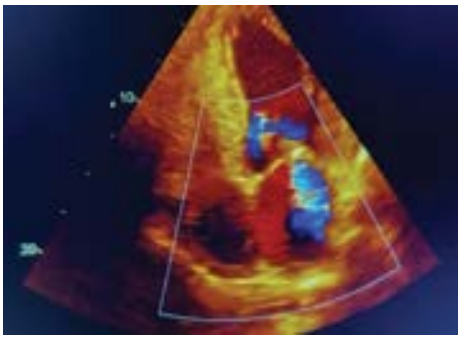


Mitral Valve Gradient 18 mmHg

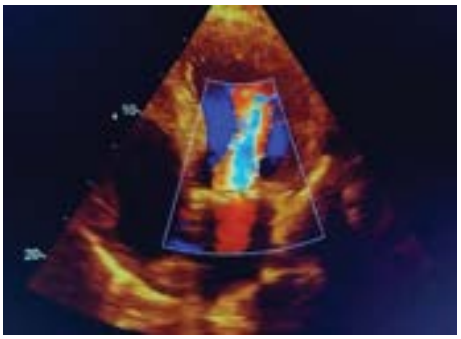
Post-op Echo



Mean Mitral Valve Gradient of 1.6 mmHg



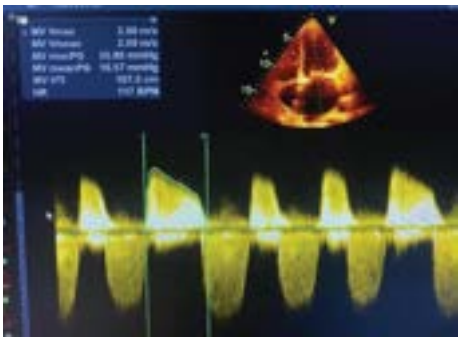
Severe Mitral Regurgitation



No residual Mitral Regurgitation

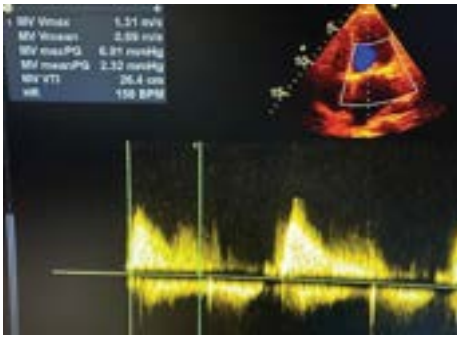
Case 3: The post-op echo revealed excellent results with a **mean mitral valve gradient of 2.3 mmHg, PA pressures of 28 mmHg, and no residual mitral regurgitation. Mitral valve area of 2.5 cm².**

Pre-op Echo:

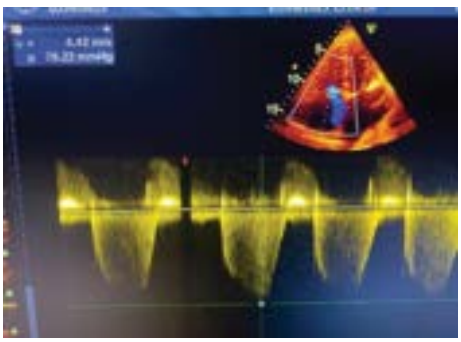


Mitral Valve gradient 36 mmHg

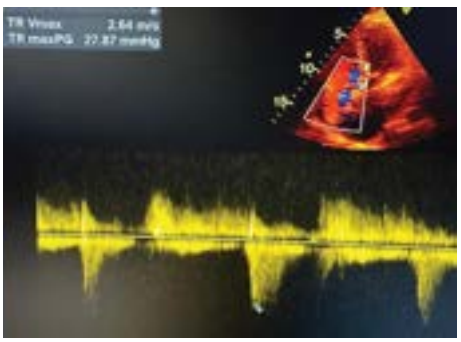
Post-op Echo:



Mean Mitral Valve Gradient 2.3 mmHg



Pulmonary Artery Pressure 90 mmHg



Pulmonary Artery Pressure 28 mmHg

DISCUSSION

Rheumatic Heart Disease contributes to a significant number of cases of acquired heart disease, mainly in the Asian population. These patients are primarily young, with a female preponderance. The presentation is aggressive, with extensive fibrosis, fusion, and even calcification of the mitral leaflets, commissures, and subvalvular structures. In these patients, a mechanical valve becomes the choice if replacement is considered. The risks of bleed and/or stroke with anticoagulation therapy are detrimental.

This is unlike the Western population, where degenerative disease takes the lead. Also, their patients are older and may be suitable candidates for tissue valves, if at all considered.

It becomes a responsibility to repair the rheumatic valves in our population. Rheumatic repairs are challenging due to the aggressive behaviour of the disease pattern. The rheumatic process continues even after repair in these young patients. Surgeons often replace these rheumatic valves due to the complexity of the disease.

In our practice, we repair these rheumatic mitral valves with special focus on the complete commissural reconstruction. The ship technique for commissural reconstruction has delivered excellent outcomes, with patients returning to NYHA-I. The post-operative echo revealed excellent haemodynamics, with the mitral valve appearing as a normal native valve.

We use this technique for isolated mitral stenosis, mixed lesions, and also for isolated mitral regurgitation.

Mitral valve repair is particularly beneficial in young women of childbearing age, as it preserves the native valve function and avoids complications of anticoagulation, thereby enabling safer pregnancy and childbirth compared with mitral valve replacement.

CONCLUSION

Rheumatic mitral valve repairs are possible only in expert hands and experienced surgeons. Our patients presented with aggressive involvement of the mitral valve, and the aortic valve was also involved in one of the patients. All these patients were advised to undergo valve replacement at other centres. We preserved their natural valves, maintaining normal physiology and performance throughout the cardiac cycle. With this, we offered our patients the freedom from anticoagulants and their troublesome side effects.

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1. Shrivastava S, et al. New Technique of Commissural Reconstruction: "Ship Technique" in Rheumatic Mitral Repairs. Ann Thorac Surg Short Rep. 2024 Mar 22;2(3):354-357. doi: 10.1016/j.atssr.2024.02.013. PMID: 39790386.
2. Dillon J, Yakub MA, et al. Leaflet extension in rheumatic mitral valve reconstruction. Eur J Cardiothorac Surg. 2013 Oct;44(4):682-9. doi: 10.1093/ejcts/ezt035. PMID: 23407161

Renal Cell Carcinoma: Contemporary Diagnosis and Management

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INTRODUCTION

Renal cell carcinoma (RCC) represents a major global health concern, ranking 14th among cancers and accounting for around 2% of all malignancies worldwide (1). In 2022, an estimated 434,840 new RCC cases and 155,953 deaths were reported globally, reflecting its considerable epidemiological burden (1). The incidence of RCC is notably higher in developed regions, where kidney cancer represents about 2% of all cancer diagnoses and deaths (2).

The rising global incidence of RCC is largely attributed to the widespread use of cross-sectional abdominal imaging, which has facilitated the incidental detection of small renal masses and led to a shift toward diagnosis at earlier, localised stages (1,3). Despite being detected early in recent times, RCC remains the most lethal urological malignancy, characterised by marked biological heterogeneity and complex clinical behaviour (4). Clear cell RCC, the predominant histological subtype, accounts for the majority of kidney cancer–related deaths (2).

RCC continues to impose a substantial clinical burden, with up to 30% patients with localised tumours eventually developing metastatic disease (2). Addressing this complexity requires a multidisciplinary approach integrating expertise from urology, oncology, radiology, and pathology to optimise diagnosis and treatment across the disease continuum (2).

This article examines the biology of RCC and outlines diagnostic pathways, surgical and systemic treatment approaches, and future-focused models of personalised care. Each section builds on international guideline recommendations and current evidence to support optimal multidisciplinary practice.

RCC BIOLOGY AND CLINICAL SPECTRUM

RCC represents a biologically diverse group of epithelial tumours that arise from renal tubular cells, accounting for about 90% of all kidney cancers (2). This heterogeneity directly affects diagnostic accuracy, surgical planning, and systemic therapy choices across the disease spectrum (2).

RCC comprises three predominant histologic subtypes that differ in cellular origin, molecular alterations, and expected behaviour (1,2). Beyond histologic classification, RCC displays substantial molecular and intratumoral heterogeneity, which influences recurrence risk and response to therapy (1).

A. **Clear cell RCC (ccRCC)** is derived from the proximal tubule and is driven by VHL (von Hippel–Lindau) loss and 3p deletion.

B. **Papillary RCC (pRCC)** arises from the proximal tubular epithelium and includes Type 1 (mesenchymal-epithelial transition factor-driven) and Type 2 (FH-mutated) variants.

C. **Chromophobe RCC (chRCC)** originates from the distal nephron's intercalated cells, generally following an indolent course (1,2).

Figure 1 illustrates the key histologic, anatomic, and clinical distinctions between these subtypes, relevant for surgical planning.

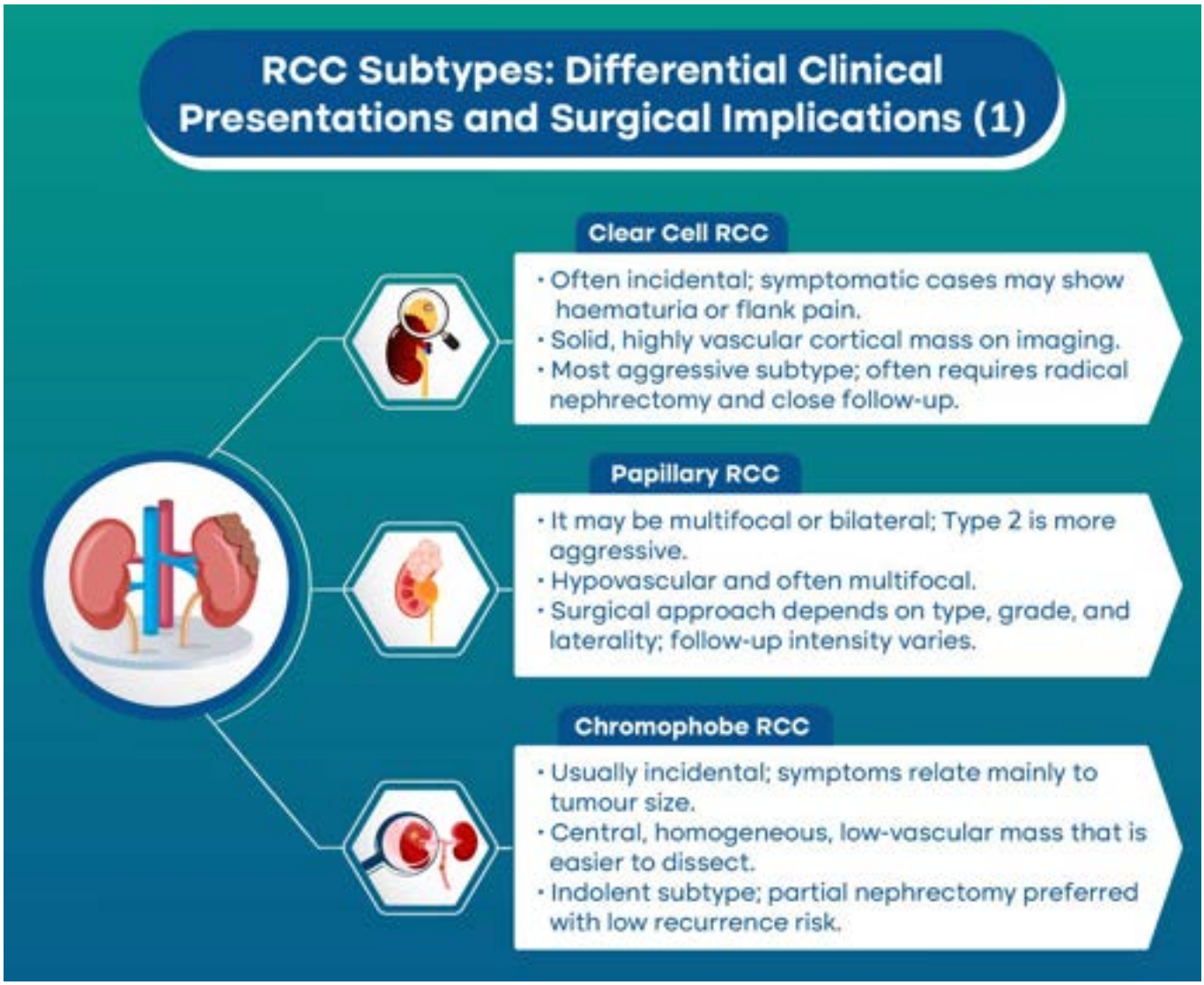

Figure 1. Overview of major RCC histological subtypes (1)

(Abbreviations: RCC – renal cell carcinoma)

Another impact of the incidental diagnosis of RCC through imaging has been noted in the clinical presentation, which traditionally followed the classic triad of flank pain, haematuria, and a palpable mass, now reported in fewer than 10% of patients (3). An important outcome of this has been the earlier-stage detection and improved surgical outcomes (3). Up to 20% of patients develop paraneoplastic syndromes, such as hypercalcaemia, polycythaemia, hepatic (Stauffer) syndrome, or hypertension, which can complicate evaluation but seem to resolve once the tumour is removed (2,5).

Factors such as age, gender, obesity, smoking habits, hypertension, and chronic kidney disease (CKD) tend to be associated with RCC incidence and impact postoperative renal outcomes (3,5). Around 3% of cases are hereditary, transmitted through autosomal-dominant VHL, Birt–Hogg–Dubé syndrome (BHD), and hereditary leiomyomatosis, each defined by unique germline mutations and characteristic histologic patterns (5). Identifying these syndromes supports early genetic counselling, active surveillance, and nephron-sparing intervention where feasible (1).

APPROACH TO DIAGNOSIS AND STAGING OF RCC

Accurate evaluation of RCC relies on a structured diagnostic approach, with cross-sectional imaging being the cornerstone for diagnosis, staging, and therapeutic planning (1). The diagnostic assessment proceeds through the following key steps:

Step 1: Initial imaging and clinical assessment

Most RCCs are detected incidentally during abdominal imaging performed for other indications, reflecting earlier detection and stage migration (6). Contrast-enhanced computed tomography (CT) of the chest, abdomen, and pelvis is the first-line modality for both diagnosis and staging, offering high sensitivity and specificity for evaluating renal masses, venous invasion, and metastatic spread (1,6,7). CT demonstrates staging accuracy between 72–90%, confirming its role as the diagnostic standard across all disease stages (8). For advanced presentations, neuroimaging with CT or magnetic resonance imaging (MRI) is recommended, and bone scans may be indicated before systemic therapy, whereas positron emission tomography (PET) is not routinely recommended (6).

MRI provides superior soft-tissue contrast and is particularly useful for evaluating small cystic renal lesions and the extent of inferior vena cava tumour thrombus, with negative predictive values up to 98–99% for venous invasion (8). It complements CT by refining preoperative planning and guiding nephron-sparing approaches (7).

Step 2: Histopathological confirmation and biopsy evaluation

Histopathological confirmation is mandatory before initiating systemic therapy, ensuring diagnostic accuracy and enabling subtype-directed management (6). Core needle biopsy remains the preferred method for obtaining tissue from either the primary tumour or metastatic sites, offering a sensitivity of 99.1% and a specificity of 99.7%, with minimal seeding risk (8).

Indications for biopsy include (1):

- Radiologically indeterminate renal masses where malignancy cannot be confirmed by imaging alone.
- Pre-systemic therapy assessment in advanced or metastatic RCC.
- High surgical risk due to comorbidities or limited renal reserve.

Although core biopsy offers high diagnostic accuracy, it may be limited by sampling error or non-diagnostic results in up to 22% of cases, occasionally requiring repeat procedures at an expert centre (8). Once adequate tissue is obtained, histopathological evaluation should determine the RCC subtype and assess for sarcomatoid or rhabdoid differentiation, as these features hold significant prognostic and therapeutic importance (6).

Step 3: Staging and risk stratification

RCC staging follows the 8th edition of the Union for International Cancer Control (UICC) TNM classification, which describes tumour size and extent (T), regional lymph node involvement (N), and the presence or absence of distant metastasis (M) (1,6). For operable RCC, risk stratification is guided by **the KEYNOTE-564 trial model,** which defines adjuvant therapy eligibility as follows (6):

- **Intermediate–high risk:** Pathological stage T2 with grade 4 or sarcomatoid features, node-negative and non-metastatic (N0M0), or T3 any grade, N0M0.
- **High risk: T4 any grade,** N0M0, or any T, any grade with lymph-node involvement and no distant metastasis (N+, M0).

For advanced RCC, the International Metastatic RCC Database Consortium (IMDC) score remains the

preferred model for outcome prediction, classifying patients into favourable, intermediate, or poor risk categories to guide systemic therapy decisions (6).

Step 4: Laboratory and functional evaluation

Baseline laboratory evaluation complements imaging and biopsy by assessing renal function and systemic status (1). Key investigations include serum creatinine, estimated glomerular filtration rate (eGFR), complete blood count (CBC), and C-reactive protein (CRP), which together inform prognostic models such as the IMDC score (6). In selected cases, urinary cytology provides additional diagnostic support, particularly when tumours are close to the collecting system (8).

Step 5: Differential diagnosis and multidisciplinary review

Distinguishing RCC from benign or mimicking renal lesions is essential for accurate management planning (1). Common conditions with similar radiologic presentations of RCC include oncocytoma, angiomyolipoma, and hybrid oncocytic tumours (7).

Certain imaging patterns, such as multifocal papillary tumours or fumarate-hydratase-deficient lesions, can suggest specific subtypes but remain non-definitive without histopathological confirmation (6). Therefore, integrating clinical, radiological, and pathological findings remains central to ensuring diagnostic precision and guiding multidisciplinary treatment decisions (1).

MANAGEMENT OF LOCALISED AND ADVANCED DISEASE

Management of RCC requires a structured, stage-adapted framework that balances oncological control with renal function preservation (1).

In **localised RCC,** nephron-sparing surgery (NSS) remains the cornerstone of management, as it preserves renal parenchyma and significantly reduces the long-term risk of CKD and associated cardiovascular events (8). Among NSS techniques, partial nephrectomy is the preferred approach for T1 tumours and may also be extended to selected T2 lesions, offering oncological outcomes comparable to radical nephrectomy when performed in appropriately chosen cases (9). Conversely, radical nephrectomy remains indicated when tumour size, anatomic complexity, or involvement of adjacent structures preclude a nephron-sparing option (8).

For patients unsuitable for surgery or those presenting with small renal masses, minimally invasive ablative techniques, such as cryoablation and radiofrequency ablation (RFA), provide viable alternatives with satisfactory local control in properly selected candidates (9). In older or medically frail patients, active surveillance may also be appropriate, recognising that small renal tumours often demonstrate slow growth and low metastatic potential (10).

In **locally advanced RCC**, management complexity increases, requiring detailed anatomical evaluation and multidisciplinary surgical planning (1). Preoperative cross-sectional imaging is essential to define the extent of venous tumour thrombus, which directly determines the surgical approach and the need for vascular or cardiothoracic assistance (6). While routine lymphadenectomy is not recommended, regional lymph-node dissection is considered when radiological or intraoperative findings indicate nodal involvement (11). Optimal surgical outcomes depend on close coordination among urologic oncologists, vascular surgeons, anaesthetists, and perioperative specialists, ensuring oncological efficacy while minimising operative risk (1).

Perioperative management focuses on maintaining renal function and tailoring surgical strategy to individual physiological risk profiles (8). Special attention is required for patients with a solitary kidney or pre-existing CKD, where nephron-sparing techniques are prioritised to prevent postoperative renal

insufficiency (10).

Figure 2 summarises these key perioperative considerations in RCC surgery, highlighting renal preservation priorities across different patient profiles.

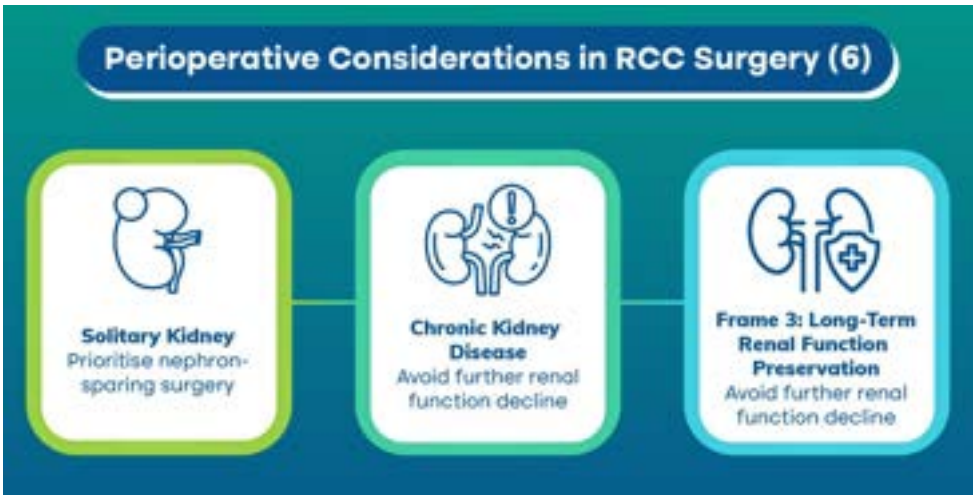

Figure 2. Evidence-based perioperative considerations in RCC surgery (6)

(Abbreviations: RCC – renal cell carcinoma)

EVOLVING LANDSCAPE OF SYSTEMIC AND MULTIMODAL THERAPIES

Systemic therapy has fundamentally reshaped the management of advanced RCC, driven by the success of immune checkpoint inhibitors (IO) that have significantly improved overall survival and response durability compared with tyrosine kinase inhibitor (TKI) monotherapy (12). Their integration into first-line care has expanded therapeutic possibilities, combining immune-mediated mechanisms with targeted anti-angiogenic activity (12).

Figure 3 illustrates the evolving systemic and multimodal treatment framework in advanced RCC, highlighting how modern therapy approaches align across complementary mechanisms and clinical settings.

Figure 3. Evidence-based systemic and multimodal therapy framework in advanced RCC (6)

(Abbreviations: IO – immuno-oncology; TKI – tyrosine kinase inhibitor; VHL – Von Hippel–Lindau)

Checkpoint inhibitor combinations form the cornerstone of systemic management, producing durable immune-mediated responses and long-term survival benefits across prognostic categories (6). For patients who cannot receive immunotherapy, **TKI monotherapy** remains an established option, offering reliable disease control with a manageable toxicity profile (6). As the disease progresses, treatment sequencing is guided by prior mechanisms of action, emerging resistance, and individual tolerance, ensuring that therapeutic benefit is sustained over time (1).

The role of **cytoreductive nephrectomy** (CN) continues to evolve within this multimodal framework (13). Evidence supports a selective or deferred approach, particularly for patients with good performance status or limited metastatic burden who have responded to systemic therapy (13,14). Similarly, in oligometastatic disease, integrating systemic therapy with stereotactic body radiotherapy (SBRT) or metastasectomy can achieve durable local control and extend progression-free survival in well-selected cases (15,16).

Looking ahead, HIF-2α inhibitors such as belzutifan are redefining therapeutic strategies by targeting hypoxia-driven molecular pathways central to RCC progression, particularly in VHL-associated disease (16). In parallel, next-generation **immunotherapy combinations** aim to enhance response durability and overcome treatment resistance, reflecting the field's ongoing shift toward precision-driven, personalised care (6).

LONG-TERM MANAGEMENT AND FUTURE PERSPECTIVES IN RCC

Long-term follow-up in RCC focuses on detecting recurrence early, maintaining renal function, and supporting survivorship through coordinated multidisciplinary care (1). Follow-up should be tailored according to recurrence risk, stage, and patient comorbidity to balance clinical benefit with imaging burden (6).

Figure 4, adapted from the European Association of Urology (EAU) Guidelines 2025, outlines the recommended surveillance schedule for low-, intermediate-, and high-risk patients, integrating early, intermediate, and long-term follow-up intervals.

Postoperative Surveillance Schedule Based on Recurrence Risk in RCC (1)

Risk Category	Early Imaging (0–12 months)	Intermediate Surveillance (12–36 months)	Long-Term Surveillance (>36 months)	Clinical Focus
Low Risk	CT at 6 months	CT at 18–30 months	CT every 2 years	• Minimise unnecessary imaging • Detect rare late recurrences • Monitor baseline renal status
Intermediate Risk	CT at 6 and 12 months	CT at 18 and 30 months	Annual CT → biennial after 5 years	• Balance recurrence detection with imaging burden • Assess renal function trajectory • Identify early metastatic progression
High Risk	CT at 3, 6, and 12 months	CT at 18, 24, and 30 months	Annual CT → biennial after 5 years	• High vigilance for early relapse • Monitor for distant metastasis • Guide timely systemic therapy decisions

Figure 4. Risk-adapted surveillance schedule after RCC treatment (1)

(Abbreviations: RCC- renal cell carcinoma; CT- computed tomography)

Risk-adapted surveillance remains the foundation of postoperative management (1). Low-risk patients typically require limited imaging to avoid over-surveillance, while intermediate- and high-risk groups benefit from more frequent cross-sectional imaging within the first three years, when recurrence risk peaks (1). Prognostic models like the University of California Integrated Staging System (UISS), SSIGN (Stage, Size, Grade, and Necrosis), Leibovich, and IMDC help personalise follow-up by integrating clinical, pathological, and biochemical variables to guide intensity and timing (5,6,17). Early-year surveillance is particularly critical, as predictive accuracy declines beyond the initial postoperative phase (17).

Patients with a solitary kidney or pre-existing CKD are particularly vulnerable to renal decline after nephrectomy (1). Regular evaluation of serum creatinine, eGFR, and related parameters enables early detection of dysfunction, while nephrology collaboration supports long-term preservation of renal function (1).

Advances in molecular and computational diagnostics are redefining follow-up paradigms (6).

Liquid biopsy, encompassing circulating tumour DNA, circulating tumour cells, and exosomal biomarkers, offers a non-invasive means to detect recurrence and assess therapeutic response (18). In parallel, artificial intelligence (AI) and radiomics are being integrated with molecular data to improve prognostic accuracy and enable real-time risk modelling (18). These technologies represent the transition toward precision-based RCC surveillance and dynamic treatment adjustment (1).

Systemic therapy advances continue to influence long-term outcomes. IOs remain essential for metastatic disease management, while HIF-2α inhibitors such as belzutifan are emerging as targeted options in VHL-associated RCC (15). Ongoing adjuvant and neoadjuvant trials are exploring strategies to reduce recurrence and extend disease-free survival (1). A multidisciplinary approach bridging urology, oncology, nephrology, and radiology ensures comprehensive, stage-adapted management across the RCC continuum (6).

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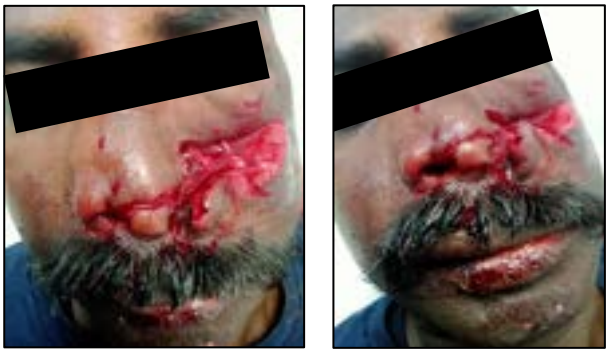
Successful Complex Reconstruction of Severe Panfacial Trauma with Open Reduction and Internal Fixation (ORIF) at Aster Hospital, Mankhool, Dubai

PRESENTATION

- 42-year-old male
- History of accidental self-fall due to an episode of syncope on a rotating saw-blade machine, collapsed after trauma.
- Admitted to ER with:
  - Bleeding from multiple deep lacerated wounds involving the face, nose, and eye regions measuring around 15x7 cm
  - Pain and swelling on the left side of the face region associated with severe bleeding
  - Double vision, facial nerve weakness in the left side
  - Nasal bleeding, complex nasal bridge collapse with extensive loss of soft tissue
  - Difficulty in breathing and mouth opening
  - Multiple fractures involving the maxillofacial regions



Pre-op Images

Primary wound closure in view of extensive bleeding and comminuted soft tissue injury in the nasal septal region

FINDINGS

Extra-oral examination:

- Multiple deep lacerated wounds
- Pain: 4/10
- **Diplopia: Left eye**
- **Facial nerve palsy: Left side**
- Nasal septal oedema
- Septal bridge collapse with extensive loss of cartilage
- Step deformity: Mandibular region
- Bilateral occlusion: Deranged
- Trismus
- Sub-conjunctival haemorrhage: Bilateral

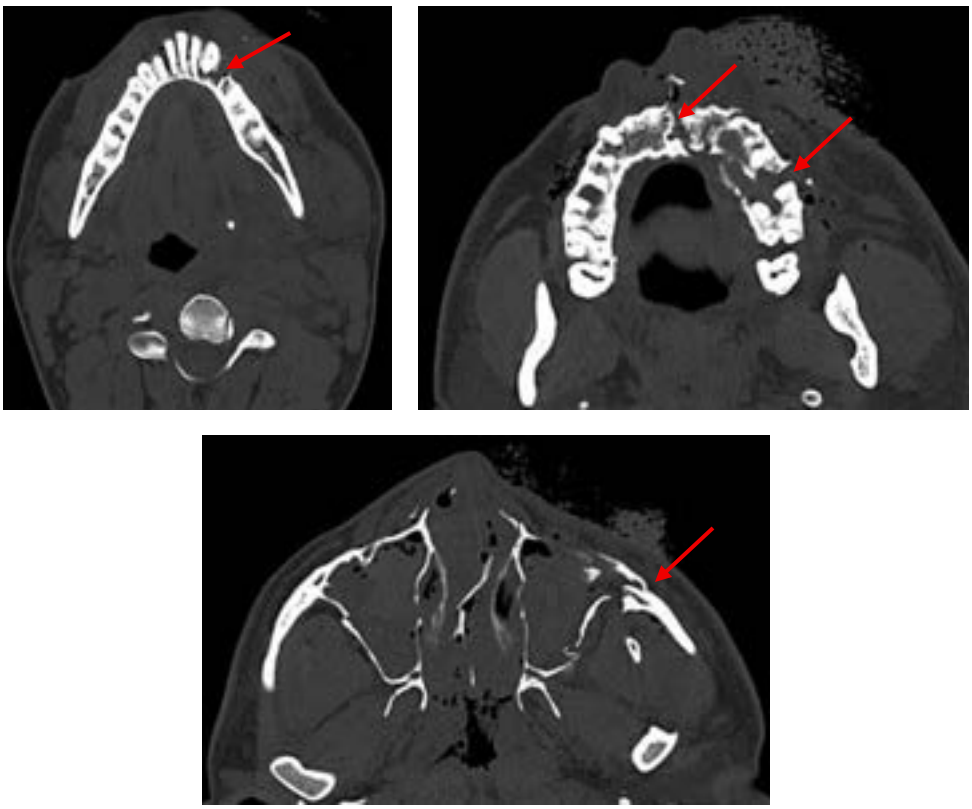
Intra-oral examination:

- Multiple avulsed maxillary and mandibular teeth
- Step-deformity mandibular para-symphysis region
- High Le-Fort Type II fracture with complete disjunction of the maxillary complex
- Trismus
- Bilateral occlusion: Deranged
- Midline palatal split

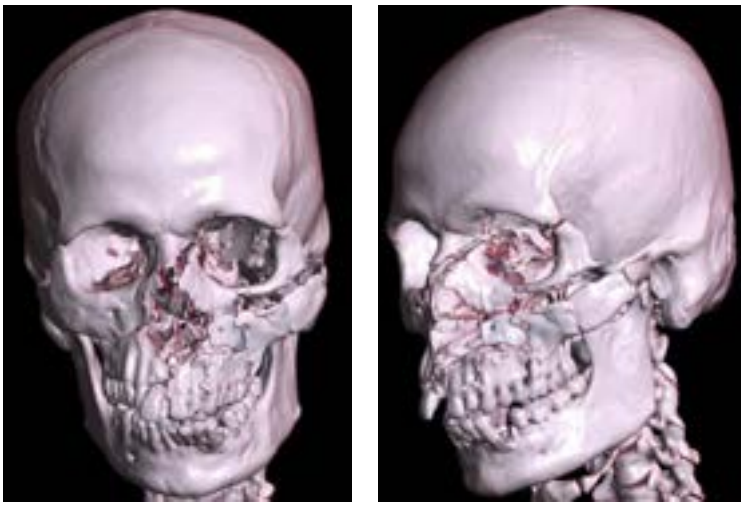
RADIOLOGICAL EXAMINATION FINDINGS

CT Face and Brain Impressions:

- Extensive fracture involving the left zygomatic arch
- Maxillary Sinus Walls: Bilateral fractures, with greater comminution on the left side
- Nasal Septum: Comminuted fracture of the midline bony nasal septum
- Inferior Turbinates: Bilateral fractures
- Orbits: Left orbit: Fractures of the inferior and medial walls; Right orbit: Fracture of the medial wall
- Alveolar process of maxilla: Comminuted three-part fracture with displacement of the right lateral incisor
- Fracture involving the right lateral pterygoid process
- Hard palate: Displaced comminuted fractures of the bilateral palatine bone and the palatine process of the maxillary bone
- Fracture of the buccal surface of the left hemimandible, with fracture line extending to the left lateral incisor



CT images showing fractures in the maxillofacial Skeleton

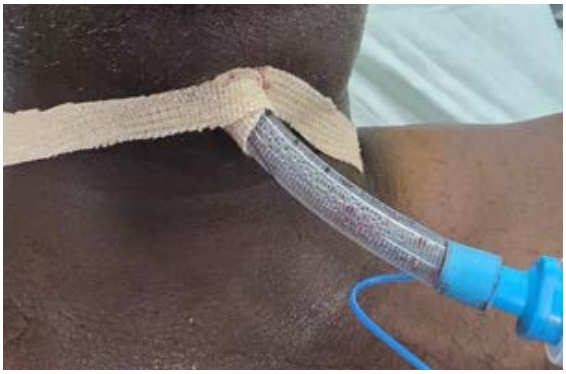


3D images showing severe panfacial fractures

DIAGNOSIS

- Bilateral Le Fort II fractures
- Bilateral zygomatico maxillary complex + orbital floor fractures
- Lateral orbital fracture
- Comminuted nasal bone fracture
- Comminuted zygomatic arch fracture
- Mandibular fracture

PROCEDURE



Submental intubation preferred in view of extensive soft/hard tissue injuries, making nasal/oral intubation difficult

The patient underwent Open Reduction and Internal Fixation (ORIF) and Reconstruction of Bilateral Orbital Floor under general anaesthesia:

Intraoral Approach:

- The eyelet wiring was done, occlusion restored bilaterally, and interdental fixation was done along the left posterior maxillary region.
- Multiple avulsed segments of bone with teeth were seen detached, and the extraction of avulsed teeth was done along the anterior maxillary/mandibular quadrants.
- A circumvestibular incision was given in the left maxillary mucobuccal fold region, and the periosteum was reflected. Avulsed segments of bone were seen devoid of periosteal blood supply, and the segments were removed. The maxillary hemosinus was evacuated, the fracture was reduced, and fixation was performed with titanium miniplates and screws.
- Multiple avulsed segments of bone with loss of blood supply were seen along the left posterior maxillary/buttress regions; the fracture was reduced, and fixation was done.
- Nerve detachment was seen along the infraorbital foramen, and the nerve was repositioned.
- Periosteum was tunnelled along the left zygomaticomaxillary region.
- Fractures were reduced, stabilised, and fixation was done with titanium mini plates + screws along the zygomatic arch.
- A circumvestibular incision was given in the right maxillary mucobuccal fold region.
- Periosteum was reflected, fracture reduced, and fixation was done with titanium mini plates and screws along the posterior maxillary buttress region.

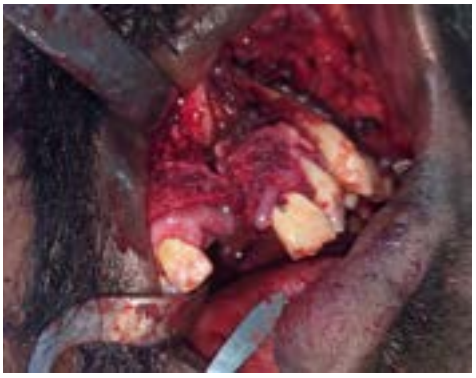
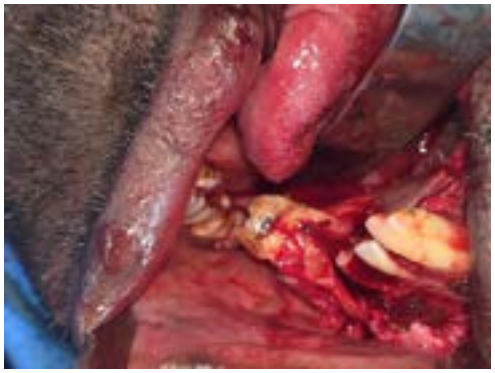
Extraoral – Subciliary Approach:

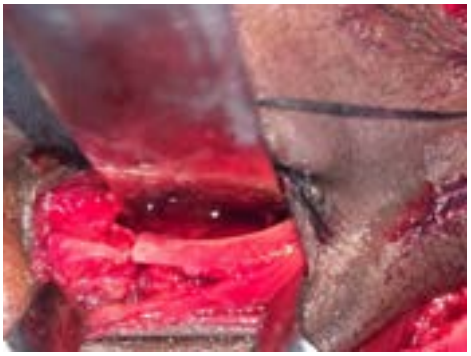
- An incision was given in the left subciliary region, 2-3 mm inferior and parallel to the lower eyelid skin crease region.
- Blunt dissection was done through the skin, subcutaneous tissues, orbicularis oculi muscle, gaining access to the infraorbital rim.
- The orbital fascia was detached along the floor, and multiple avulsed segments of bone were seen along the orbital floor.
- Segments were removed, and nerve compression was released.

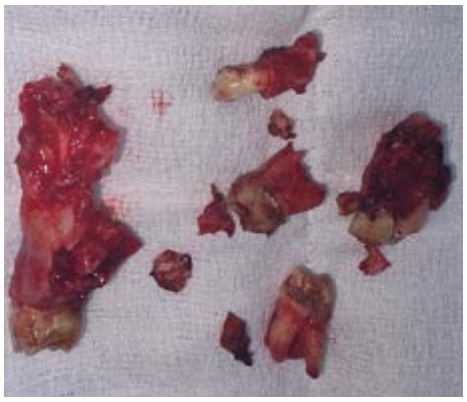
- Orbital floor reconstruction was done with orbital mesh and screws.
- The same procedure was repeated on the right side of the orbital region.
- Lateral orbital incision/FZ region: incision was given in the left FZ region.
- Periosteum was reflected, the fracture was reduced, and fixation was done.
- Nasal region: Bilateral nasal bones were reduced and stabilised in position; the fracture was reduced, and fixation was performed with titanium miniplates and screws.
- An extensive loss of nasal cartilage was seen, and intranasal packing was done.
- The wound cavity was cleaned thoroughly, haemostasis secured, and a mini-vac drain placement was done in the left zygomaticomaxillary region.
- Wound closure was done in multiple layers with 3-0 Vicryl, and skin closure was done with 5-0 /6-0 Ethilon.

Post-operatively, the patient was shifted to the ICU for further monitoring.

Intra-operative images



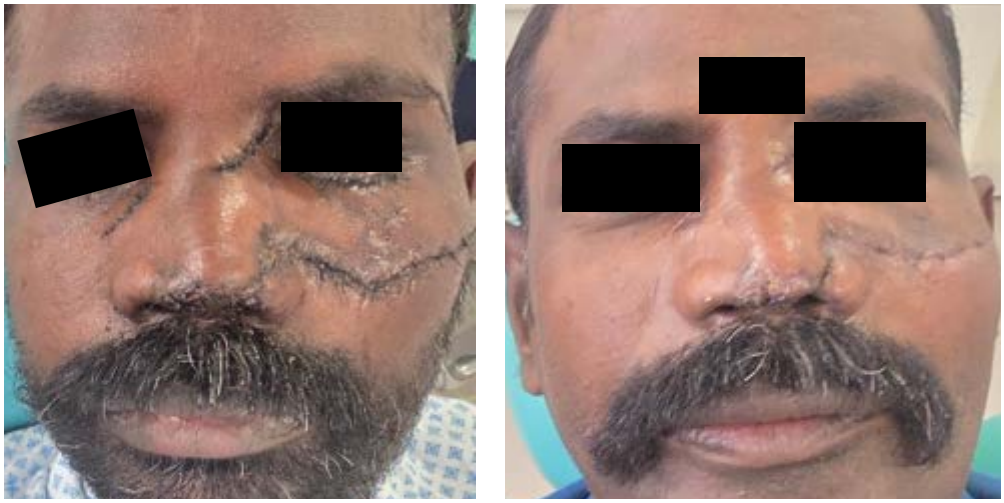


Extensive loss of bones/teeth along the left posterior maxillary region

POST PROCEDURE

The patient withstood the procedure well; post-anaesthesia recovery was uneventful. He was monitored in the ICU, extubated, and his condition was optimised with Cardiology and Nephrology evaluations. Post-operatively, the patient was shifted to the ward and started on RT feeds. Periodic wound dressings were done on a daily basis.

Later, the patient started oral fluids and a semi-solid diet and ambulated. He was discharged in asymptomatic stable condition.



Immediate post-op and post-op after 3 weeks

DISCUSSION

A Panfacial trauma is defined as concomitant fractures in the upper, middle, and lower thirds of the face. These fractures are often secondary to high-speed blunt, penetrating, or avulsive mechanisms of injury.

Consequently, these patients often present with multisystem trauma, necessitating complex multidisciplinary care. Concomitant life-threatening injuries may affect surgical timing and further complicate the management of these facial injuries.

Panfacial fracture management aims to restore the patient's face and function as close to their premorbid condition as possible, and to avoid any long-term sequelae caused by suboptimal correction.

Panfacial fractures pose a technical challenge to many facial surgeons due to the extensive nature of the injuries, significant comminution of the segments and loss of anatomical references for reduction. The sequence of fracture repair is thereby considered critical.

1. Anatomical considerations

Knowledge of facial buttresses is paramount to optimal treatment of panfacial trauma.

There are three paired vertical buttresses:

- Nasomaxillary
- Zygomaticomaxillary
- Pterygomaxillary

Three horizontal buttresses:

- Alveolar process of maxilla
- Inferior orbital rim/zygomatic arches
- Superior orbital rim/glabella

2. Airway Management

Once the patient is adequately resuscitated and deemed stable enough to undergo operative intervention, a decision must be made on the appropriate airway management for the patient.

Oral intubation is generally not feasible, and nasal intubation also carries the risk of intracranial intubation if the patient has significant fractures of the skull base.

A submental pull-through is a useful airway adjunct in these cases.

3. Exposure of all fractures

4. Direction based approaches to Sequencing

- **Top-Down Approach (Gruss) -** uses orbital bar as the initial starting point for establishing upper facial width. This is based on the notion that the frontal-orbital bandeau is comprised of thick, dense bone that is not prone to gross comminution.
- **Bottom-Up Approach (Manson)** - uses the mandible instead of the frontal-orbital bandeau as the foundation for the reduction sequence.

This is often considered the standard choice of many Oral and Maxillofacial surgeons.

Once the mandible has been fixated, the resulting mandibular occlusion can serve as a reference for the maxilla, and the patient is placed in MMF. The approach then proceeds to the upper face and zygomas.

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Bladder Cancer Management: From Early Detection to Advanced Disease

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INTRODUCTION

Bladder cancer (BCa) remains a major global health concern, accounting for around 573,000 newly diagnosed cases and about 213,000 deaths in 2020, with men exhibiting nearly four times greater incidence and mortality rates compared to women (1). The disease predominantly affects older adults, as tumours of the bladder rarely occur before the age of 40–50 and are most common in the seventh decade of life, reflecting its strong age-related pattern (2). International epidemiological data also highlight considerable geographic variation, with higher incidence observed in regions where environmental and lifestyle exposures are more prevalent (1).

Cigarette smoking continues to be the most influential risk factor, as current smokers have an approximately threefold higher risk and smoking accounts for 50% of BCa in men (2). Occupational exposure to aromatic amines, including 2-naphthylamine and benzidine, remains another established contributor to disease risk (1). In addition, chronic inflammatory conditions are implicated, since chronic bladder inflammation may arise from infections such as schistosomiasis or from irritants like indwelling catheters (3).

Although less common, hereditary susceptibility is recognised (4). Evidence indicates that carriers of germline MutS Homologue 2 (MSH2) mutations and first-degree relatives within Lynch syndrome families have a higher risk of BCa (4). Preventive strategies increasingly reflect these risk patterns, with smoking cessation, workplace safety measures, and schistosomiasis prevention shown to decrease risk significantly (5). Furthermore, the need for sustained clinical vigilance is clear, as BCa often requires continued evaluation, treatment and surveillance (6).

This article provides a comprehensive overview of BCa, beginning with its molecular foundations and clinical behaviour before examining diagnostic pathways and contemporary management strategies. It further explores systemic treatment advances across localised, advanced and metastatic disease. Finally, it highlights future directions in personalised therapy and technology-enabled care, offering a cohesive framework for modern clinical decision-making.

MOLECULAR PATHOGENESIS AND CLINICAL SPECTRUM

Urothelial carcinoma (UC) represents the predominant histologic subtype of BCa, whereas squamous cell carcinoma accounts for 1–6% and adenocarcinoma <2% of cases (7). Several recognised histologic variants, including micropapillary, plasmacytoid and sarcomatoid UC, are associated with aggressive biological behaviour and adverse outcomes (7,8). To contextualise these patterns, the classification of BCa is illustrated in **Figure 1.**

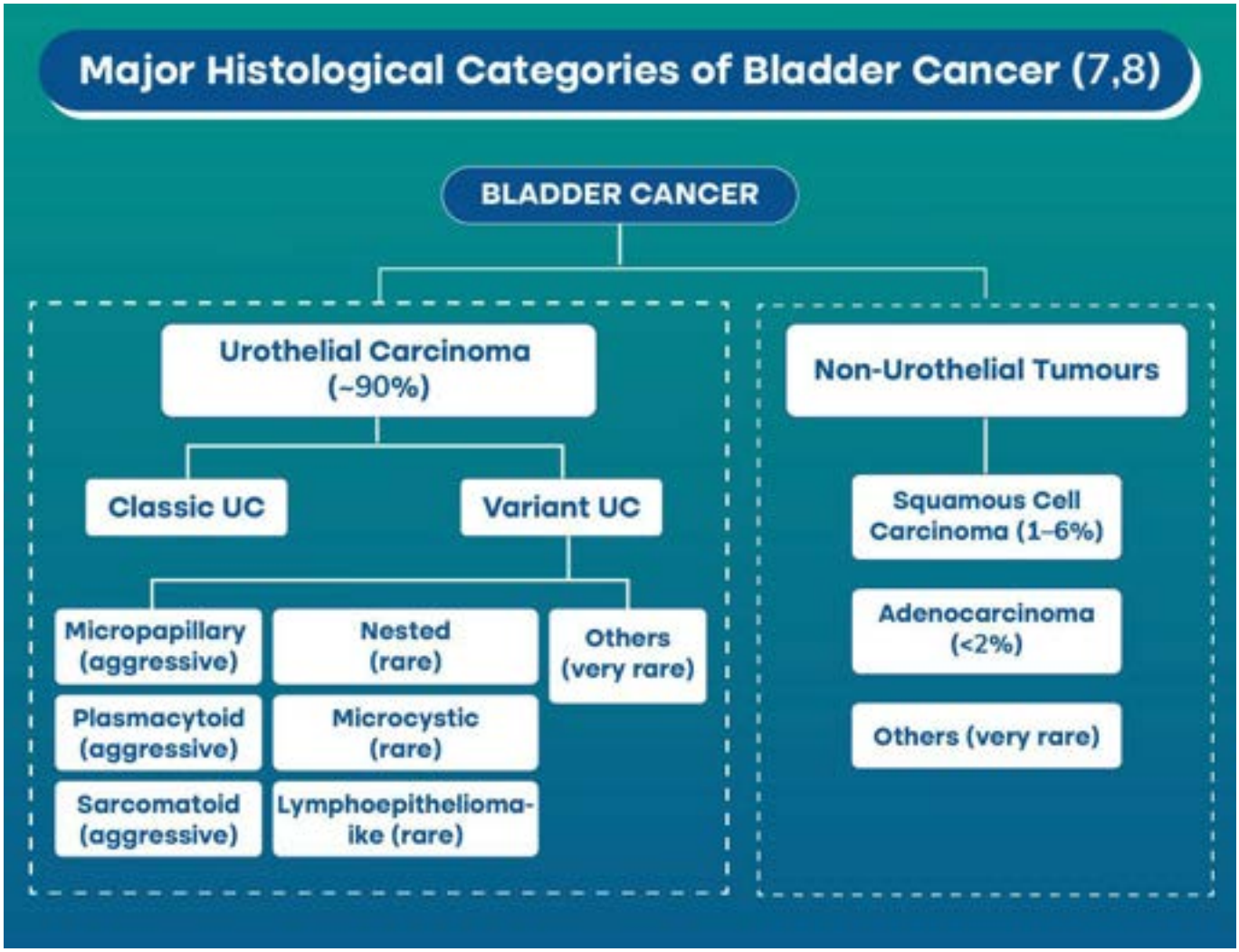


Figure1. Major histological categories of Bca (7,8)

(Abbreviations- UC: urothelial carcinoma)

These histologic distinctions reflect deeper molecular routes that govern tumour development (9). UC evolves through two principal genomic pathways:

1. **Fibroblast growth factor receptor 3 (FGFR3)** mutations are frequently identified in non-invasive, low-grade papillary tumours.
2. **Protein p53 (TP53) and Retinoblastoma gene 1 (RB1) alterations**, which are characteristic of high-grade, muscle-invasive UC (9).

These molecular alterations define two recognised pathways of urothelial tumorigenesis, with one pathway leading to papillary non-invasive disease and the other associated with carcinoma in situ and subsequent invasive progression (7,9).

Figure 2 illustrates these pathways according to their initiating genomic changes.

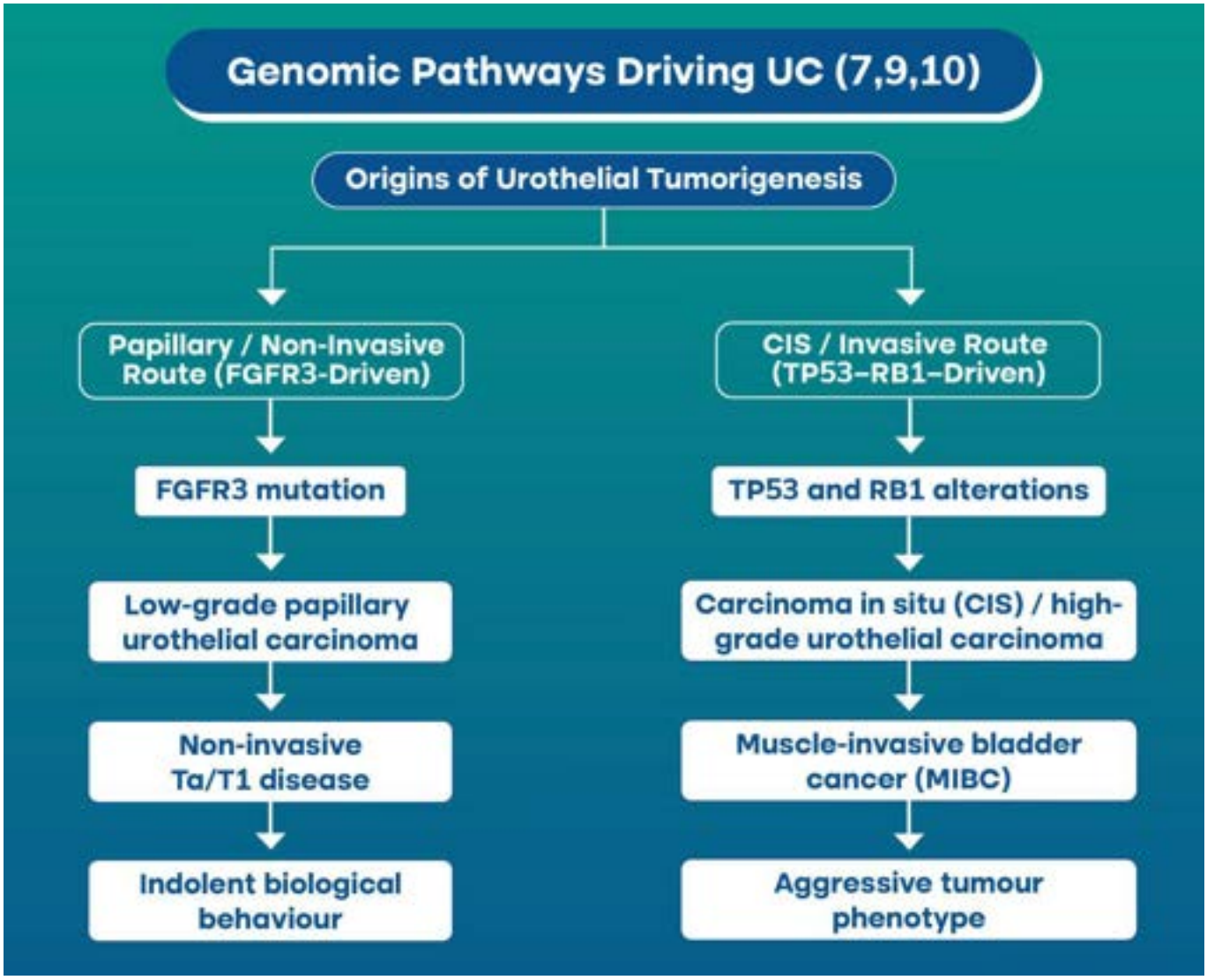


Figure 2. Dual molecular pathways of urothelial tumour evolution (7,9,10)

(Abbreviations – FGFR3: fibroblast growth factor receptor 3; CIS: carcinoma in situ; TP53: tumour protein 53; RB1: retinoblastoma 1; Ta/T1: non-muscle-invasive tumour stages)

These genomic pathways underpin the clinical presentation of UC (9). Haematuria (macroscopic or microscopic) represents the most frequent initial symptom observed in non-muscle-invasive bladder cancer (NMIBC) (11). When the disease presents as carcinoma in situ (CIS), patients may develop irritative voiding symptoms such as urgency and frequency (11). As the disease advances, symptoms may include pain, weight loss, fatigue and a palpable mass, while flank pain may indicate upper urinary tract obstruction (12). Although rare, systemic neurological complications may occur, and reported examples include opsoclonus-ataxia, cerebellar degeneration, Lambert–Eaton-type myasthenia gravis and polymyositis in BCa patients (13).

DIAGNOSTIC PARADIGMS AND RISK-ADAPTED STRATIFICATION

The diagnosis of BCa incorporates multiple coordinated assessments, beginning with endoscopic evaluation and supported by adjunct tests, imaging, and histopathology, which together inform accurate staging and risk categorisation (14). Each diagnostic component provides complementary information that collectively defines tumour extent, informs tumour behaviour and supports personalised management (14).

Current clinical guidelines describe the diagnostic workup as progressing from cystoscopy to urine-based tests, then to cross-sectional imaging and histological confirmation, with these findings subsequently integrated into established NMIBC risk stratification systems (14–18).

1. Endoscopic assessment

Cystoscopy is identified in major international guidelines, including the European Association of Urology (EAU) Guidelines and the National Institute for Health and Care Excellence (NICE) Guideline on Bladder Cancer, as the essential initial investigation in the evaluation of suspected disease, forming the foundation of diagnostic assessment (11,12,17).

- **White-light cystoscopy (WLC):** Standard initial investigation in patients suspected of BCa (18). Allows direct visualisation of intravesical lesions and supports diagnostic planning (18).
- **Blue-light cystoscopy (BLC):** Enhances detection of carcinoma in situ (CIS) and subtle lesions that may be missed on WLC (14). Provides complementary optical information that improves diagnostic accuracy (14).

2. Adjunct urinary tests

Urine-based tests are recommended as complementary tools, particularly for detecting high-grade disease and CIS when cystoscopic findings are inconclusive (14). Urine cytology adds important specificity for identifying high-grade disease and CIS (14). It is especially valuable when cystoscopic visualisation is limited or when high-grade pathology is suspected (16). When interpreted alongside cystoscopy, urine cytology strengthens overall diagnostic certainty (14).

3. Cross-sectional imaging

Cross-sectional imaging is performed following endoscopic and cytological assessment to evaluate local tumour extension, upper urinary tract involvement and, when indicated, the depth of invasion (14,16,17).

Computed tomography (CT) urography serves as the primary modality in this stage of evaluation (17). It provides a comprehensive assessment of upper urinary tract involvement while also defining the extent of local tumour spread, making it central to radiological staging (17). In situations where more detailed characterisation of bladder wall invasion is required, multiparametric magnetic resonance imaging (mpMRI) provides enhanced soft-tissue definition and can accurately differentiate NMIBC from muscle-invasive bladder cancer (MIBC) (19).

Interpretation of mpMRI findings is guided by the Vesical Imaging–Reporting and Data System (VI-RADS), which applies a standardised scoring framework that reflects the likelihood of muscle invasion and supports consistency in reporting (17). This scoring system provides the basis for the structured assessment shown in **Figure 3** and strengthens diagnostic confidence while guiding subsequent treatment planning (17).

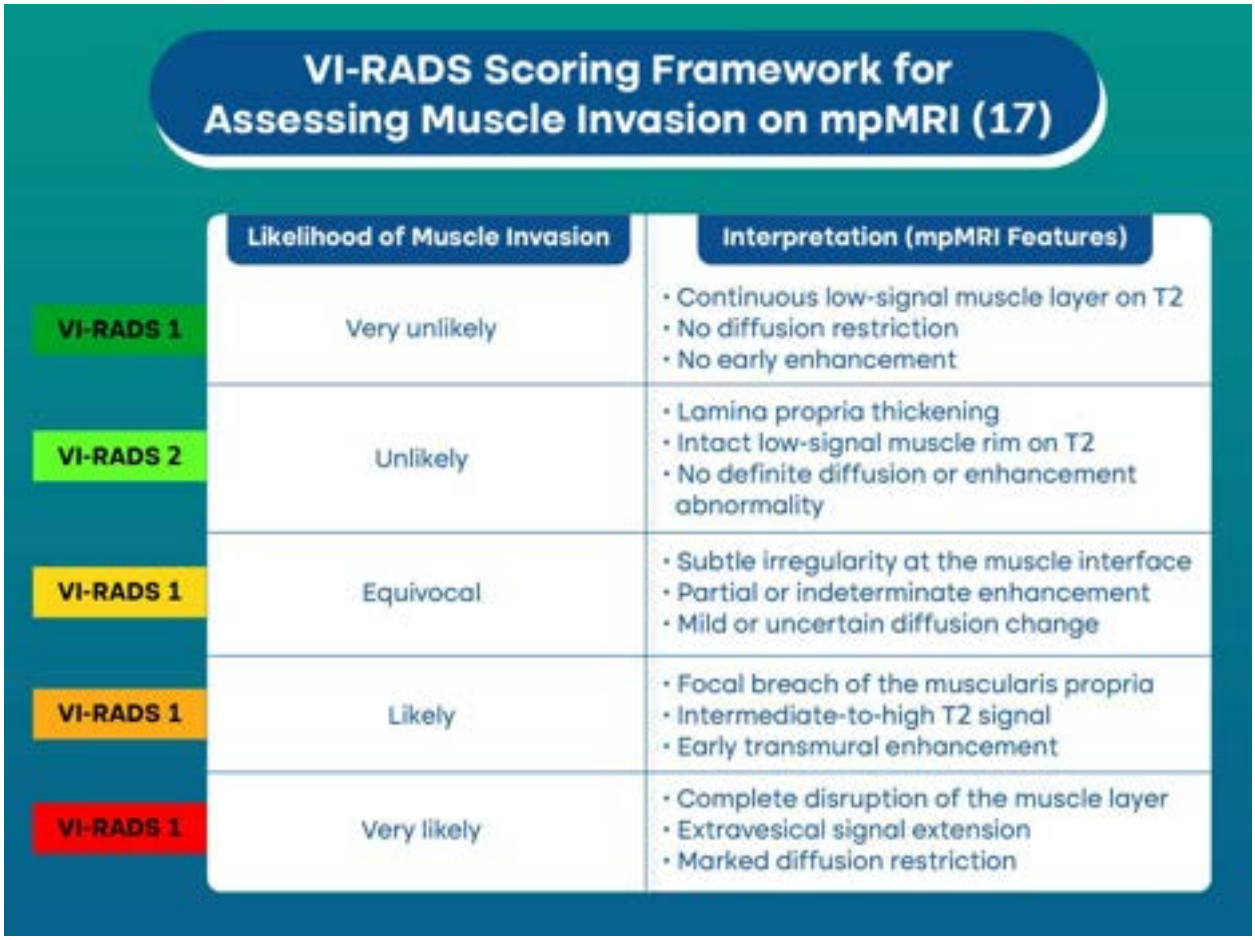


Figure 3. VI-RADS scoring system on mpMRI for muscle invasion assessment (17)

(Abbreviations – VI-RADS: Vesical Imaging Reporting and Data System; mpMRI: multiparametric magnetic resonance imaging; T2: T2-weighted imaging)

Positron emission tomography–computed tomography (PET-CT) is reserved for cases where metastatic disease is suspected or where conventional imaging provides inconclusive findings, enabling more precise metastatic work-up (18).

4. Histological confirmation

Definitive diagnosis and staging require histological confirmation, making transurethral resection of bladder tumour (TURBT) a critical step in the diagnostic pathway (14). TURBT establishes the pathological diagnosis and stage, and the presence of detrusor muscle in the specimen is essential to avoid under-staging and to ensure that subsequent treatment decisions are appropriate (14). The adequacy of the initial resection also determines whether a repeat procedure is needed and guides planning for intravesical therapy (14).

5. Adjunct molecular and cytogenetic biomarkers

Molecular and cytogenetic assays provide supplementary biological information that supports surveillance and risk assessment, but do not replace cystoscopy or imaging (18). Commonly used biomarkers include:

- FGFR3 mutation analysis
- Nuclear matrix protein 22 (NMP22)
- UroVysion fluorescence in situ hybridisation (FISH)

These biomarkers assist in refining risk assessment, particularly in surveillance settings (16).

6. Integration of diagnostic findings into NMIBC risk stratification

Diagnostic findings from cystoscopy, cytology, imaging and histopathology are combined to classify patients into NMIBC risk categories that guide treatment and surveillance intensity (14,19).

Patients are categorised as low, intermediate, or high risk based on tumour stage, grade, size, multiplicity, and presence of CIS (14,15). This risk-adapted model allows tailoring of treatment intensity and surveillance intervals to the patient's individualised risk of recurrence and progression (14).

MANAGEMENT OF LOCALISED DISEASE: NMIBC AND MIBC

Management of localised BCa depends on whether the tumour is confined to the mucosa (NMIBC) or invades the detrusor muscle (MIBC). This distinction guides the choice of intravesical therapy, radical surgery, and systemic treatment (14).

Figure 4 presents a visual summary of the treatment pathways for NMIBC and MIBC, positioning the major interventions within the continuum of care for localised disease.

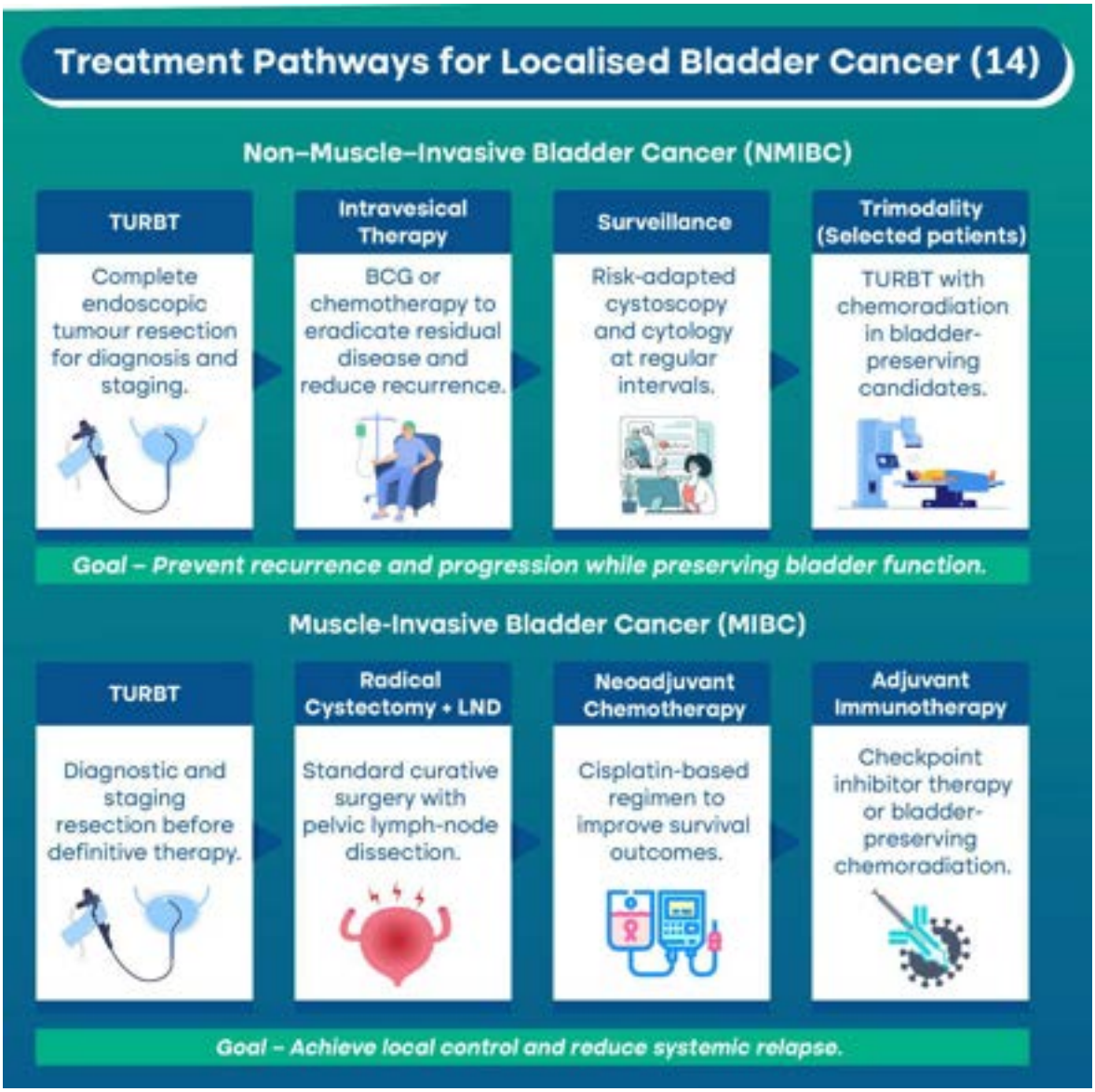


Figure 4. Overview of standard treatment pathways for localised bladder cancer, highlighting NMIBC and MIBC management approaches (14)

(Abbreviations – TURBT: transurethral resection of bladder tumour; NMIBC: non-muscle invasive bladder cancer; MIBC: muscle invasive bladder cancer; BCG: Bacillus Calmette Guerin; LND: lymph node dissection)

A. Management Strategies for NMIBC

The initial management of NMIBC involves endoscopic tumour removal by TURBT, which serves both diagnostic and therapeutic purposes (14). In patients with high-grade T1 tumours, a repeat TURBT is recommended due to the high risk of residual disease and potential under-staging (14).

Intravesical therapy plays a key role in decreasing recurrence and progression risk (14). Bacillus Calmette-Guérin (BCG) remains the standard intravesical adjuvant therapy for patients with high-risk NMIBC; however, long-term data show that up to 50% of patients lose response within five years (11). Alternative chemotherapeutic agents such as mitomycin C and gemcitabine have shown promising efficacy in early-phase trials (20).

Surveillance protocols are tailored according to the patient's recurrence risk, employing cystoscopy and urine cytology at intervals consistent with established NMIBC risk categories (21).

B. Therapeutic Approaches for MIBC

For operable MIBC, the standard management involves radical cystectomy along with bilateral pelvic lymph-node dissection (22). Systemic therapy is integrated as appropriate (22). Neoadjuvant cisplatin-based chemotherapy improves survival outcomes, as demonstrated by a randomised trial comparing MVAC (methotrexate, vinblastine, doxorubicin, and cisplatin) combined with cystectomy against cystectomy alone (23). Meta-analyses further confirm the benefits of MVAC and gemcitabine–cisplatin regimens in this setting (22).

In high-risk patients, adjuvant systemic therapy may be considered (24). The IMvigor010 trial evaluated adjuvant atezolizumab versus observation following radical cystectomy (24).

For patients who refuse or are unsuitable for radical cystectomy, bladder-preserving trimodality therapy, comprising maximal TURBT, chemotherapy, and radiotherapy, represents a viable alternative, provided selection criteria such as unifocal disease and adequate bladder capacity are met (14).

Advanced and Metastatic Disease: Systemic and Targeted Therapies

Systemic therapy is the cornerstone in managing advanced and metastatic UC (25). Treatment decisions rely on factors such as platinum eligibility, prior therapies, and the presence of targetable genomic alterations (25).

Figure 5 outlines the principal systemic treatment classes used in this setting and provides the framework within which individual therapeutic decisions are made.

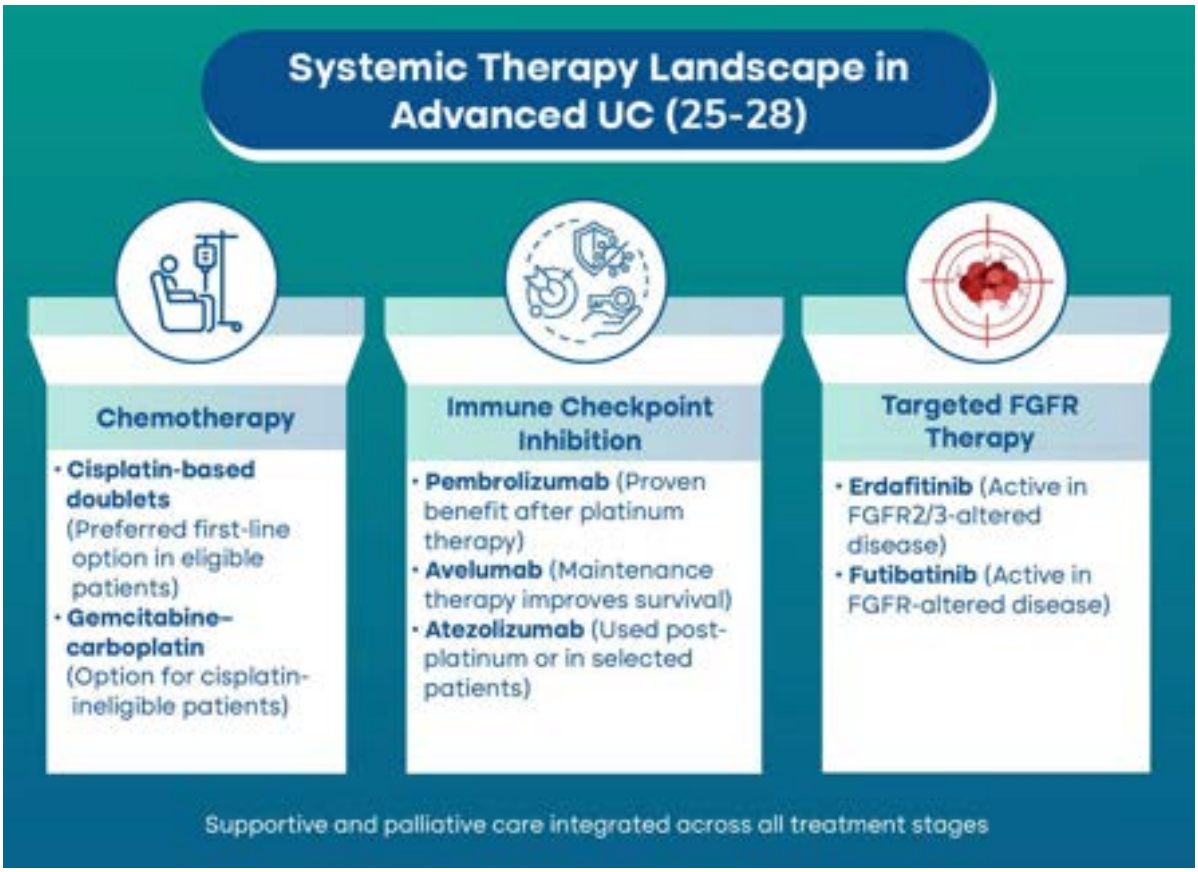


Figure 5. Core Therapeutic classes in advanced and metastatic UC (25–28)

(Abbreviations – UC: urothelial carcinoma; FGFR: fibroblast growth factor receptor)

Beyond the selection of systemic therapy, supportive and palliative care form a critical part of advanced-disease management (26). Contemporary evidence highlights the importance of proactively addressing symptom burden, treatment-related toxicities and functional decline as the disease progresses (24). Measures that support pain control, nutritional status, fatigue management and psychosocial wellbeing are essential for maintaining quality of life (24). Early integration of palliative care also facilitates more effective shared decision-making, ensuring that treatment goals remain aligned with patient preferences while systemic therapy options continue to evolve (25).

PROGNOSTIC MODELLING AND STRUCTURED SURVEILLANCE

Recurrence and progression patterns significantly impact outcomes in BCa and differ across established clinical risk groups (27). The European Organisation for Research and Treatment of Cancer (EORTC) risk tables, widely applied prognostic instruments, estimate recurrence and progression probabilities based on factors such as stage, grade, tumour size, tumour number, history of recurrence, and coexistence of CIS (28).

In contrast, the Club Urológico Español de Tratamiento Oncológico (CUETO) scoring system, developed from BCG-treated patient cohorts, incorporates variables including age, gender, grade, stage, tumour multiplicity, and CIS, providing risk predictions that can differ from those of the EORTC tables (29). Recent analyses suggest that the predictive accuracy of these tools may vary when applied to contemporary patient populations (29).

To support clinical interpretation, **Figure 6** summarises the main prognostic tools and surveillance elements using a four-option infographic, aligning risk modelling with NMIBC and MIBC follow-up requirements.

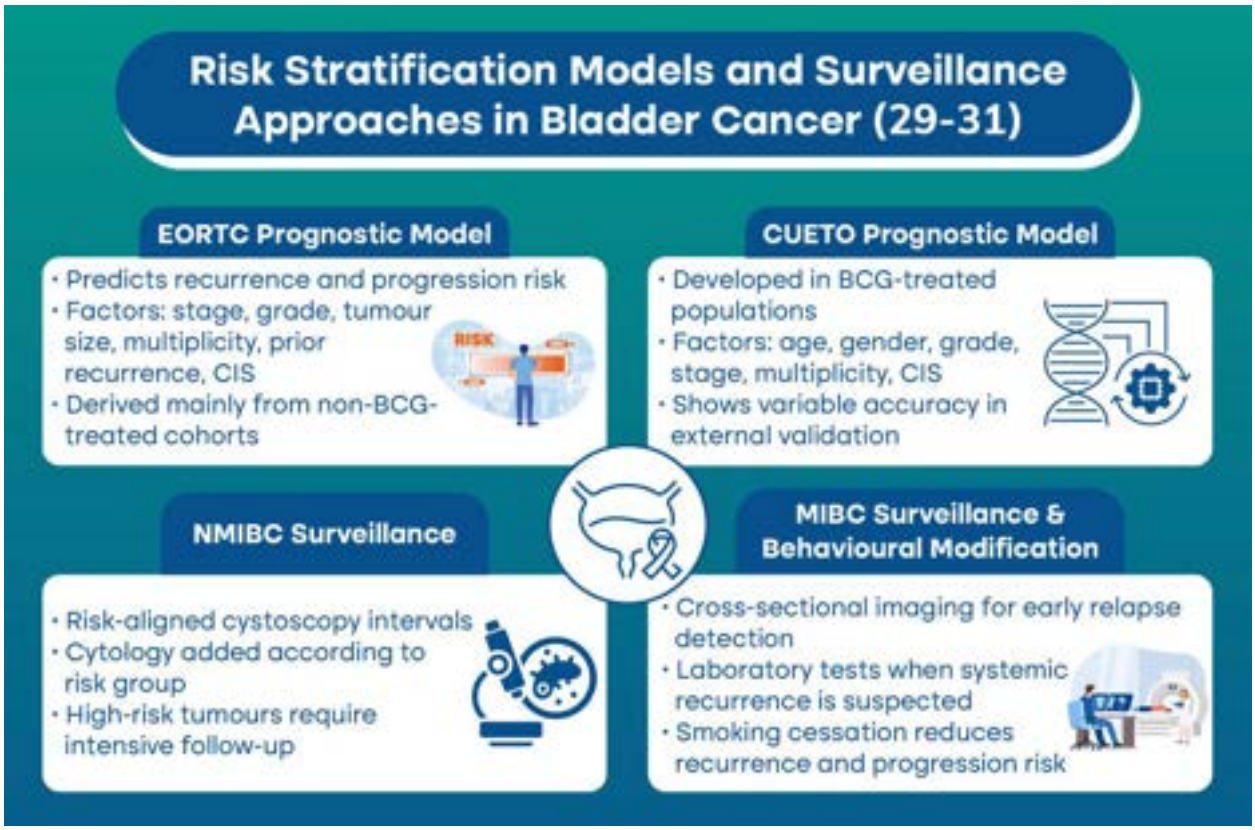


Figure 6. Prognostic tools and surveillance principles in localised Bca (29–31)

(Abbreviations – EORTC: European Organisation for Research and Treatment of Cancer; CUETO: Club Urológico Español de Tratamiento Oncológico; BCG: Bacillus Calmette Guerin; CIS: carcinoma in situ; NMIBC: non-muscle invasive bladder cancer; MIBC: muscle invasive bladder cancer)

Surveillance strategies for NMIBC are risk-adapted, with the scheduling of cystoscopy and cytology tailored according to low-, intermediate-, or high-risk categories (32). High-risk NMIBC patients require more intensive endoscopic follow-up because of their increased likelihood of recurrence and progression (32). MIBC, post-treatment surveillance prioritises early detection of relapse, with cross-sectional imaging playing a central role in follow-up protocols (12). Laboratory assessments are reserved for cases where systemic recurrence is suspected (12).

Behavioural modification also plays a critical role in clinical outcomes (31). Tobacco exposure is associated with increased risks of recurrence and progression, making smoking cessation an important factor in modifying disease course (31).

FUTURE DIRECTIONS: PERSONALISED AND TECHNOLOGY-DRIVEN CARE

Advances in BCa management increasingly emphasise precision-based approaches that integrate tumour biology with treatment response (30). Central to this progress are novel therapeutic combinations, especially the approval of ICIs and the FGFR tyrosine kinase inhibitor erdafitinib, representing two significant breakthroughs (30). Combining immunotherapy with targeted agents emerges as an appealing as an appealing strategy to enhance response rates and reduce resistance, underscoring the growing role of rationally designed systemic combinations (30).

Concurrent with therapeutic innovation, personalisation in BCa care is strengthened through deeper molecular characterisation. Messenger ribonucleic acid (mRNA) expression profiling has revealed biologically relevant molecular subtypes of UC, enabling more precise patient stratification (33).

The consensus molecular classification system further supports multiple distinct molecular subtypes, promoting subtype-based therapeutic selection as an emerging and promising direction (34).

To visually consolidate these developments, Figure 7 summarises four key innovation domains: immunotherapy combinations, biomarker-driven treatment, refined molecular subtyping, and AI-enabled surveillance.

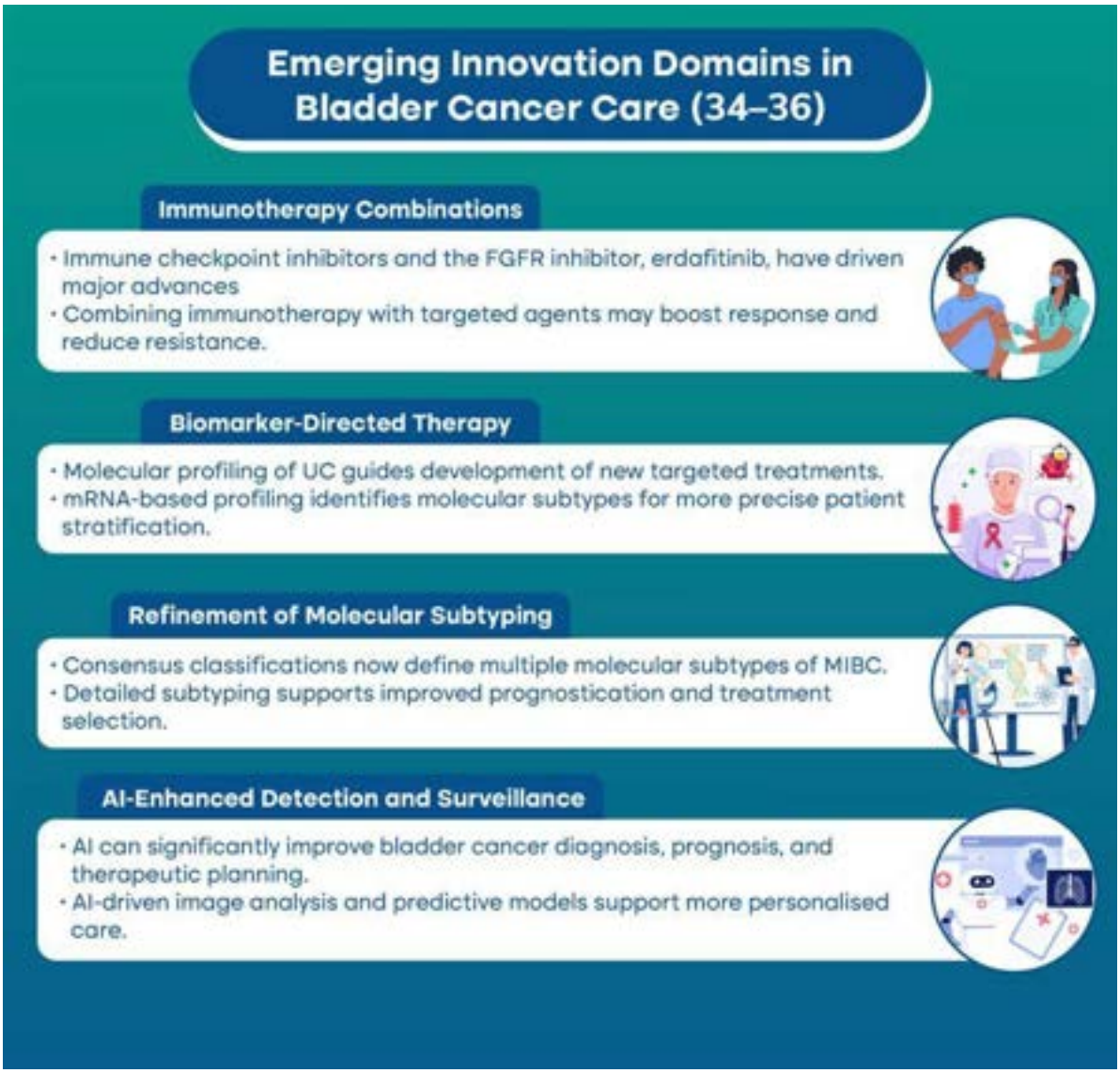
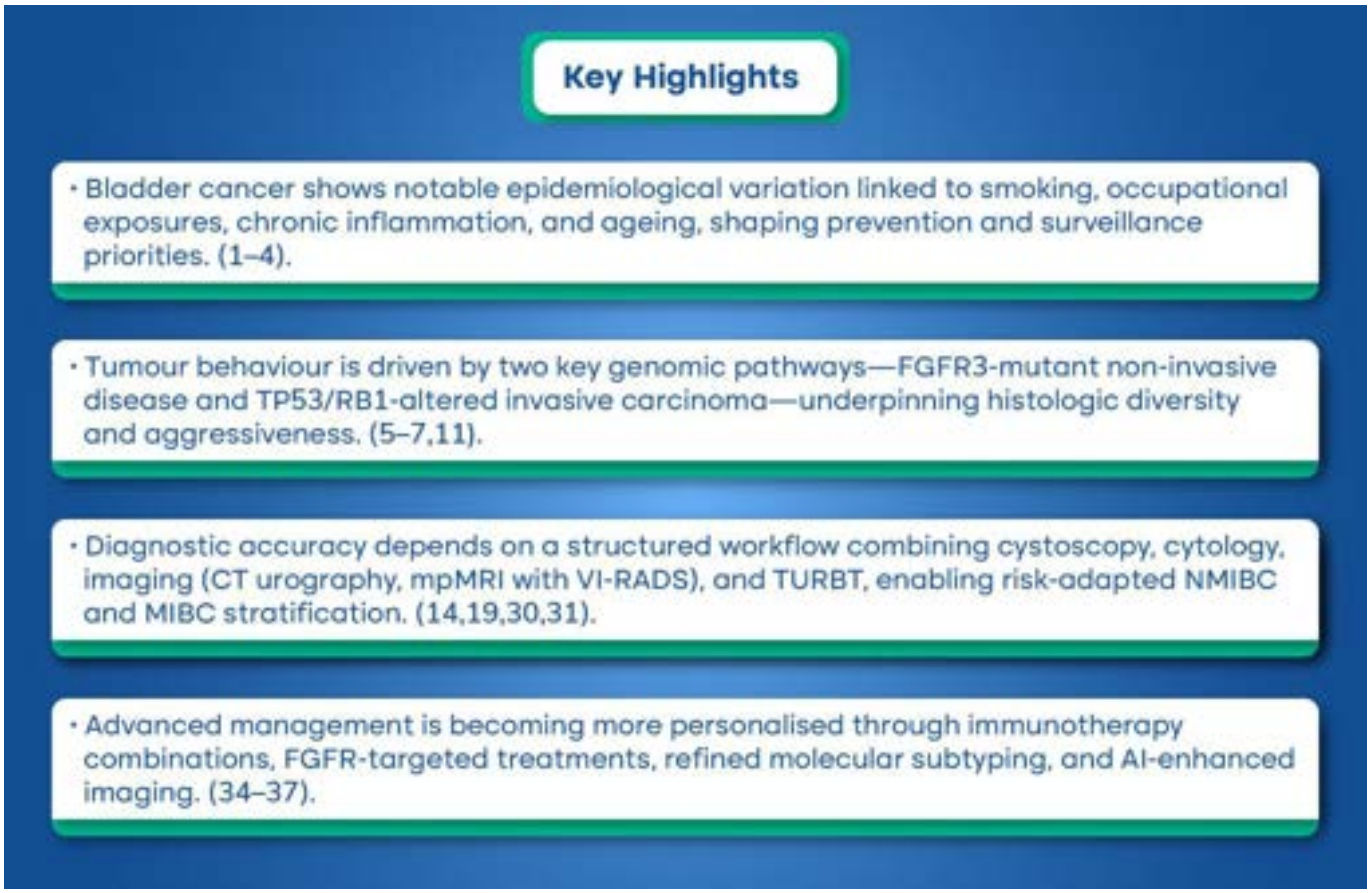


Figure 7. Key innovation domains driving personalised BCa care (34–36)

(Abbreviations – FGFR: fibroblast growth factor receptor; UC: urothelial carcinoma; mRNA: messenger ribonucleic acid; MIBC: muscle invasive bladder cancer)

Beyond biological and therapeutic advances, technology is positioned to further revolutionise clinical pathways (30). Artificial intelligence (AI) holds enormous potential for improving the diagnosis, prognosis, and treatment of bladder cancer (35). Current evidence highlights AI's capacity to transform diagnosis through enhanced image processing, predictive modelling, and personalised treatment planning (35,36). AI integration into cystoscopy and imaging techniques may improve early lesion detection, strengthen surveillance accuracy, and reduce observer variability (35).

Together, developments like therapeutic combination strategies, biomarker-guided pathways, molecularly informed categorisation and AI-enabled diagnostic refinement will signal a transition toward increasingly precise and adaptive management models in BCa (36).



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Breaking Boundaries in Urology:

Successful Laser Cystolithotripsy via Mitrofanoff at Aster Hospital, Muhaisnah, Dubai

Dr. Kunal Ranjit Meshram
Urology (Specialist)

PRESENTATION

- 28-year-old female
- Medical history of cloacal exstrophy with urinary augmentation surgery 20 years back. On tab ciproflox for 3 days. Allergic to IV ciproflox, but not tab ciproflox
- The patient is using a Nelaton Catheter for draining urine from the Mitrofanoff
- Admitted with complaints of:
  - Bilateral loin pain for 3 days, more in the right than the left
  - Lower abdominal pain
  - Fever
  - 2-3 episodes of vomiting per day
  - No shortness of breath, angina or palpitations

FINDINGS

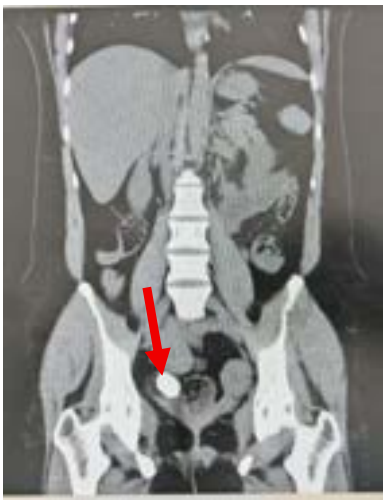
During Examination:

- Conscious and oriented
- No pallor/cyanosis/lymphadenopathy/icterus/pedal oedema
- Stable vitals
- Soft Abdomen
- Mitrofanoff present at the umbilical opening
- Bladder not palpable

CT Urogram showed:

- The augmented urinary bladder was positioned towards the right of the pelvis.
- The urethra was not demonstrable.
- Large dependent calculus measuring 2.2 cm/1100 HU.
- Self-catheterisation of a fistulous tract from the umbilicus to the urinary bladder along the right iliac fossa.
- Focal parenchymal thinning/lobulations in the middle thirds of the right kidney.
- The left kidney was relatively larger than the right, with a mildly striate nephrogram.
- Both adrenals were of normal size and density.
- No mass lesion / calcification and no renal calculi/ hydronephrosis.
- No focal mass lesion was seen, and perinephric areas appeared normal.

- No bladder wall thickening. No mass lesion was seen.
- A periumbilical adherent segment of ileo-sigmoid anastomosis was noted. The rest of the large bowel was non-visualised.
- Anorectal pull-through anastomosis sutures.
- Uterus didelphys with complete duplication of the uterus and cervix.
- Left tubo-ovarian complex with largely dilated convoluted tubal components, lying in the left iliac fossa, measuring 5.8 x 4 cm.
- A short segment of reactively thickened proximal small bowel was adherent anteriorly to the left tubo-ovarian complex.
- Adjacent thickened migrated omentum.
- Right tubo-ovarian complex in midline presacral position measuring 4.4 x 3.4 cm.
- L5 Spina Bifida Occulta (SBO).

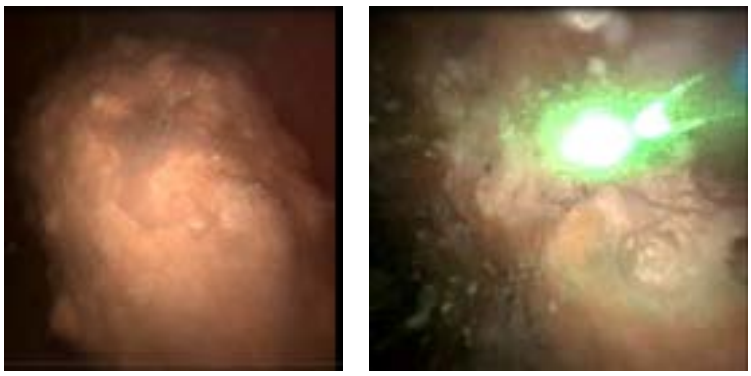


Pre-op image showing Bladder Calculus

DURING PROCEDURE

The patient underwent Laser Cystolithotripsy Surgery via Mitrofanoff:

- All parts were painted and draped under general anaesthesia in the supine position.
- The guide wire was inserted into the bladder via the Mitrofanoff.
- A semi-rigid URS scope was introduced into the Mitrofanoff, e/o retracted opening present with the rest of the passage normal.
- Around 3 cm single stone was seen in the bladder.

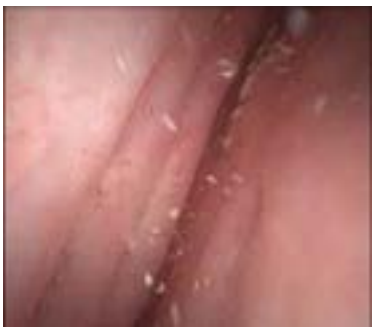


Endoscopic view of the calculus during surgery

- A flexible URS scope was introduced via an access sheath and a laser-dusted calculus.
- Fragments and dust were suctioned with the FANS sheath.
- A 12 Fr Silicone Foley catheter was inserted.

POST PROCEDURE

The patient tolerated the procedure well and had an uneventful post-procedure. Patient was tolerating an oral diet well and was discharged in a stable condition with a Foley catheter in situ.



Endoscopic view after surgery with complete clearance of the stone

DISCUSSION

Bladder calculi are a known complication of bladder augmentation that affects 12–52% of patients after enterocystoplasty. Risk factors include poor compliance with bladder washout, the need for CIC, the presence of foreign bodies, recurrent urinary tract infections, the type of bowel segment used for augmentation, immobility, and bladder neck closure. Various methods of cystolithotripsy have been reported, including open and percutaneous lithotripsy, but cases with Mitrofanoff conduits are rare. There is concern about conduit damage during surgery; however, in the present case and in previous reports, damage was prevented by using a ureteral access sheath over the conduit. Thomas et al. reported that 18–28 Fr ureteral access sheaths were used and that no patients had obvious conduit injuries that would have resulted in urinary incontinence. The larger the sheath, the easier it is to expel stone debris and deliver and withdraw irrigation fluid; however, a larger sheath also increases the burden on the conduit. Accordingly, they advocated the use of an 18Fr sheath.

Sakly et al. first dilated the conduit with a 10–16 Fr navigation catheter and then a 15/16 Fr ureteral access sheath to care for conduit injuries. The ureteral access sheath allows free, unobstructed drainage of irrigation fluid around the working instrument, reducing both intravesical pressure within the augmented bladder and the risk of rupture. Previous reports have shown that the surgical operation time is approximately 2.5–3 hours and that patients can leave the hospital within a few days after surgery.

In the present case, the patient underwent surgery due to cystitis symptoms associated with an enlarged bladder stone. The patient's symptoms improved after surgery with no residual stones. In this case, we restricted the surgical operation time to less than 3 hours. Further study is needed to establish the ideal surgical management for complicated cases.

CONCLUSION

We reported a case of transurethral laser lithotripsy using the Mitrofanoff urethral conduit for a patient with bladder stones. Using the FANS ureteral access sheath made lithotripsy and the retrieval of the bladder stone more effective. But careful management is necessary to avoid injury to the urethral mucosa, and the operation should take less than 2 hours.

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