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HealthNews DIGEST

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33rd Edition

Dr. Sherbaz Bichu

CEO & Specialist Anaesthetist
Aster Hospitals & Clinics, UAE

On behalf of Aster's leadership, I am excited to welcome you to the 33rd edition of our HealthNews Digest.

As we observe Pink October this month, we are reminded of the critical importance of breast cancer awareness. Early detection and timely intervention, promoting awareness, encouraging regular screenings, and empowering patients through informed decision-making remain essential components of our responsibility as clinicians.

I am also pleased to acknowledge the continued success of our COSMOS, a heartfelt community initiative dedicated to conquering cancer through compassion, collaboration, and courage. By sharing experiences, offering emotional support, and uplifting others, COSMOS exemplifies the integration of advanced oncology practices with a deeply human-centred approach to care.

As we continue our efforts in oncology and beyond, I implore everyone to sustain this momentum of embracing innovation, fostering patient trust, and promoting preventive healthcare at all levels. Together, we can strengthen our role as leaders in shaping a healthier, more cancer-aware society.

Dr. Ramanathan V

Medical Director
Aster Hospitals & Clinics, UAE

As the Group Medical Director of Aster Hospitals and Clinics, I am pleased to share my thoughts in the 33rd edition of HealthNews.

I am happy to introduce the COSMOS Crew, an extension of our COSMOS Community to Conquer Cancer initiative. In this initiative, our patients are paired with carefully selected volunteers who have either triumphed over cancer themselves or supported a loved one through it. This human connection embodies our commitment to holistic healing by filling the emotional and mental health gap that medical treatment alone cannot bridge, providing strength, understanding, and hope at every stage of recovery.

Parallelly, I am proud to highlight the success of our HIPEC (Hyperthermic Intraperitoneal Chemotherapy) cases, which significantly advanced our oncological and surgical capabilities at Aster Hospital, Al Qusais, Dubai. These achievements are a testament to our surgical teams' precision, interdisciplinary collaboration, and commitment to evidence-based care.

Let us continue to blend innovation with empathy, ensuring that our patients receive the most advanced treatments and the compassionate support that fosters true healing.

Successful Repair of Carpal-Hamate Dislocation by Closed Reduction and Internal Fixation at Aster Hospital, Muhaisnah, Dubai

Dr. Masoodh Basha
Orthopaedics (Specialist)

PRESENTATION

- 24-year-old male, referred from Aster Clinic
- History of an accidental fall of an air conditioner on the right hand 1 day ago
- Admitted with:
  - Complaints of severe pain and swelling over the right palm and wrist

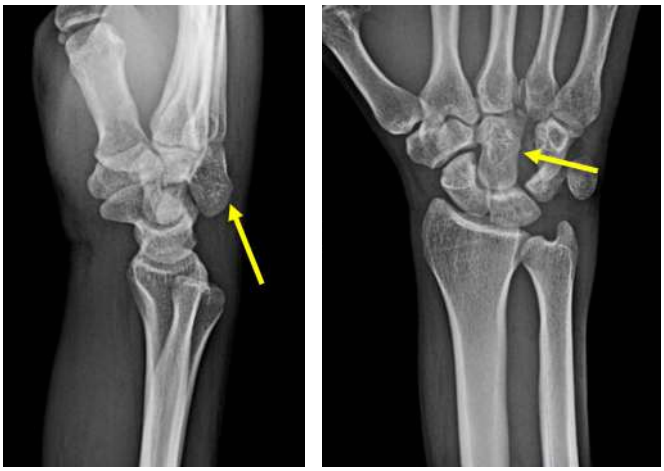
FINDINGS

During Examination:

- Severe swelling over the right wrist and hand
- Diffuse tenderness over the hand and wrist
- Wrist movements restricted due to pain
- No deficit
- Active finger movements: Present, painful
- Distal fingers: All fingers showed 100% SpO2, radial pulse present

X-ray showed:

- Disruption of Gilula lines with an empty space at the hamate position in the AP view
- Hamate was seen dorsally in the lateral view

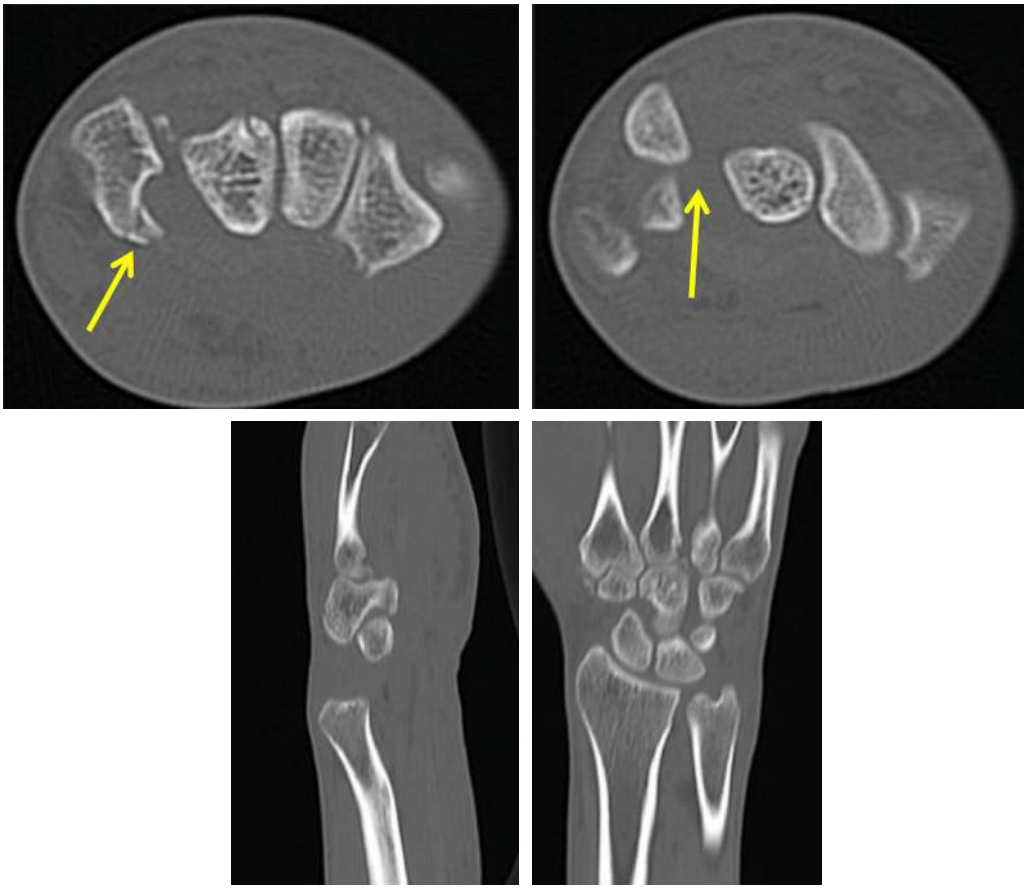


(a) (b)

Pre-op X-ray images showing (a) Dorsally Displaced Hamate and (b) "Empty Hamate Sign"

CT showed:

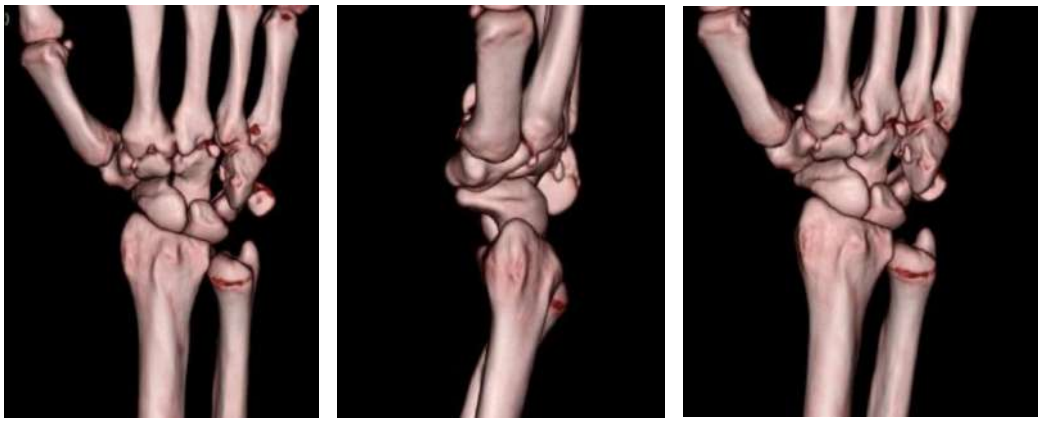
- Fracture of the capitate, hamate and 3rd metacarpal base
- Dislocation of the hamate proximally and dorsally with disruption of the 3/4 metacarpal base joint
- Disruption of all 3 Gilula's lines suggestive of carpometacarpal dislocations and carpal incongruence
- The gap between 3 and 4 MC base, capitate and hamate, hamate with 4,5 MC displaced proximally and dorsally and the proximal pole of hamate dislocated dorsally over the rim of triquetrum and lunate



Pre-op CT Images showing Dorsally Displaced Hamate with minor fractures

3D Reconstruction:

- The below images showed the 3D orientation of the displaced hamate



3D Reconstruction

PLANNING

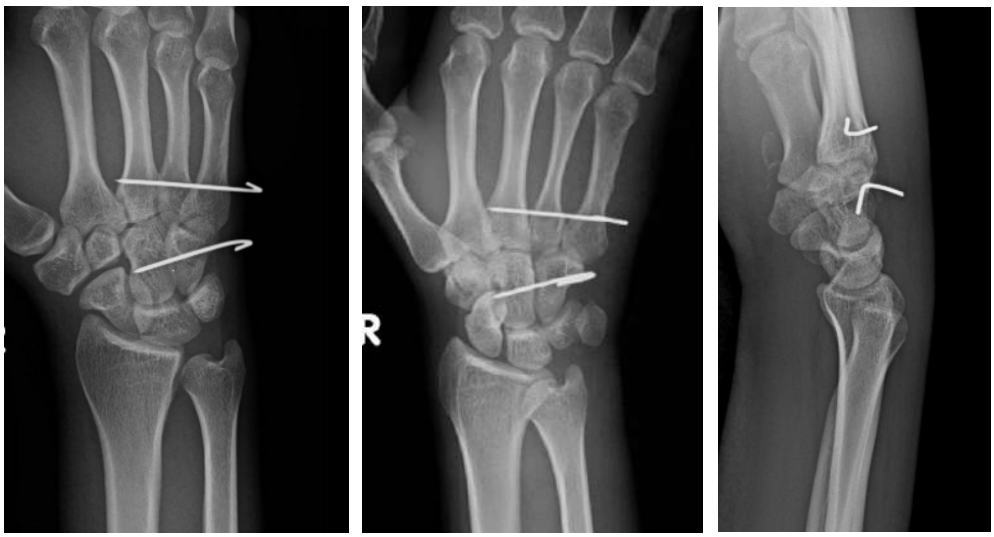
The main pathology was the dorsally displaced hamate. The fractures of the carpals and metacarpals were minor, and the fragments were too small to be fixed. The fractures were possibly avulsion fractures due to the disruption of the ligaments.

The condition and options were discussed with the patient, along with the chance of failure to reduce the closed, which may need open reduction, stiffness, and arthritis later.

DURING PROCEDURE

The patient underwent Closed Reduction and K-wire Fixation for Right Hand Carpal - Hamate Dislocation:

- Under C-arm, traction to 4,5 fingers was given.
- The hamate was palpated over the dorsum of the wrist and milked distally with pressure over the hamate dorsally.
- Traction was reduced gradually, and stability was assessed.
- 2 K-wires were inserted into the hamate-capitate and 3/4/5 MC base.
- A bulky bandage was applied along with the splint.



Intra-op Images

POST PROCEDURE

The patient tolerated the procedure well and was in stable condition on discharge. The K-wires were removed after a month, and the patient was advised to undergo physiotherapy.

DISCUSSION

Isolated dislocation of the hamate without a major fracture is a rare injury. Concomitant fracture of other carpals or metacarpals can be associated with this injury. It can lead to injury of the ulnar nerve and compartment syndrome. Stiffness, arthritis, grip weakness, and ulnar neuropathy can be long-term complications of this injury. Some cases can require open reduction and fixation.

Our case had a lot of clinical swelling but did not have symptoms of acute compartment syndrome or any nerve injury. We reduced the hamate closed and fixed it with K-wires for stability. Over the next few days, he was closely monitored for compartment syndrome, and precautions were taken to reduce oedema. He recovered well over the next four weeks, and the K wires were removed.

He had stiffness of his fingers and wrist and was advised physiotherapy here, but he left for his home country 6 weeks after the index surgery. He did not have any neuropathy or weakness at the last follow-up.

CONCLUSION

Hamate dislocations are rare hand injuries with potentially severe complications. Stiffness is the main complication, and physiotherapy is essential for return to normal function.

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Understanding MASLD Diagnosis, Risk Stratification and Comprehensive Care

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OVERVIEW OF MASLD: DEFINITION, DETERMINANTS, AND CLINICAL IMPLICATIONS

Metabolically-dysfunction-associated steatotic liver disease (MASLD) is characterised by fat accumulation in the liver, accompanied by a minimum of one cardiometabolic risk factor, while excluding other detectable causes (1). As illustrated in Figure 1, the presence of cardiometabolic risk factors-including obesity, dysglycaemia, dyslipidaemia, and hypertension-forms the basis of the diagnostic framework linking metabolic dysfunction to hepatic steatosis in MASLD (1).

Cardiometabolic Determinants of MASLD (1)

Risk Factor	Criteria (Adults)
Overweight/Obesity	• BMI ≥25 kg/m² (≥23 kg/m² in Asians) • Waist circumference: ≥94 cm (men) & ≥80 cm (women) in Europeans; ≥90 cm (men) & ≥80 cm (women) in South Asians/Chinese; ≥85 cm (men) & ≥90 cm (women) in Japanese
Dysglycaemia / Type 2 Diabetes	• Prediabetes: HbA1c 5.7–6.4% or FPG 100–125 mg/dL (5.6–6.9 mmol/L) or 2h-OGTT 140–199 mg/dL (7.8–11 mmol/L) • Type 2 Diabetes: HbA1c ≥6.5% or FPG ≥126 mg/dL (≥7.0 mmol/L) or 2h-OGTT ≥200 mg/dL (≥11.1 mmol/L) or on treatment
Plasma Triglycerides	≥1.7 mmol/L (≥150 mg/dL) or on lipid-lowering treatment
HDL-Cholesterol	<1.0 mmol/L (<39 mg/dL) in men; <1.3 mmol/L (<50 mg/dL) in women, or on treatment
Blood Pressure	≥130/85 mmHg or on antihypertensive treatment

Figure 1. Cardiometabolic risk factors defining MASLD (1)

(Abbreviations – MASLD: metabolic dysfunction–associated steatotic liver disease; BMI: body mass index; HbA1c: glycated haemoglobin; FPG: fasting plasma glucose; OGTT: oral glucose tolerance test; HDL: high-density lipoprotein; mmHg: millimetres of mercury; mg/dL: milligrams per decilitre; mmol/L: millimoles per litre)

This term was introduced in 2020 to better reflect fatty liver disease linked to metabolic dysfunction (2). Replacing the previous designation, non-alcoholic fatty liver disease (NAFLD), with MASLD represents an important step forward in the field (2). MASLD covers a range of conditions, from isolated steatosis (metabolic dysfunction-associated steatotic liver, MASL) to metabolic dysfunction-associated steatohepatitis (MASH), and fibrosis and cirrhosis as well (1). MASH is identified by histological evidence of hepatocellular ballooning together with lobular inflammation (1). The updated consensus on steatotic liver disease (SLD) includes MASLD within its definition (1). In addition to MASLD, the SLD category includes MetALD (MASLD with moderate alcohol intake), alcohol-related liver disease (ALD), drug-induced and monogenic forms, as well as cryptogenic SLD (1).

In the Asia-Pacific region, prevalence is estimated at 28–40%, similar to global rates (3). MASLD represents the hepatic component of metabolic syndrome and has a strong, bidirectional association with other comorbidities (4). Because of the similarity in pathobiology shared between T2DM and MASLD, both disorders commonly coexist in many patients, and this coexistence may potentiate disease-related outcomes, leading to more rapid progression and increased complications of both individual diseases (4).

Given its multisystem involvement and cardiometabolic risks, optimal and holistic MASLD management requires a multidisciplinary team approach involving relevant specialists (4).

This article outlines a structured clinical framework for MASLD diagnosis and management, integrating metabolic risk assessment, non-invasive fibrosis stratification, and multidisciplinary strategies to deliver individualised, patient-centred liver care.

UNDERSTANDING DISEASE MECHANISMS AND PHENOTYPIC DIVERSITY

MASLD is a heterogeneous and multifactorial disease (5). Its pathophysiology is explained by a lot of interlinked processes, including insulin resistance (IR), which is the main part of metabolic syndrome, and lipotoxicity caused by the build-up of toxic lipid species (5). Among genetic factors, the single-nucleotide polymorphism (SNP, I148M variant) in the PNPLA3 gene is most strongly related to metabolic dysfunction-associated steatohepatitis (MASH) (5). In addition, the TM6SF2 E167K variant increases the risk of MASH (5).

Alongside these genetic factors, gut microbiota and their metabolites cause intestinal barrier dysfunction after damaging intestinal mitochondria, which allows these metabolites and the harmful substances they produce to easily cross the intestinal barrier and enter the liver through the hepatic–intestinal cycle (6). Once in the liver, these factors trigger hepatic mitochondrial dysfunction, the release of pro-inflammatory factors, and other mediators that promote hepatocellular steatosis and accumulation of fat in the liver, causing MASLD (6).

As the disease progresses, mitochondrial structure becomes abnormal and fatty acid oxidation is reduced in advanced stages (6). In MASLD, oxidative phosphorylation is limited, the respiratory chain complexes lose activity, and electron leakage is increased, leading to excess ROS production (6). Disease severity is marked by oxidative stress, reduced antioxidant activity, raised oxidative markers, and damage to mtDNA (6). These effects add to the inflammation response and promote further lipid droplet accumulation (6).

As shown in Figure 2, these changes link the initial gut microbiota–driven hepatic mitochondrial injury to downstream structural and functional mitochondrial abnormalities, which lead to oxidative stress, metabolic changes, and inflammatory responses, thereby advancing MASLD progression (6).

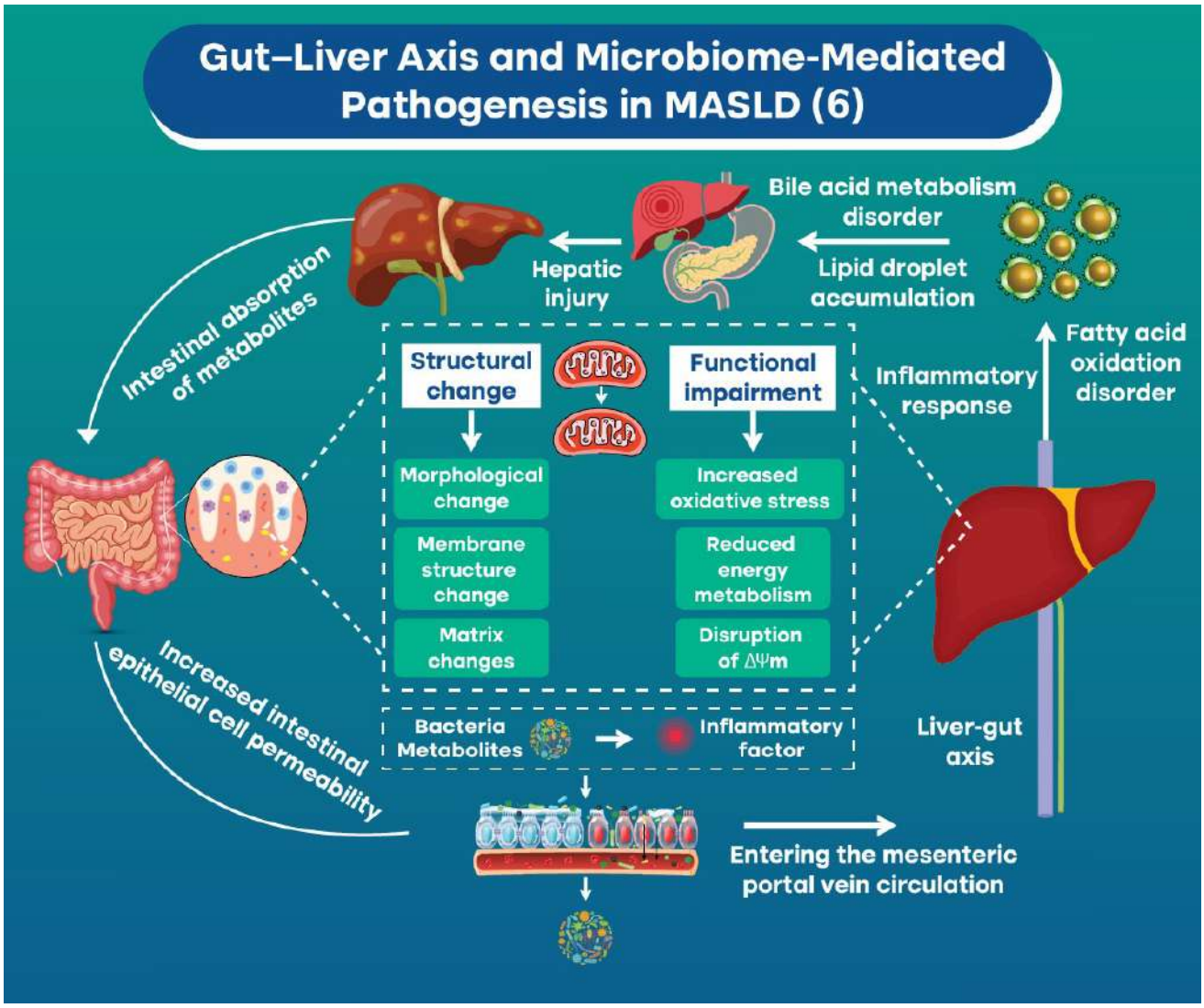


Figure 2. Gut Microbiota–Mitochondria Interactions in MASLD Pathogenesis (6)

(Abbreviations: MASLD – metabolic associated steatotic liver disease; ΔΨm – mitochondrial membrane potential)

Dysbiosis allows bacterial products to enter the systemic circulation (5). Once there, they activate macrophages and Kupffer cells, leading to inflammation (5). This inflammation contributes to fibrosis, where platelet-derived growth factor plays a key role by regulating the expression of metallopeptidases (collagenases) and promoting the accumulation of the extracellular matrix, thereby hindering its breakdown (7). Evidence also shows that South Asians are more likely to develop MASLD (5). This higher risk is linked to their greater proportion of visceral body fat, increased abdominal obesity, stronger insulin resistance, and relatively lower muscle mass (5).

REFINING DIAGNOSIS WITH METABOLIC CRITERIA AND FIBROSIS RISK TOOLS

MASLD is a shift towards a diagnosis of inclusion based on the presence of metabolic dysfunction, the key driver of the disease (3). Applying positive diagnostic criteria for MASLD allows clinicians to recognise its overlap with other liver conditions (3). Individuals with steatohepatitis and advanced fibrosis are at the highest risk of liver complications and death (3). For this reason, early identification of patients with severe disease, particularly those with extensive fibrosis, is of great importance (3).

To assist in this, a tiered strategy incorporating non-invasive assessments, advanced imaging, and AI-based risk models is increasingly applied, as summarised in Figure 3.

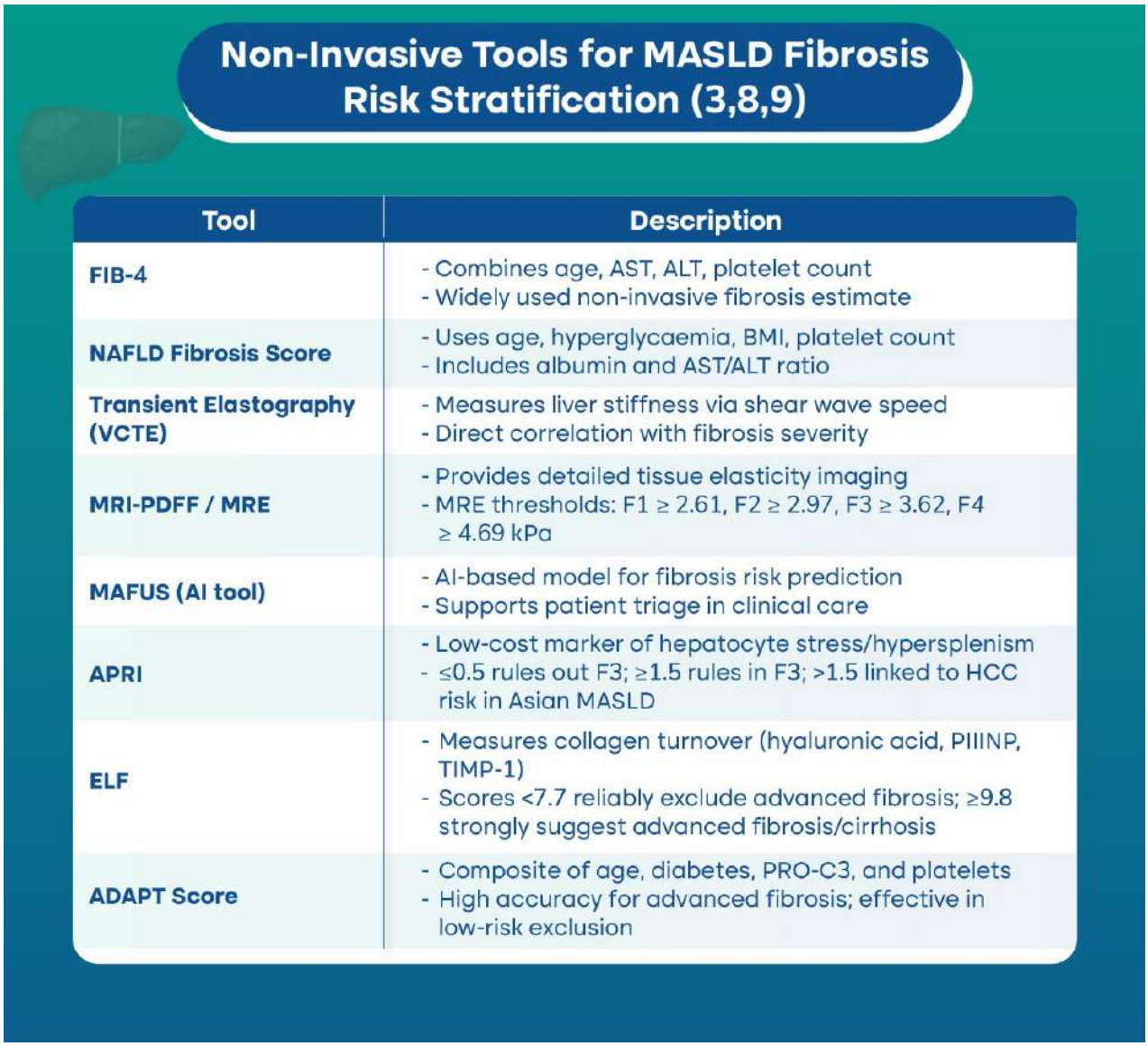


Figure 3. Non-invasive modalities used in MASLD risk stratification, ranging from biochemical scores to advanced imaging and AI-supported models (3,8,9)

(Abbreviations – FIB-4: fibrosis-4; AST: aspartate aminotransferase; ALT: alanine aminotransferase; NAFLD: non-alcoholic steatotic liver disease; MASLD: metabolic dysfunction–associated steatotic liver disease; BMI: body mass index; VCTE: vibration-controlled transient elastography; MRI-PDFF: magnetic resonance imaging–proton density fat fraction; MRE: magnetic resonance elastography; MAFUS: machine-learning assisted fibrosis utility system; kPa: kilopascal)

Even with progress in diagnostics, liver biopsy continues to be the gold standard for staging and diagnosing MASLD (8). Histology makes it possible to directly assess hepatocellular ballooning, lobular inflammation, steatosis, and fibrosis, helping confirm steatohepatitis and separate MASH from simple steatosis (8,10). Biopsy is most useful when NIT (Non-invasive tests) results are unclear, when clinical features are unusual, or when trials require histological endpoints (10). But because its invasive, costly, and carries some risks, it is not practical for routine use (8). As a result, NITs play the main role in assessment, with biopsy reserved for selected patients (3).

MANAGING MASLD WITH LIFESTYLE AND TARGETED THERAPEUTICS

As a metabolic disorder closely linked to unhealthy lifestyles, such as excess caloric intake and physical inactivity, MASLD management should primarily focus on lifestyle modifications and behavioural changes (4). A key

component of managing all forms of MASLD involves adopting healthier dietary habits and increasing levels of physical activity (10).

KEY COMPONENTS OF EFFECTIVE FIRST-LINE MANAGEMENT INCLUDE:

- **Diet:** A Mediterranean-type dietary pattern is the most evidence-based for MASLD, beneficial for non-cirrhotic MASLD (3).
- **Exercise:** Moderate-intensity aerobic or resistance training for 150–300 minutes/week improves hepatic and cardiometabolic outcomes (4).
- **Weight loss goals:** A 5% reduction in body weight may help reverse hepatic steatosis (10). Losses of 7–10% can improve MASH, while reductions greater than 10% may reverse fibrosis (10).
- **Combination approach:** Managing MASLD is more effective when dietary modification is paired with physical activity, rather than using either approach on its own (10).

Figure 4 presents the multidisciplinary care pathway, moving from lifestyle-based interventions to pharmacological and surgical options.

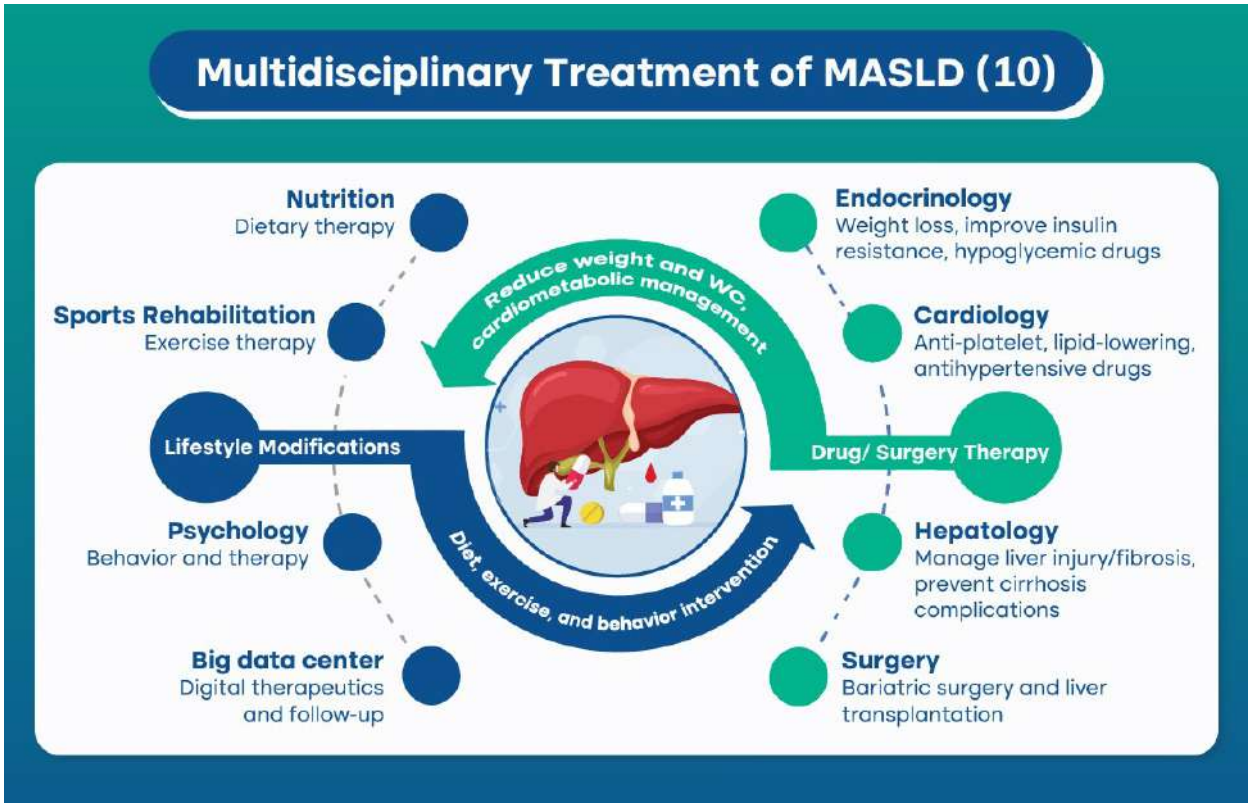


Figure 4. Multidisciplinary Management of MASLD (10)

(Abbreviations- MASLD- metabolic-associated steatotic liver disease)

PHARMACOLOGICAL TREATMENT

Resmetirom, a selective thyroid hormone receptor-beta (THR-β) agonist, received FDA (Food and Drug Administration) approval in March 2024 for treating patients with MASH and fibrosis at stage F2–F3 (11). For patients weighing under 100 kg, the daily dose is 80 mg, while those above 100 kg receive 100 mg (11). However, prescribing resmetirom requires a thorough patient assessment by a specialist and should be supervised within a multidisciplinary context (3).

Recently, the FDA authorised semaglutide, a GLP-1 receptor agonist, for the management of MASH in adults with moderate or advanced fibrosis (12). A pivotal clinical trial demonstrated that while 63% of patients who received Semaglutide showed MASH resolution and no aggravation of liver fibrosis, only 34% of patients who received a placebo achieved the same result (13). Improvement in liver fibrosis, with no added steatohepatitis, was reported in 37% of patients on semaglutide, versus 22% of those receiving placebo (13).

Peroxisome proliferator-activated receptor (PPAR-γ) agonist pioglitazone has been reported to improve histological features of steatohepatitis, though its effect on fibrosis regression remains uncertain (14). Its use is further limited by the concerns over side effects (14). Another agent, lanifibranor, a pan-PPAR agonist, demonstrated a dose-related improvement in both steatohepatitis and fibrosis (14). Saroglitazar, a dual PPAR α/γ agonist, has also shown benefits in reducing liver steatosis and improving enzyme levels, and it is approved in India for managing Type 2 Diabetes Mellitus as well as MASH (14).

Vitamin E therapy in non-diabetic MASH patients has been linked with improvements in steatosis, although no clear evidence supports fibrosis benefits (15). Concerns remain about its long-term safety, particularly regarding cardiovascular outcomes and prostate cancer risk, which limit its broader use (15).

For patients requiring more substantial weight loss, bariatric surgery can be considered, but its invasive nature and possible adverse outcomes require careful patient selection (2).

INTEGRATING MASLD INTO SPECIALIST-DRIVEN HEPATOLOGY PATHWAYS

MASLD is common in the general population, but only a small yet notable fraction progresses to advanced liver fibrosis (≥ F3 stage) (3). Many of these patients frequently remain without symptoms until hepatic decompensation appears, which often results in missed chances for preventive care (3). Early identification is therefore crucial (2). Imaging, liver biopsy, and non-invasive scoring tools are needed to detect those at risk of progression to cirrhosis or liver cancer (2).

Among non-invasive assessments, the Fibrosis-4 Index (FIB-4) has shown prognostic value in monitoring fibrosis changes over time and in stratifying future risks of morbidity and mortality (2). Other tools, including vibration-controlled transient elastography and enhanced liver fibrosis scores, are also widely used to follow disease progression and predict outcomes (3). Patients who fall into intermediate or high-risk categories on biochemical or imaging tests should be referred promptly to hepatology specialists for further assessment (4).

To optimise patient outcomes, integrated multidisciplinary care models must be established, which:

- Provide timely, standardised management
- Improve clinical effectiveness and cost-efficiency
- Enhance patient satisfaction (3).

A well-defined referral algorithm needs to be developed and put in place to make this approach part of routine clinical practice (4). Figure 5 highlights this specialist-driven management and referral framework, emphasising non-invasive risk stratification, referral pathways, and the involvement of multidisciplinary team's referral pathways, and multidisciplinary team.

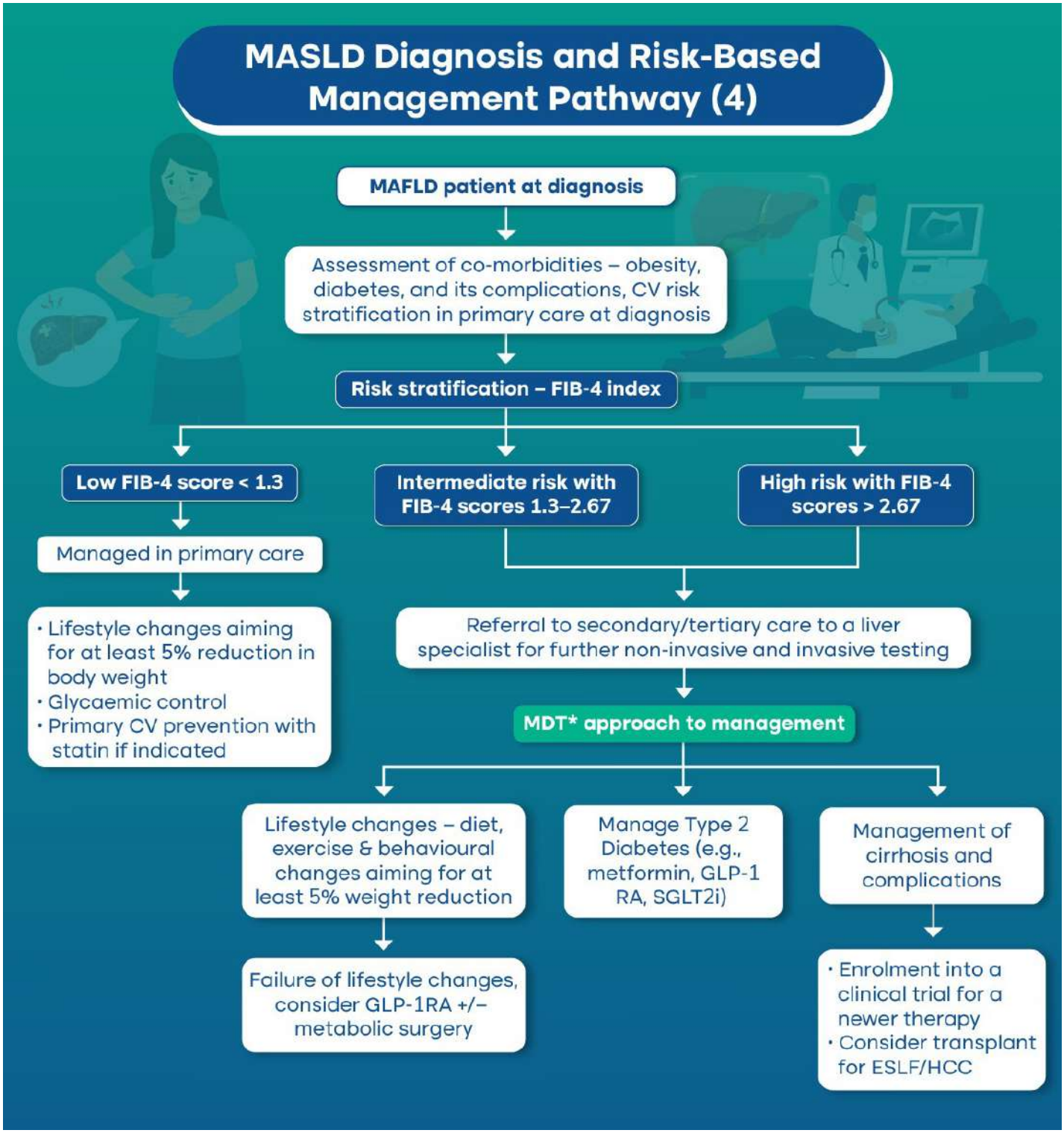
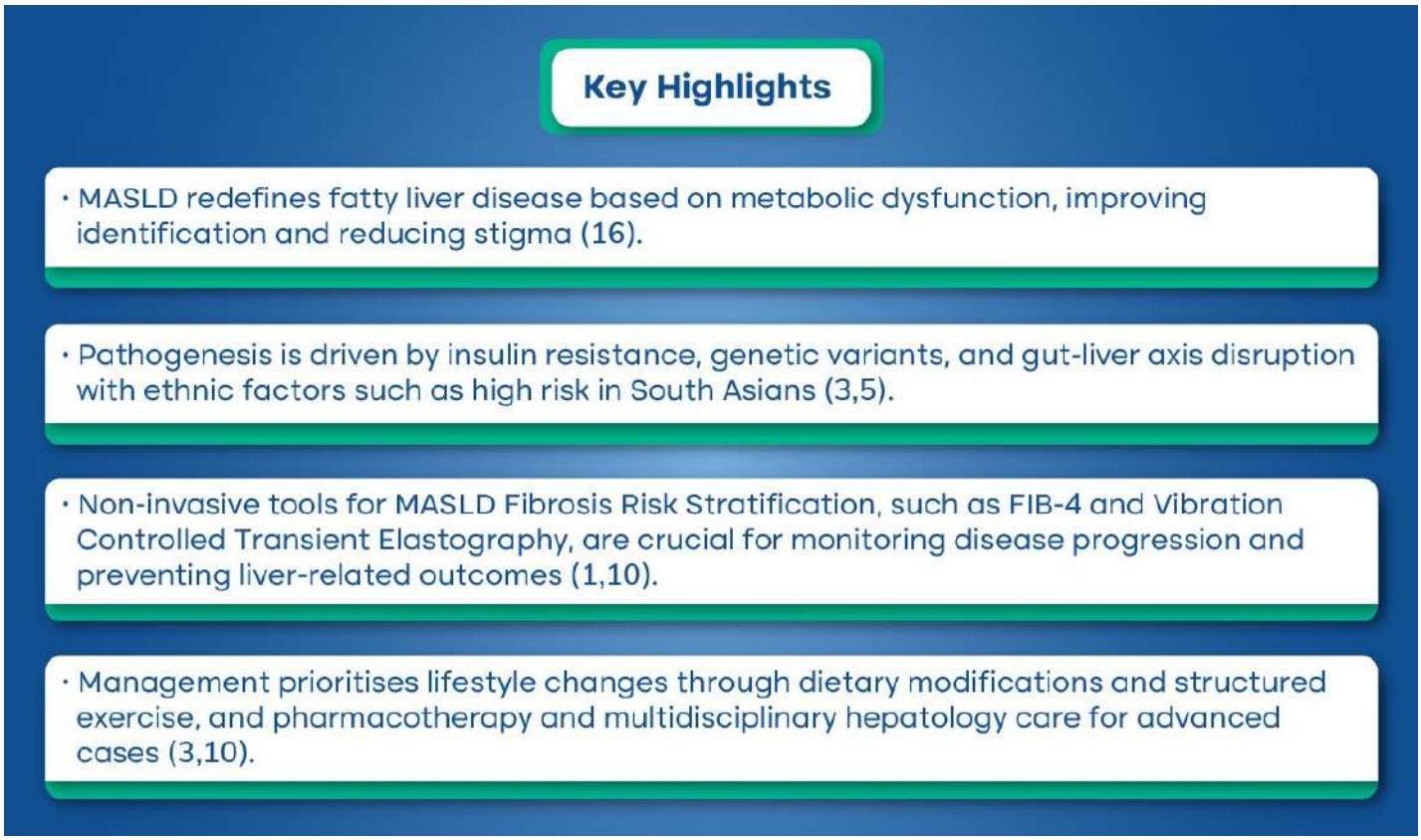


Figure 5. Specialist-Driven Management and Referral Framework for MASLD (4)

(Abbreviations- MASLD – metabolic dysfunction-associated steatotic liver disease; CV – cardiovascular; FIB-4 – Fibrosis-4 index (a non-invasive score to estimate liver fibrosis); MDT – multidisciplinary team; GLP-1 RA – glucagon-like peptide-1 receptor agonist; SGLT2i – sodium-glucose cotransporter-2 inhibitor)

Looking forward, advances in technology promise to enhance risk stratification further, including the use of AI in MASLD to reduce the need for invasive liver biopsies by improving diagnostic accuracy and overcoming limitations such as misclassification (3).



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Dr. Namitha Naduvath Kalathil
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A Rare Case of Inflammatory Fibroid Polyp resulting in an Unusual Cause of Adult Ileo-Ileal Intussusception treated successfully at Aster Hospital, Al Qusais, Dubai

PRESENTATION

- 44-year-old female presented with recurrent right lower quadrant pain and vomiting for 4 months
- Medical history of Type 2 Diabetes Mellitus, dyslipidaemia and hypertension, on medication
- No family history of medical illness

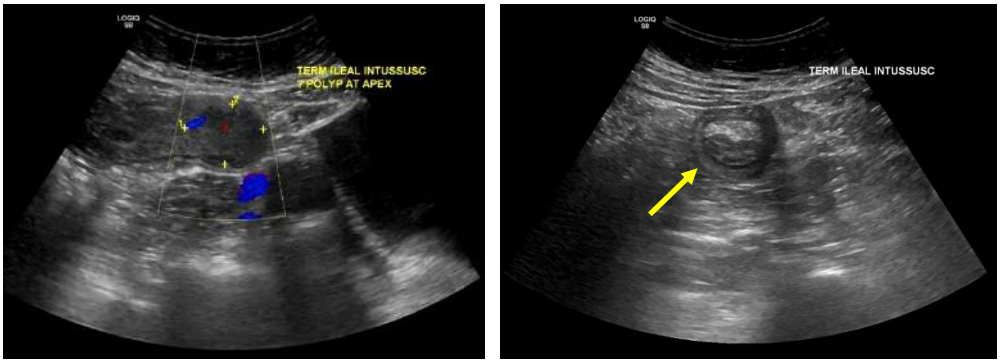
FINDINGS

On examination:

- Vitals were stable with no signs of dehydration
- Per abdomen soft, mild distention +
- Diffuse tenderness +
- No palpable mass
- No guarding/rigidity
- Bowel sounds: Present

Ultrasonography showed:

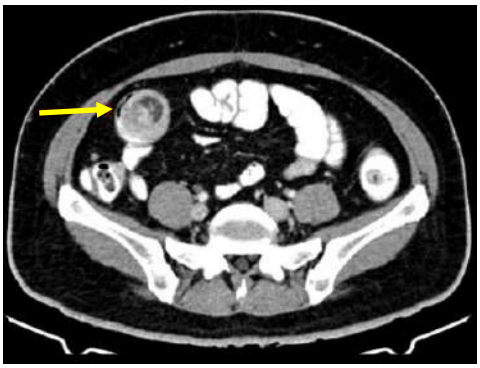
- A focus of intussusception in the terminal ileum with no proximal bowel dilatation and a mildly echogenic lesion with central vascularity measuring 3 x 2.3 cm in the apical region, possibly a lipoma.
- CECT abdomen was advised for further characterization of the lead point.



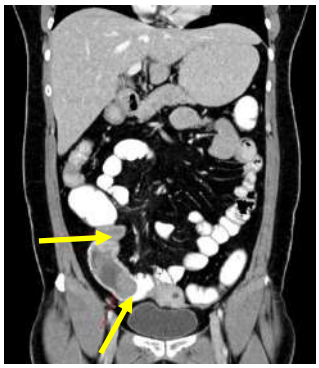
Terminal ileal intussusception

CECT Abdomen showed:

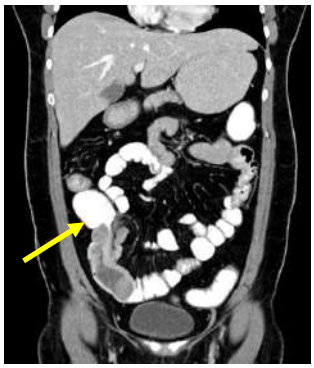
- A short segment of intussusception in the proximal ileum in right iliac fossa with presence of a well-defined hypodense elongated mass lesion as a lead point. It measured ~ 6.2 x 2.7 x 2.8 cm and showed heterogeneous enhancement.
- Intervening areas of macroscopic fat densities noted.
- No foci of calcification or ulceration.
- No surrounding fat stranding or lymphadenopathy.
- Short segment of mild (up to 4.8 cm diameter) bowel dilatation noted just proximal to the level of intussusception.
- The involved bowel loop showed no features of ischaemia.
- Features of **Ileo-Ileal Intussusception** with possibly lipoma variant mass as lead point.



Axial image with intussusception



Coronal image with mass as the lead point

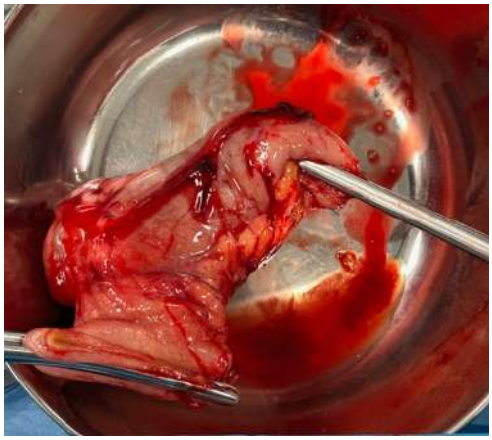


Coronal image depicting mild proximal small bowel dilatation

MANAGEMENT

Care plan was explained to the patient and bystanders and was posted for Diagnostic Laparoscopy +/- Reduction of Intussusception.

- After obtaining consent, the patient was prepped and draped.
- A pneumoperitoneum was created with a Veress needle at Palmer's point.
- Two 5 mm at right MC line and lower midline were inserted.
- As the intussusception was irreducible, midline laparotomy was done.
- Resection of the intussusception with the lead point was done with a 5 cm margin.
- Then an end-to-end anastomosis was done in two layers with 2-0 Vicryl and 3-0 silk.
- Midline was closed with a no. 1 PDS loop.



Resected specimen with ileo-ileal intussusception

POST PROCEDURE

The postoperative period was uneventful. She was afebrile, and her vitals were stable. She was tolerating all the oral feeds, and her bowels were also open.

HISTOPATHOLOGY EXAMINATION

Gross Description:

Received in formalin, labelled with the patient's name and number and designated on the container as from "Ilea Intussusception", consisted of part of the ileum measuring 12 cm long showing in the middle portion, the presence of a polypoid mass within the lumen and protruding to the outer surface measuring 5.5 x 5 x 3.5 cm in greatest dimensions. On cutting, it showed greyish-white homogeneous cut surface. No lymph node was identified.

Microscopic Description:

Sections showed well demarcated tumour composed of the proliferation of bland mesenchymal cells on a background with small vessel proliferation, part of them were ectatic, with little collagen, inflammatory infiltrates rich in eosinophils and variable degrees of oedema. This tumoral tissue was extending through the whole wall. No enlarged lymph nodes were identified.

Interpretation:

- Clinically "Partial Ileectomy"
- Histopathological findings consistent with **Inflammatory Fibroid Polyp**
- No evidence of Dysplasia

DISCUSSION

Inflammatory Fibroid Polyp (IFP) is a rare, usually solitary, intraluminal polypoid benign tumour that can affect any part of the gastrointestinal (GI) tract, although in most cases, it affects the stomach. This lesion is characterised by proliferation of highly vascular fibrous tissue and infiltration by a variable number of different inflammatory cells.

A plethora of different names have been suggested to describe IFP, such as eosinophilic granuloma, granuloblastoma or gastric fibroma with eosinophilic infiltration, granuloma with eosinophils, hemangiopericytoma, inflammatory fibroid tumour, and inflammatory pseudotumor. The many possible names reflect our ignorance of the exact mechanism of IFP development and the different hypotheses

on the aetiology of IFP. Local infection, allergic reaction, autoimmune processes or excessive host response to an unknown stimulus have all been described as possible causes of IFP development. Due to its unknown aetiology, the malignant potential of what is otherwise described as a benign tumour is currently under debate.

Immunohistologically, IFPs are characterised by spindle cells with unclear origin, positive for CD34 and vimentin, and negative for CD117. The overexpression of platelet-derived growth factor receptor alpha (PDGFRA) and oncogenic PDGFRA mutations in most analysed IFP suggest that this tumour might develop through activated PDGFRA. A variety of clinical signs and symptoms are linked to IFP, mainly GI bleeding or abdominal pain, but the clinical presentation may significantly vary, and it mimics other pathologies.

In a study published in PubMed, pain was recorded to be the most frequent symptom (17.0%), closely followed by acute abdominal symptoms (12.8%). Other frequent symptoms were vomiting (7.8%), nausea (5.4%), lower GI bleeding (4.5%), abdominal distension (3.5%) and anaemia (3.5%). Several patients (7.0%) were asymptomatic, and the findings were incidental in 10 cases (2.4%). In our case, the patient presented with intussusception.

The patient was taken up for a diagnostic laparoscopy, which was converted to an open laparotomy as the intussusception was irreducible. A limited resection of the affected proximal ileum was resected, and the patient had an uneventful recovery.

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Coordinated Clinical Approach for Diagnosis and Long-Term Management of Adult ADHD

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INTRODUCTION

Attention deficit hyperactivity disorder (ADHD) is a complex neurodevelopmental disorder associated with age-inappropriate hyperactivity, impulsivity, and inattention, with a tendency to critically affect the lives of affected individuals, families, caregivers, and the community (1). Although this common psychiatric disorder is mainly reported in school-age children, ADHD can persist in later stages of life as well (1). Studies estimate a worldwide prevalence of 5.3-7.1% in children and 4.4-5.0% in adults (2). Translating to more than 360 million affected adults globally, a systematic review and meta-analysis estimated the prevalence of persistent adult ADHD at 2.58% and symptomatic adult ADHD at 6.76% (3).

Even though ADHD was originally considered a childhood disorder, it is now known that the impairments caused by ADHD frequently continue well into adulthood in about 65% patients (2). Untreated adult ADHD leads to functional difficulties across social, educational, and professional areas, and is linked to greater risks of accidents, premature mortality, and a decline in quality of life (2,4). It can affect many dimensions of life, often contributing to poor academic performance, unemployment, unstable relationships, and involvement in criminal activities (3).

The Diagnostic and Statistical Manual of Mental Disorders (DSM), widely used for diagnosing adult ADHD, requires a childhood-onset and retrospective questioning on the symptoms and related impairments noted in childhood in the presence of a family member (3,5). However, in most cases, such information may be unavailable, leading to delays in the diagnosis of adult ADHD (5). This issue is compounded by the lack of epidemiological studies in adult patients, owing to the absence of clearly established diagnostic criteria (3).

Considering this, the recent 2023 National Institute for Health and Care Excellence (NICE) guidelines emphasise the need for a multidisciplinary team for the diagnosis and management of ADHD to ensure a smooth transition between child and adult services (6).

ADVANCED DIAGNOSTIC FRAMEWORK FOR ADULT ADHD WITH COMORBIDITY CONSIDERATIONS

A full psychiatric and neurodevelopmental review aiding the assessment of adult ADHD often includes structured interviews, discussions on developmental history and functional impairment, and comorbidity evaluation (7). Accurate assessment is ensured by the use of validated tools, corroborative history, and a systematic assessment of psychiatric and medical conditions (5).

Various psychometric tools that assist practitioners in the diagnosis of adult ADHD are summarised in Figure 1 (5).

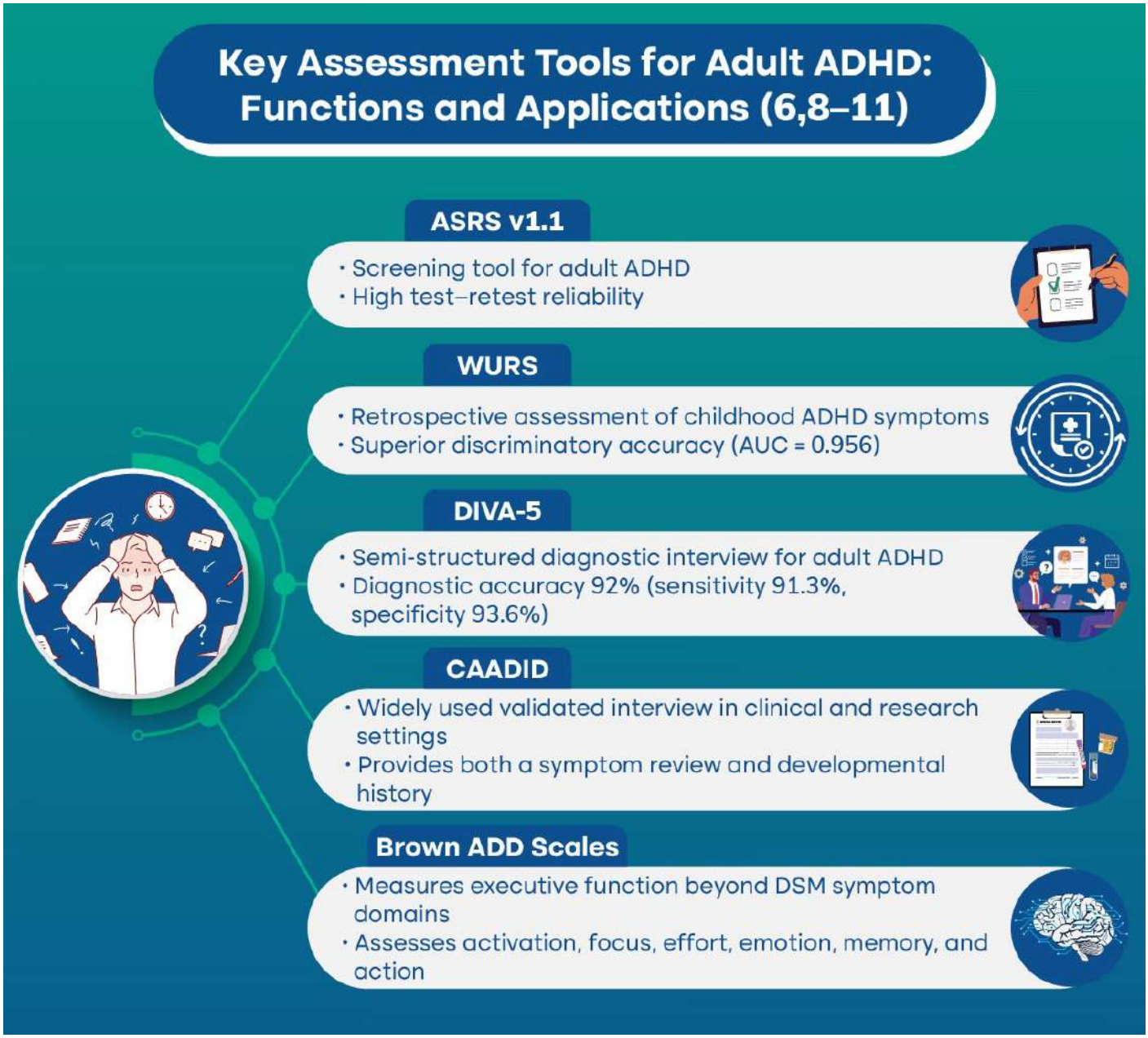


Figure 1. Clinical utility of common diagnostic instruments used for adult ADHD assessment (6,8–11)

(Abbreviations – ASRS v1.1: Adult ADHD Self-Report scale version 1.1; WURS: Wender Utah rating scale; DIVA-5: Diagnostic Interview for ADHD in adults, 5th edition; CAADID: Conners' Adult ADHD Diagnostic Interview for DSM-IV; Brown ADD scales: Brown Attention-Deficit Disorder scales)

A comprehensive patient history, along with objective/third-party information, plays a crucial role in identifying the nature and severity of impairment in adult ADHD (7,12). Parents and teachers, or any other evidence of childhood behaviour (school reports or employment records), can be useful to get an objective insight into the patient's childhood (12,13). Obtaining patient history through retrospective informants is often debated and must always be interpreted with caution (7).

As ADHD symptoms often overlap with those of other psychiatric or medical conditions, making a differential diagnosis is an important step in assessment (14). Studies note that up to 80% adults with ADHD have an additional psychiatric illness, most commonly mood or anxiety disorders, substance misuse, and personality disorders, being the most prevalent (15). ADHD also shows high comorbidity with anxiety and depressive

disorders due to overlapping and interacting symptoms (16). Medical causes such as thyroid dysfunction and sleep disorders may also contribute to attention or behavioural symptoms, and should be considered during evaluation (17). In such cases, the most impairing disorder should be treated first (15).

Another important criterion for diagnosis and assessment of adult ADHD is functional impairment (18). Patients reporting significant impairment in more than one setting – among education, work, or relationships – are assessed (18). The Weiss Functional Impairment Rating Scale (WFIRS) can be used with other evidence to assess impairment in suspected cases (7,19).

High-quality assessment must align with established standards outlined by global guidelines (19).

- **Adult ADHD Assessment Quality Assurance Standard (AQAS)** - defines minimum requirements, including psychiatric review, corroborated developmental history, structured interviews, and functional assessment (19).
- **2018 NICE Guideline (NG87)** - recommends multidisciplinary services and clear referral pathways into specialist care (6).
- **Royal College of Psychiatrists' CR235 Guidance** - highlights corroborated developmental history, structured interviews, and systematic evaluation of comorbidities and differential diagnoses as integral to best practice (12).

True remission requires both symptomatic and functional recovery (14).

TAILORED PHARMACOLOGICAL AND PSYCHOSOCIAL INTERVENTIONS FOR ADULT ADHD MANAGEMENT

Stimulants are considered the first-line pharmacological treatment for adult patients with ADHD (5). The NICE guideline recommends lisdexamfetamine or methylphenidate as the first-line options, and switching to non-stimulant medications such as atomoxetine if patients don't respond or tolerate stimulants (5,6). Other non-stimulant options, bupropion, viloxazine, and alpha-2 agonists, are mostly considered for patients with other comorbid conditions (20). For example, stimulants are often linked to modest yet clinically relevant elevations in blood pressure and heart rate, which makes regular cardiovascular monitoring necessary (19,21).

Figure 2 presents a concise overview of the comparative efficacy, tolerability, and comorbidity-specific considerations for these pharmacological agents.

Comparative Overview of Adult ADHD Pharmacotherapies (5,20–25)

Drug	Clinical Efficacy	Adverse Effects	Comorbidity Considerations
First-Line Stimulants			
Methylphenidate	• Reduces core ADHD symptoms in adults	• Insomnia • Decreased appetite • Increase in BP/HR	• Risk of misuse/diversion • Caution in cardiovascular disease
Amphetamines / Lisdexamfetamine	• Among the most efficacious agents in adults		• Avoid in stimulant use disorder • Caution in cardiovascular disease
Non-Stimulant Alternatives			
Atomoxetine	• Comparable efficacy to methylphenidate	• Nausea • Insomnia • Weight/BP changes	• Preferred in ADHD + SUD • Option in anxiety or tics
Bupropion	• Shows some benefit but with less robust evidence and wide confidence intervals.	• Insomnia • Dry mouth • Seizure risk in predisposed patients	• Option in ADHD + depression • When stimulants unsuitable
Viloxazine XR	• Proven effective in adult ADHD in recent trials.	• GI upset • Somnolence • Fatigue	• Consider when ADHD co-exists with depression

Figure 2. Comparative overview of adult ADHD pharmacotherapies (5,20–25)

(Abbreviations – ADHD: attention-deficit hyperactivity disorder; BP: blood pressure; HR: heart rate; GI: gastrointestinal; XR: extended release; SUD: substance use disorder)

In addition to pharmacological treatment, psychosocial interventions are recommended as part of a multimodal treatment approach for adult ADHD (5).

1. **Cognitive behavioural therapy (CBT)-based structured programmes** focus on organisational skills, planning, and time management, while developing durable problem-solving strategies and emotional regulation (24,26).
2. **Other supportive approaches,** including social skills training, sleep and physical activity interventions, meditation, and hypnotherapy, can also help (20).
3. **Targeted workplace interventions** improve organisational skills, productivity, and daily functioning in adults with ADHD (27).

4. **Cognitive remediation** involves training the working memory through computerised tools like COGMED (5). This technique targets the underlying neurocognitive processes of ADHD rather than focusing directly on functional outcomes (16). Studies have, however, reported variable benefits following such training (5).
5. Another novel approach, **neurofeedback,** has shown promising preliminary results in reducing ADHD symptoms but needs to be evaluated further before recommending it in clinical practice (5).

Certain patient profiles require careful consideration and adapted treatment strategies. ADHD medications are safe to use in pregnant patients, as in utero exposure does not increase the risk of long-term neurodevelopmental disorders in the offspring (28). In epilepsy patients, the use of OROS-methylphenidate does not significantly worsen the condition; however, higher dosages may carry a somewhat greater risk of seizures occurring daily (25).

The NICE treatment guidelines for adult ADHD recommend integrating pharmacological and non-pharmacological interventions to address core symptoms, improve daily functioning, while tailoring treatment to individual comorbidities and patient needs (5).

INTEGRATIVE CLINICAL PATHWAY FOR DIAGNOSIS AND LONG-TERM MANAGEMENT OF ADULT ADHD

Accurate recognition and structured care for ADHD patients is important due to its persistence into adulthood as well as due to its association with social, educational, and occupational impairments (5). This can be done through coordinated and patient-centred pathways that extend beyond isolated assessments or symptomatic treatment (1).

The AQAS specifies a comprehensive psychiatric review, structured interviews with real-life examples, and a detailed evaluation of impairment as minimum requirements for assessment (19). Complementing this, NICE guideline NG87 recommends that services for adults with ADHD be multidisciplinary and operate within clear referral pathways to ensure consistency and quality of care (6). Further emphasising best practice, the Royal College of Psychiatrists' CR235 guidance highlights the critical role of obtaining a corroborated developmental history, conducting structured interviews, and systematically evaluating comorbidities and differential diagnoses during the assessment process (12).

Figure 3 shows a stepwise approach for screening, referral, diagnostic confirmation, and initiation of management within specialist services.

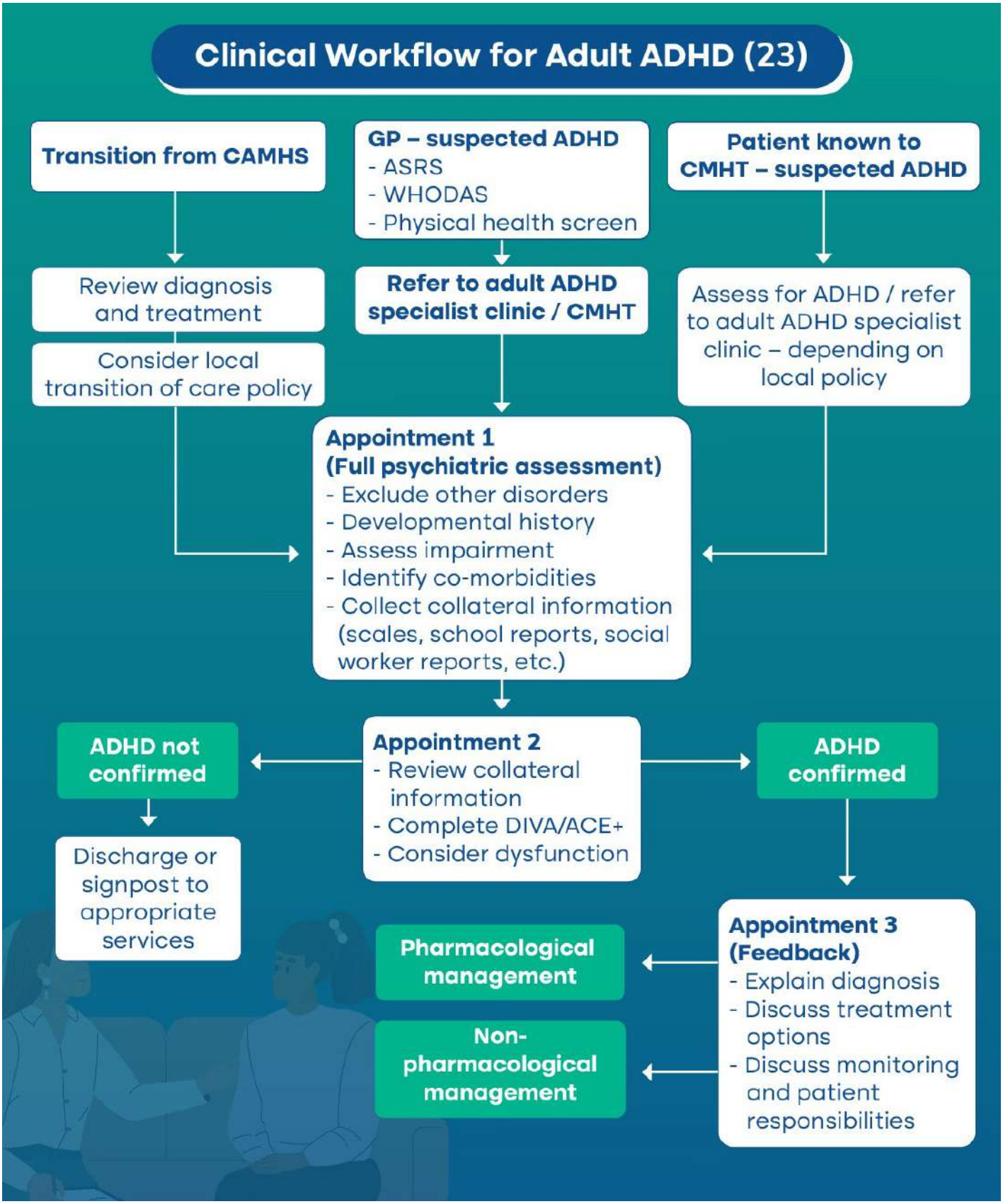
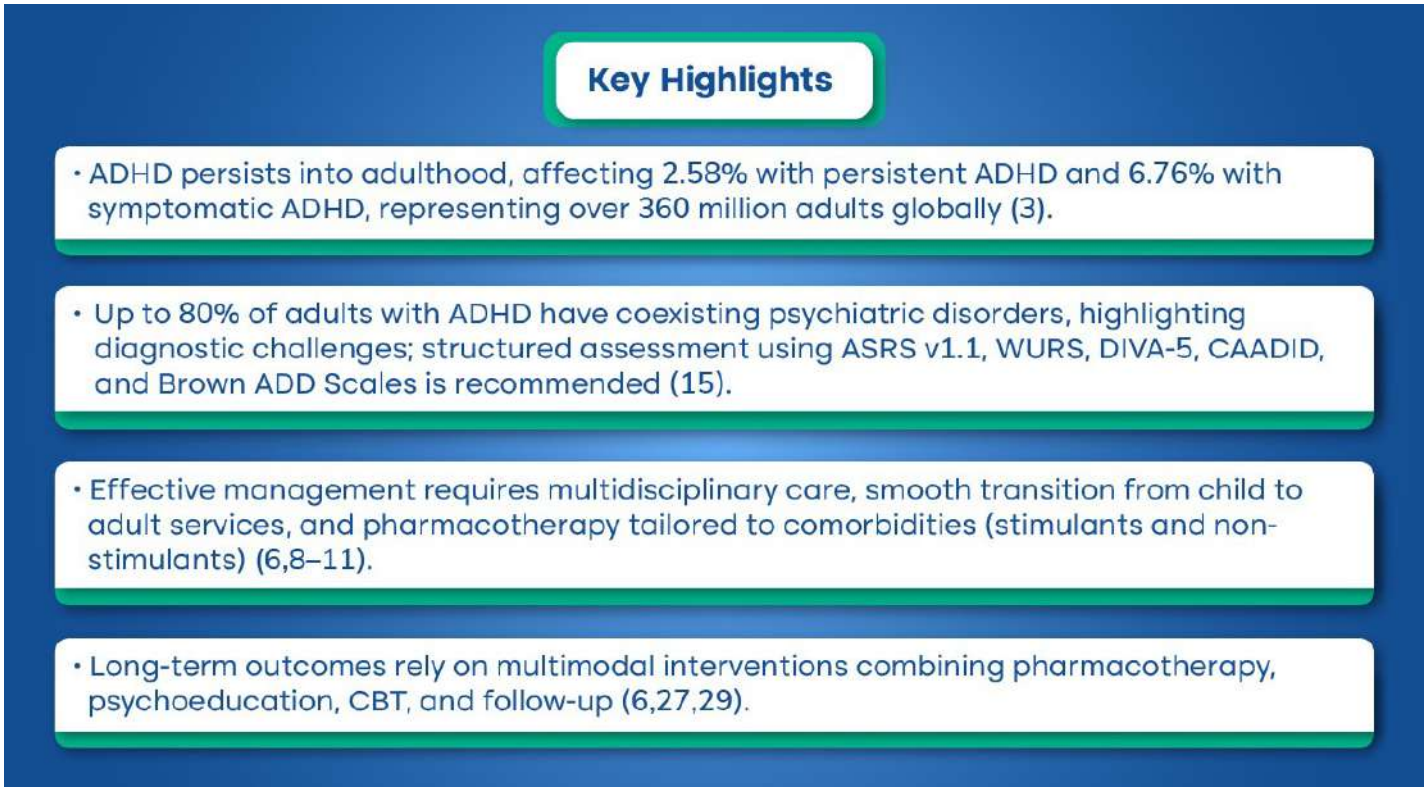


Figure 3. Clinical workflow for adult ADHD diagnosis and care transition (23)

(Abbreviations – CAMHS: Child and Adolescent Mental Health Services; GP: general practitioner; ADHD: attention-deficit hyperactivity disorder; ASRS: Adult ADHD Self-Report scale; WHODAS: World Health Organisation Disability Assessment Schedule; CMHT: Community Mental Health Team; DIVA: Diagnostic Interview for ADHD in adults; ACE+: ADHD Child Evaluation Plus)

International consensus underscores that effective ADHD management involves a combination of pharmacological and non-pharmacological interventions (4). Among psychosocial strategies, psychoeducation and cognitive behavioural therapy are especially important (29). Additionally, group interventions offer a cost-effective option that enhances accessibility and clinical delivery (27).

A coordinated approach must involve not just treatment initiation but also a structured follow-up process (19). The CR235 guidance encourages best practices such as comprehensive post-diagnostic care, promotion of self-management, and shared-care arrangements between primary and specialist services (12). These measures can help ensure a smooth transition across healthcare services and support sustainable, patient-centred care throughout the lifespan (23).



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Monkeypox (Mpox) mimicking as Pharyngotonsillitis: A case-based diagnostic puzzle treated effectively at Aster Hospital, Muhaisnah, Dubai

PRESENTATION

A 29-year-old male, with no known comorbidities or relevant family history, presented to the Emergency Department with complaints of high-grade fever, throat pain, dysphagia, and dry cough for two days, associated with myalgia and headache.

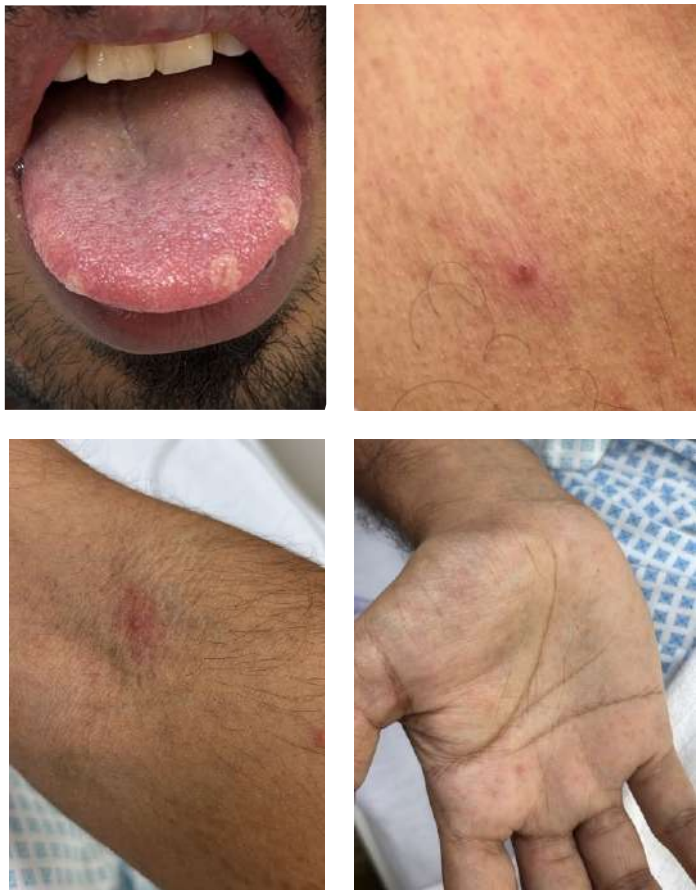
FINDINGS

On examination:

- Conscious and oriented
- Vitals: HR:78/min, BP: 122/76 mmHg, SpO2: 98% on room air, Temp: 36.9°C
- Oropharynx: Bilateral tonsillar swelling with significant exudates
- Respiratory: Bilateral conducted sounds with few crepitations in the left infrascapular region
- Nervous System: No meningeal signs or neurological deficits

On day 2 of admission, the patient developed:

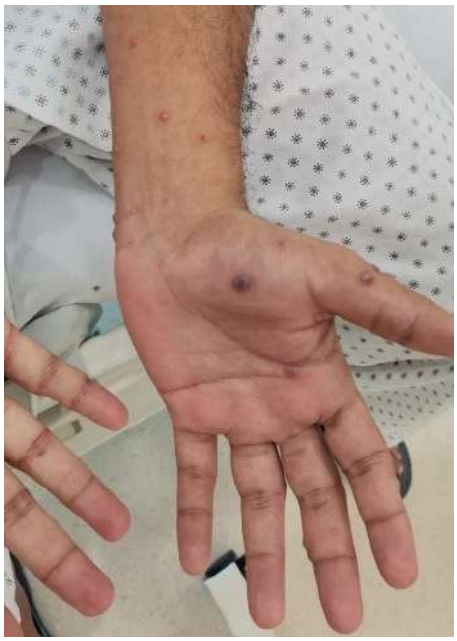
- Multiple painful oral ulcers
- Diffuse maculopapular rash over the chest, back, upper limbs, and notably the palms and soles
- Tender multiple cervical lymphadenopathy
- Increasing odynophagia with pooling of oral secretions



COURSE

A possible drug reaction was initially considered, and the antibiotic regime was accordingly modified.

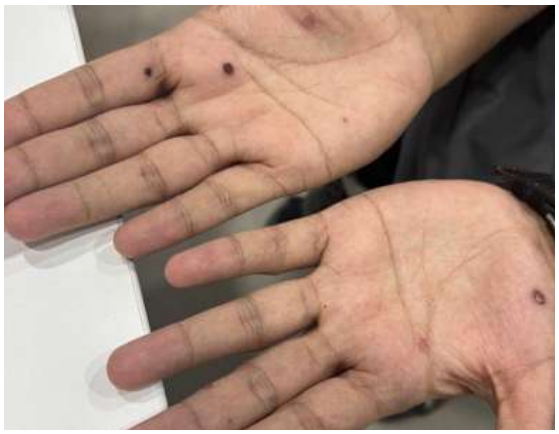
However, over the next 24-48 hours, the rash evolved into multiple vesicular lesions, and a solitary papulovesicular lesion on the scrotum was noted. Notably, the appearance of a solitary papulovesicular lesion on the scrotum with a few vesiculopustular lesions over the body prompted consideration of Monkeypox and led to diagnostic confirmation via PCR.



Day 4

The patient was immediately started on Cap. Doxycycline 100 mg twice daily, after which the patient improved drastically and was discharged in stable condition.

He denied any history of high-risk sexual behaviour, including homosexual activity or illegal sexual contact.



Day 14

DISCUSSION

Monkeypox (Mpox) is a zoonotic viral infection caused by the Monkeypox virus (MPXV), an orthopoxvirus related to smallpox. Historically endemic to Central and West Africa, it caused a global outbreak in 2022, notably affecting non-endemic regions.

Transmission:

Traditional: Animal-to-human via contact with infected animals (e.g., rodents, primates); human-to-human via respiratory droplets, body fluids, or lesion material.

Recent outbreaks: Primarily via close physical or sexual contact, particularly among men who have sex with men (MSM).

Typical Presentation:

Incubation: 5–21 days

Prodrome: Fever, malaise, and lymphadenopathy (a key differentiator from smallpox)

Rash: Evolves through macular → papular → vesicular → pustular → crusting stages; typically begins on the face and spreads centrifugally, including palms and soles.

Atypical Presentation:

Recent outbreaks revealed several atypical patterns, especially in non-endemic settings:

- Localized Lesions
- Limited to genital, perianal, or oral regions; may mimic STIs (e.g., herpes, syphilis). Lesions may appear at different stages or lack classic progression.
- No prodrome
- Rash may be the only symptom, complicating early diagnosis.
- Anorectal involvement: Severe rectal pain, tenesmus, and discharge reported, particularly in MSM; may occur without skin lesions, mimicking IBD or STIs.
- Oropharyngeal & ocular involvement: Sore throat, tonsillar swelling, conjunctivitis, and even keratitis have been reported.

Diagnostics:

- PCR testing from lesion material is the gold standard.
- A broader sampling (e.g., throat or rectal swabs) may be necessary in atypical cases.
- Rule out STI coinfections in genital presentations.

Management:

- Primarily supportive care
- Tecovirimat (TPOXX) is used in severe or high-risk cases (FDA approved).
- Isolation until lesions crust over and new skin forms.
- Role of Doxycycline: Though not antiviral, doxycycline has anti-inflammatory and antioxidant properties. It may aid healing and prevent secondary bacterial infections, potentially benefiting MPXV patients per emerging evidence.

CONCLUSION

Mpox can present in unexpected ways. In this case, oropharyngeal symptoms mimicking bacterial tonsillitis delayed the diagnosis. Recognising characteristic signs and investigations can lead to timely diagnosis and good outcomes in Mpox. Early diagnosis not only improves patient outcomes but also supports public health measures in controlling the spread of infection in non-endemic settings.

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