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Review Article

Surgery Section

Wandering Spleen: A Narrative Review of Clinical Spectrum, Pathogenesis, Emerging Diagnostics, and Management Approaches

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ABSTRACT

Wandering Spleen (WS) is an uncommon clinical disorder in which the spleen is abnormally mobile as a result of either congenital ligamentous anomalies or acquired laxity and thus put at risk of torsion, infarction and variable abdominal symptoms. WS has a bimodal distribution in the age group, which includes children and women of reproductive age and is characterised by non specific symptoms, such as a mobile mass in the abdomen or pelvis, intermittent pain, in few severe cases, an acute abdomen. Diagnosis is not easily done and is dependent much on imaging methods like ultrasound with Doppler, Contrast-Enhanced Computed Tomography (CECT), nuclear scintigraphy, dynamic Magnetic Resonance Imaging (MRI) and newer modalities like Contrast-Enhanced Ultrasound (CEUS). Spleen preservation through splenopexy which is laparoscopic, open, or robotically done is prioritised in the management and splenectomy is used in case of non viable spleen or infarction or sepsis complications. It is important to prevent severe complications by recognising them early and applying proper intervention. The present review will outline the pathogenesis, clinical manifestation, emerging diagnostic methods and the modern management of WS including the most minimal invasive and spleen-sparing techniques.

Keywords: Abdominal pain, Infarction, Laxity, Paediatric, Torsion

INTRODUCTION

The spleen is abnormally mobile and no longer located in its normal position in the left upper-quadrant of the abdomen due to the absence, laxity, or elongated splenic suspensory ligaments, known as WS [1]. This abnormal mobility, most commonly due to malformation of the dorsal mesogastrium, but sometimes acquired (following pregnancy, splenomegaly or connective-tissue laxity) forms an elongated vascular pedicle making the organ susceptible to torsion, infarction, as well as intermittent or acute abdominal manifestations [2,3]. It is a rare clinical entity for which incidence has been estimated less than 0.5 percent of splenectomies thus underscoring its infrequent occurrence in surgical practice [4]. Due to its potentially asymptomatic nature, the real prevalence is definitely larger than the reported cases but no major population-based studies have managed to quantify it highlighting about an important area for future epidemiological research [4]. The WS is bimodal in terms of age with peaks in young children (<10 years) and young women of reproductive age [3,4]. In paediatric patients there is usually no significant gender difference, with similar incidence in boys and girls; however, in adults cases, WS is predominantly seen in females having reported female-to-male ratios of approximately 7:1 likely related to hormonal influences as well as ligamentous laxity associated with pregnancy and multiparity [3,5]. Epidemiological data on racial-ethnic variation in WS are limited as most literature consists of case reports and small series without stratification of race or ethnicity thus highlighting a large gap which warrants further population-based studies [3,4]. Due to the lack of specific symptoms, making the diagnosis has been a major diagnostic challenge that has recently depended heavily on imaging (ultrasound, CT, MR) to define the position of the spleen, its viability and its torsion of the vascular pedicle [6].

One of the first known record of a spleen with abnormal mobility was an incidental observation during an autopsy in 1667 which was performed by Johannes van Horne [7,8]. Jozef Dietl in 1854, however, first described a clinical picture of a displaced spleen in detail [7]. The surgical management was developed in the 19th century with the first splenectomy to treat a WS in the 1870s and the first planned splenfixation (splenopexy) in 1890s when surgeons developed and refined many methods of fixing the spleen [7]. Contemporary historical analyses sum up these developments and highlight the role of enhanced knowledge of the anatomy and radiology in diagnosis and changing the management to spleen-preserving surgeries (splenopexy) in cases where the organ is viable [7]. The purpose of the present article is to present an accurate understanding of WS, clinical presentation, pathogenesis and complications. It also attempts to highlight existing and new diagnostic modalities with a focus on the development of imaging methods. Moreover, it examines modern management techniques, such as surgical and non surgical ones, with emphasis on spleen-sparing interventions.

Clinical Features and Symptomatology of Wandering Spleen (WS)

The WS is characterised by a wide range of symptoms, which can be completely asymptomatic or the acute abdominal emergency [9]. Preliminary medical attention is a result of a palpable mass in the abdominal or pelvic region by a number of patients [10]. This mass can be mobile and changes its position, sometimes it will be felt in the lower abdomen or pelvis, as opposed to the typical left upper quadrant [9,10]. Others have vague, intermittent or chronic abdominal discomfort which is dull or colicky pain, could be in relation to partial or recurrent torsion and spontaneous detorsion of the vascular pedicle of the spleen [9]. Symptoms in this category that have been reported also include nausea, abdominal distension or fullness; in chronic or subacute manifestations [9].

The symptoms are more dramatic in the acute presentations [11]. The patients might experience severe and sudden onset abdominal pain (initially localised but later diffuse), along with evidence of peritoneal irritation in the form of tenderness, guarding (with or without rebound) [11]. Vomiting and systemic symptoms such as fever, tachycardia and leukocytosis might be present particularly when infarction or necrosis of the splenic tissue has developed [11]. In few other cases, bowel obstruction-like symptoms (constipation, distension) can be present, either through mass effect or vascular compromise [12]. In children or infants or very young patients, the manifestation may be less apparent or non specific, which makes it difficult to make a diagnosis [1,4]. Characteristic symptoms and clinical findings in WS are mentioned in [Table/Fig-1] [1,4,9-12]. Clinical features of WS are represented in [Table/Fig-2].

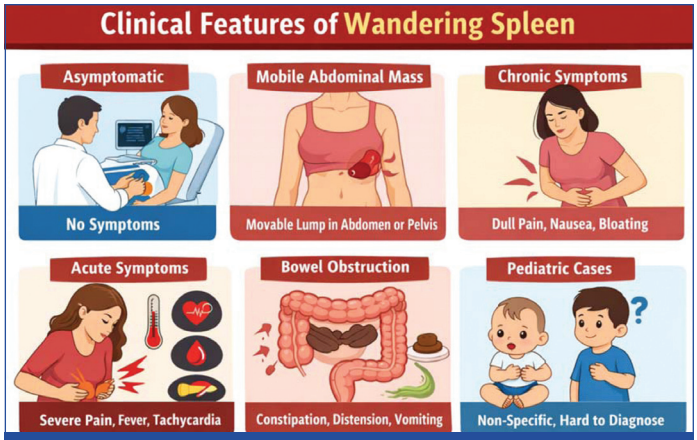
Presentation type	Clinical features/ symptoms	Possible underlying mechanism/ explanation	References
Asymptomatic	• Incidentally detected during imaging or physical examination	• No torsion or vascular compromise	[9]
Chronic/ subacute presentation	• Palpable, mobile abdominal or pelvic mass (changes position with posture) • Vague, intermittent, or chronic abdominal discomfort (dull or colicky pain) • Nausea, abdominal distension, or fullness	• Partial or recurrent torsion and spontaneous detorsion of splenic vascular pedicle • Intermittent congestion or partial ischaemia of spleen	[9,10]
Acute presentation	• Sudden, severe abdominal pain (initially localised, later diffuse) • Signs of peritoneal irritation: tenderness, guarding, rebound tenderness • Vomiting • Systemic symptoms: fever, tachycardia, leukocytosis	• Acute torsion of splenic pedicle leading to infarction or necrosis of splenic tissue	[11]
Bowel obstruction-like presentation	• Abdominal distension • Constipation • Vomiting	• Mass effect of enlarged/torsed spleen • Secondary bowel vascular compromise	[12]
Paediatric presentation	• Non specific or subtle abdominal complaints • Palpable mass may be missed clinically	• Difficulty in clinical assessment • Delayed or missed diagnosis in infants or young children	[1,4]

[Table/Fig-1]: Characteristic symptoms and clinical findings in Wandering Spleen (WS) [1,4,9-12].

Developmental and Acquired Pathogenesis of Wandering Spleen (WS)

In embryonic development, the spleen forms as a condensation of mesenchyme within dorsal mesogastrium; the dorsal mesogastrium becomes attached to the posterior abdominal wall to make the gastrosplenic, splenorenal (lienorenal) and other peritoneal attachments that hold the spleen in place properly in the left hypochondrium [13]. WS is caused by inability of the dorsal mesogastrium to fuse with the posterior abdominal wall during weeks 5-6 of foetal development thus compromising the formation of major peritoneal ligaments such as gastrosplenic, splenorenal and phrenicocolic ligaments [13].

There is a superimposition of acquired processes over a congenital predisposition and results in enhancing ligamentous laxity or allow a previously well-fixed spleen to become hypermobile [14]. Most frequently reported acquired precipitating factors are splenomegaly (resulting into stretching of ligaments because of splenic weight gain), repeated pregnancies and multiparity (hormonal and mechanical



[Table/Fig-2]: Clinical Features of Wandering Spleen (WS).
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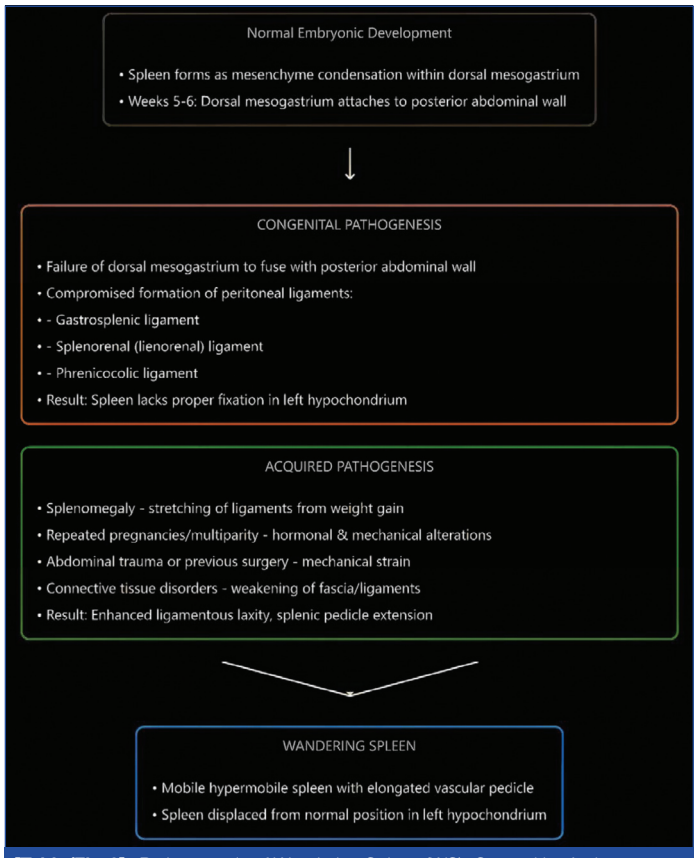
alterations that loosen peritoneal supports) and abdominal trauma or previous surgery (mechanical strain) and systemic connective-tissue disorders that cause weakening of fascia/ligamentous tissues [14,15]. These gained alterations extend the splenic pedicle and convert the spleen into a mobile one [14]. Pathogenesis of WS is depicted through [Table/Fig-3].



[Table/Fig-3]: Pathogenesis of Wandering Spleen (WS); Created by Authors.

Complications and Associated Sequelae of Wandering Spleen (WS)

The WS predisposes the organ to torsion of its vascular pedicle which is by far the most dreaded complication [14]. Twisting of the splenic vessels causes vascular occlusion, ischaemia and splenic parenchymal infarction in most of the reported cases [14,16]. The necrotic infarcted spleen can even perforate and cause gangrene and result into peritonitis [16]. In other cases, associated thrombosis of the splenic vein has been also reported in the predisposition of torsion, which only exacerbates congestion of the venous system and aggravates the ischaemic damage [16,17].

In addition to direct vascular compromise, there are secondary sequelae of compression or inflammation of the adjacent organs [16]. Mechanical entrapment or displacement by the ectopic spleen

may result in bowel obstruction which can mimic an acute surgical abdomen [18]. Another complication that has been documented is the formation of splenic abscess; which happens in infarcted spleens that are not treated [14]. In uncommon cases, the tail of the pancreas can be injured as a result of necrosis or inflammatory diffusion or cause pancreatitis [19]. Rupture or haemorrhage can also occur as a result of the weakening of the structure of the infarcted spleen, which causes internal bleeding and acute haemodynamic instability [20].

Aetiological and Anatomical Classification of Wandering Spleen (WS)

The WS is aetiologically, classified as congenital and acquired [21]. Congenital WS is caused by developmental defects during embryonic formation and causes the loss or laxity of the splenic ligaments that normally fix the spleen in the left upper quadrant of abdomen [22]. Conversely, acquired WS occurs later in life because of the conditions that cause the supporting ligaments to be weakened, e.g., trauma to the abdomen, hormonal fluctuations during pregnancy, or surgery [21,22]. It is important to identify the underlying aetiology because it may have an impact on clinical presentation and the management strategy [21].

The WS can be categorised anatomically, depending on the axis about which a torsion of the spleen occurs [23]. Organoaxial rotation takes place along the longitudinal axis of the spleen whereas mesenteroaxial rotation takes place along a transversal axis [23,24]. Combined rotation pattern entails components of organoaxial and mesenteroaxial torsion resulting in more complicated anatomical distortion [23]. Knowledge of such anatomical classifications is important to the clinicians, as they help them diagnose correctly and decide on the appropriate surgical procedures to be used including splenopexy or splenectomy to avoid complications related to this rare disease [23,24].

Evolving Diagnostic Strategies for Wandering Spleen (WS)

Diagnosis of WS is difficult to be made, mainly due to some lack of specificity of clinical manifestation [1]. The patients can be introduced with the palpable and mobile mass of the abdominal or pelvic mass, with or without intermittent or acute pain in the abdominal region or even with the features of an acute abdomen, in case of the torsion of the splenic pedicle [1]. Laboratory results mostly tend to be non diagnostic [25]. Clinicians do not usually think of WS until it is foreshadowed by imaging due to its rarity and changeable symptoms [25].

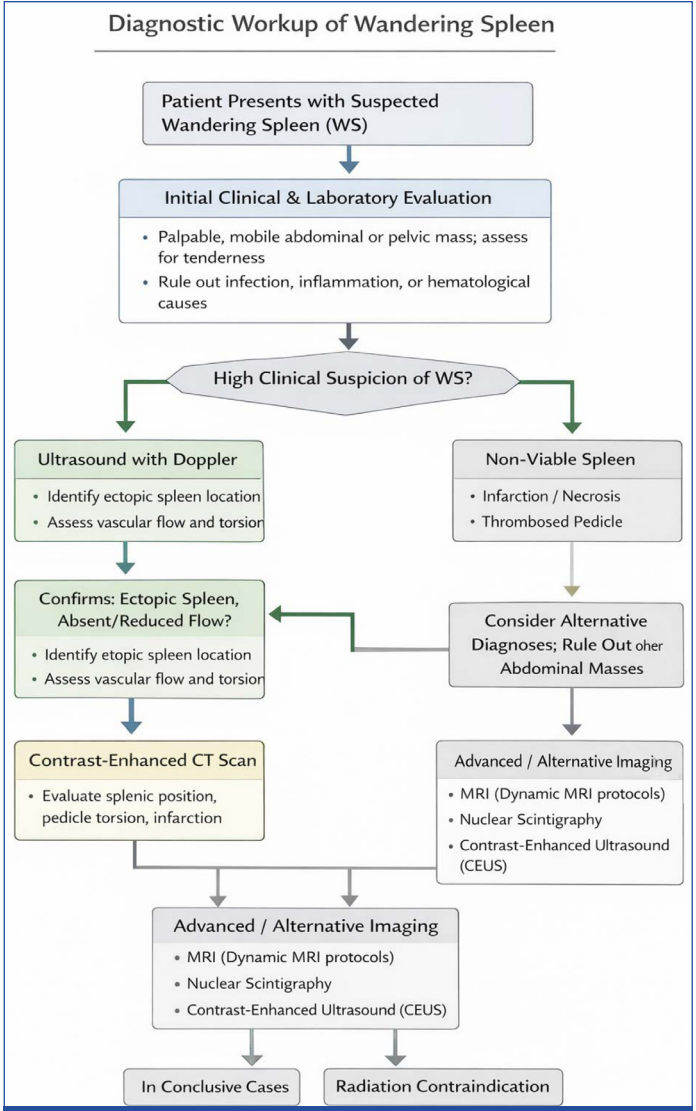
The first-line tool of diagnosis is usually ultrasound with Doppler, which can identify an ectopic spleen and evaluate vascular flow and may have no or reduced flow in case of torsion [26]. The sensitivity of Doppler ultrasound in published case series ranges between approximately 70-85% although modality remains operator-dependent as well as limited by bowel gas or obesity [26,27]. Specifically, CECT is useful: during torsion, CT can identify an ectopic, ovoid or comma-shaped mass in the spleen, a whirl sign observed with twisted splenic vessels and non enhancement of spleen parenchyma, as well as can be used to define the vascular pedicle and viability of the spleen [28]. Retrospective analyses report diagnostic accuracy of CECT exceeding 90-95% in acute torsion thus making it most robustly supported imaging modality in emergency clinical settings [28,29]. In some cases, the functional status and vascular supply can be supported with the help of nuclear scintigraphy or MR angiography, especially where the Doppler results are inconclusive [30].

A particularly interesting recent development is the application of dynamic MRI in the diagnosis of WS and in searching the postoperative outcomes including the situation following a splenopexy [31]. In a reported case of a young woman, dynamic MRI (with balanced steady-state free precession sequences) was done in various positions (supine, prone, lateral decubitus), which clearly indicated abnormal movement of the spleen according to the posture, post-splenopexy images indicated that this motion was inhibited, indicating successful fixation [31]. However, current evidence for dynamic MRI is limited to isolated case reports, small series and therefore it remains investigational rather than a standard diagnostic modality [31,32]. The new emerging diagnostic techniques is CEUS [33]. In a case of torsion of a WS, CEUS helped by demonstrating lack of enhancement of the splenic parenchyma and absence of vessels in the hilum, enabling diagnosis of infarction without having to rely solely on CT or MRI [33]. The benefit is that, the CEUS may be performed at the bedside, it is relatively safe (no ionising radiation) and may provide faster analysis of perfusion than other techniques [33,34]. Small observational studies have shown diagnostic concordance of CEUS with CT in approximately 85-90% of cases usually in detecting infarction, although availability and operator expertise remain limiting factors [33]. Diagnostic modalities and imaging characteristics of WS are mentioned in the [Table/Fig-4] [1,25-34]. Diagnostic work-up for WS is depicted in [Table/Fig-5].

Diagnostic method	Key findings/features	Diagnostic accuracy and limitations	Clinical scenario (when to use)	Cost and availability considerations	References
Clinical evaluation	Palpable, mobile abdominal or pelvic mass; intermittent or acute abdominal pain; acute abdomen in torsion.	Non specific; no defined sensitivity/specificity. High false-negative risk due to intermittent symptoms	Initial clinical suspicion in patients with unexplained abdominal mass or recurrent pain	No cost; universally available but insufficient alone for diagnosis	[1,25]
Laboratory investigations	Usually non diagnostic; leukocytosis may be present in infarction.	No diagnostic accuracy value; cannot confirm WS	Used to rule out infection, inflammatory or haematological causes.	Low cost; widely available even in peripheral centres.	[25]
Ultrasound with Doppler	Ectopic spleen; reduced/ absent vascular flow in torsion	Sensitivity ~70-85% in case series; operator-dependent; false negatives in intermittent torsion or deep pedicle location	First-line imaging in stable patients, especially children and young adults; screening tool before CT	Low cost; widely available; suitable for resource-limited settings; portable and bedside use possible	[26,27]
Contrast-Enhanced CT (CECT)	Ectopic ovoid/comma-shaped spleen; whirl sign; non enhancing parenchyma in infarction; vascular pedicle definition	Diagnostic accuracy >90–95% in acute torsion; high sensitivity for infarction. Rare false positives (whirl sign overlap with other volvulus conditions)	Preferred confirmatory test in acute abdomen or suspected torsion; preoperative planning. First-line in unstable acute presentations	Moderate-to-high cost; requires contrast and radiation; availability limited in smaller or rural centres; less ideal in paediatric or pregnant patients due to radiation	[28,29]
Nuclear Scintigraphy/ MR Angiography	Demonstrates splenic perfusion and functional tissue viability	Limited quantitative data; high specificity for perfusion; limited evidence base; longer acquisition time	Adjunct when Doppler is inconclusive or when assessing residual splenic function	High cost; limited availability; rarely feasible in resource-limited settings	[30]
Dynamic MRI	Demonstrates spleen mobility in different body positions; post-splenopexy confirms fixation.	Evidence limited to case reports/small series; no established sensitivity/ specificity; investigational.	Selected chronic cases with equivocal imaging; post-operative assessment when radiation avoidance preferred.	High cost; limited availability; not practical in emergency or low-resource settings.	[31,32]

Contrast-Enhanced Ultrasound (CEUS)	Absence of parenchymal enhancement; absent hilar vessels in infarction; real-time perfusion imaging.	~85–90% concordance with CT in detecting infarction (small observational studies); operator-dependent; limited early torsion detection.	Alternative to CT when radiation contraindicated; useful bedside in stable patients; evaluation of perfusion when Doppler equivocal.	Lower cost than CT/MRI in centres with availability; contrast agents may not be widely available in rural settings; requires trained personnel.	[33,34]

[Table/Fig-4]: Diagnostic modalities and imaging characteristics of Wandering Spleen (WS) [1,25-34].



[Table/Fig-5]: Diagnostic workup for Wandering Spleen (WS); Created by Authors.

Differential Diagnosis of Wandering Spleen (WS)

The differential diagnosis of WS is broad due to its clinical manifestations which are usually non specific also they can resemble other causes of abdominal or pelvic masses [14]. Conditions inclusive of splenomegaly, accessory spleen, splenic cysts, splenic tumours can further present having a palpable abdominal mass similar to WS [14]. However, all these conditions usually demonstrate spleen in its normal anatomical position in the left upper quadrant, whereas WS is characterised due to absence of the spleen from its normal location, identification of an ectopic spleen elsewhere in abdomen or pelvis on imaging [14,35]. Mesenteric or omental cysts, pancreatic pseudocysts as well as retroperitoneal tumours can also mimic WS clinically [35]. However, cross-sectional imaging like CECT usually shows that these lesions lack splenic architecture and vascular pedicle which are characteristic findings in WS [35,36]. The presence of a long vascular pedicle, whirl sign of twisted splenic vessels on imaging strongly supports into diagnosis of WS [36].

In acute presentations, WS having torsion can further mimic other causes of acute abdomen including appendicitis, intestinal volvulus, bowel obstruction, pancreatitis, or renal colic [37]. In women, ovarian torsion, ovarian cysts, adnexal masses can also present as a pelvic mass similar to a displaced spleen [10,36]. Differentiation is usually achieved through imaging studies where ultrasound using Doppler or CECT shows splenic tissue morphology, vascular supply and abnormal mobility of spleen [37]. Unlike other intra-abdominal masses, WS usually shows splenic parenchymal echotexture, identifiable splenic vessels and displacement from left upper quadrant, sometimes accompanied by signs of pedicle torsion-splenic infarction [36,37]. Recognition of all these characteristic imaging findings further helps clinicians distinguish WS from other abdominal pathologies as well as it facilitates timely surgical management to prevent complications such as infarction or rupture [36]. Differential Diagnosis of WS is depicted in [Table/Fig-6] [10,14,35-37].

Differential diagnosis	Similarity with WS	Differentiating feature	Reference(s)
Splenomegaly	Palpable abdominal mass	Spleen remains in Left Upper Quadrant (LUQ); no ectopic location	[14]
Accessory spleen	Additional splenic tissue may mimic mass	Normal spleen present in LUQ; accessory spleen is small and separate	[14]
Splenic cyst	Cystic abdominal mass	Spleen located in normal position; lacks mobility and vascular pedicle	[14]
Splenic tumours	Abdominal mass arising from spleen	Spleen remains in LUQ; solid lesion without displacement	[14]
Mesenteric cyst	Intra-abdominal cystic mass	No splenic architecture or vascular pedicle on imaging	[35]
Omental cyst	Mobile abdominal mass	Lacks splenic tissue characteristics and vascular supply	[35]
Pancreatic pseudocyst	Upper abdominal mass	Related to pancreas; no splenic echotexture or pedicle	[35]
Retroperitoneal tumours	Abdominal or pelvic mass	Retroperitoneal origin; no splenic morphology or vessels	[35]
Appendicitis	Acute abdomen (pain, tenderness)	Inflamed appendix on imaging; spleen normal in LUQ	[37]
Intestinal volvulus	Acute abdominal pain, obstruction	Twisting of bowel loops; no ectopic spleen	[37]
Bowel obstruction	Pain, distension, vomiting	Dilated bowel loops; spleen in normal position	[37]
Pancreatitis	Upper abdominal pain	Elevated enzymes; pancreatic inflammation on imaging	[37]
Renal colic	Acute flank/ abdominal pain	Ureteric/kidney stones on imaging; spleen normal	[37]
Ovarian torsion	Pelvic pain/mass (females)	Adnexal origin on ultrasound; absent splenic features	[10,36]
Ovarian cyst	Pelvic mass	Cystic adnexal lesion; no splenic vascular pedicle	[10,36]
Adnexal mass	Pelvic lump	Gynaecological origin confirmed on imaging	[10,36]

[Table/Fig-6]: Differential diagnosis of Wandering Spleen (WS) [10,14,35-37].

Surgical and Emerging Minimally Invasive Approaches for Wandering Spleen (WS)

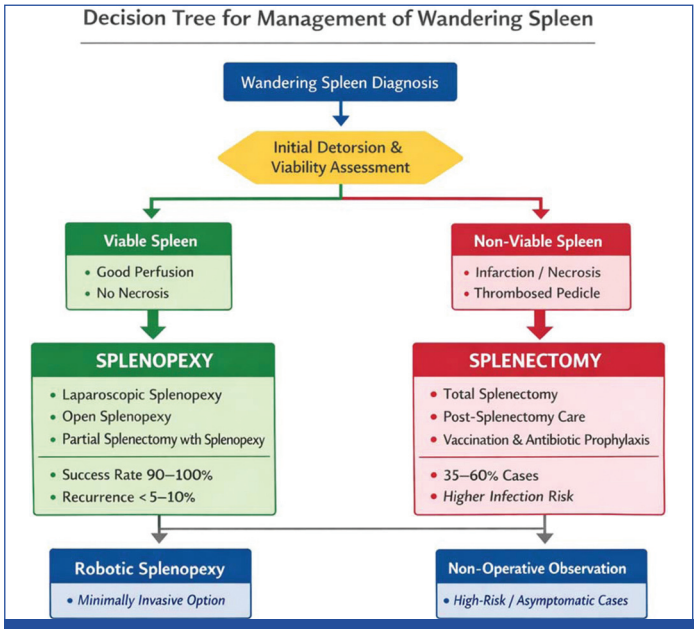
Treatment of WS primarily aims at preservation of splenic function in cases where possible. Pooled analyses suggest that up to 60-65% of untreated WS can eventually develop torsion which supports early surgical intervention in most patients [38]. when a viable non infarcted spleen is present the surgical intervention of choice is splenopexy which is increasingly done using minimally invasive approaches [38]. Laparoscopic splenopexy techniques

using mesh fixation (the so-called sandwich or keyhole composite-mesh techniques), includes making an extraperitoneal or omental pouch and suture fixation to the diaphragm or abdominal wall, all the methods aiming to restore and fix the spleen to its normal left-upper quadrant location without losing any splenic tissue or function [38,39]. Published series report success rates of 90-100% for laparoscopic splenopexy in maintaining splenic position having recurrence rates usually below 5-10% and overall perioperative complication rates ranging in between 5-15% [38]. Mean hospital stay is reported between 2-5 days [38].

Good short- and mid-term results are demonstrated using laparoscopic splenopexy, with low morbidity, quick recovery and long-time fixation [38]. The choice of absorbable versus permanent mesh, pocket or suture technique as well as the specific procedure is dependent on the size of the spleen, the experience of the surgeon and the age of the patient [38,40]. Paediatric surgeons prefer preservation of the spleen where possible [38,40]. Comparative analyses reported in recent series further suggest that minimally invasive splenopexy generally provides shorter hospital stay, faster recovery and lower postoperative pain compared to open splenopexy, while maintaining comparable long-term splenic preservation rates. However, randomised comparative studies remain limited [38-40].

Among the commonly described splenopexy techniques, "sandwich mesh technique" is inclusive of placing the spleen between two layers of mesh for stabilising it within the left upper quadrant whereas "keyhole technique" uses a mesh with a central opening through which the splenic pedicle passes allowing fixation while avoiding compression of the vascular pedicle [38,41]. Current literature also suggests that both techniques provide comparable outcomes although robust comparative trials are still lacking [38,41]. The choice of mesh type (such as absorbable versus non-absorbable) along with fixation technique is usually determined by splenic size, pedicle length, surgeon experience as well as intraoperative anatomical considerations [38,40]. Technical challenges can further arise during laparoscopic splenopexy due to factors such as an elongated vascular pedicle, splenomegaly, limited working space therefore these procedures can also require advanced laparoscopic expertise as well as familiarity with minimally invasive splenic surgery [38].

In cases of vascular compromise by torsion of the spleen leading to the lack of viable perfusion (necrosis, gangrene, thrombosed pedicle), or when there is uncontrolled sepsis or perforation, splenectomy becomes necessary [42]. Reported rates of splenectomy in contemporary series range between 35-60% which is largely influenced through delayed presentation and extent of infarction [32,42]. Intraoperative assessment after careful detorsion is followed by the decision: a possible viable spleen is to be repaired (splenopexy) and an inappropriate organ is to be removed and in other instances, some spleen tissue is saved by partial splenectomy with splenopexy of the remaining spleen [39,42]. Thus, a practical management pathway involves initial detorsion and assessment of splenic viability which is followed by splenopexy when perfusion is restored, whereas splenectomy is indicated in cases when irreversible infarction or necrosis is identified [39,42]. Nowadays the majority of the series recommend surgical management due to the high-risk of torsion and infarction [43]. Decision tree for management of WS is depicted in [Table/Fig-7].



[Table/Fig-7]: Decision tree for management of Wandering Spleen (WS) (splenopexy vs splenectomy); Created by Authors.

Age-specific considerations are important in treatment planning, as WS exhibits a bimodal distribution, with increased incidence in both children and women of reproductive age [15,43]. Paediatric cases are usually associated with congenital ligamentous laxity therefore splenic preservation through splenopexy is strongly emphasised for maintaining long-term immunological function [43].

Non operative management with close imaging and clinical follow-up has been reported also in very selected asymptomatic or high-risk patients but this method is used in extraordinary cases and with informed consent on the risk of future torsion [32,44]. Long-term natural history data for conservative management remains limited also delayed intervention is associated having increased likelihood of splenectomy [43]. Robot-assisted procedures such as robot-assisted partial splenectomy and precision splenopexy are also reported in the case of benign splenic disease and provide greater dexterity of parenchymal sparing resections or secure fixation in difficult anatomy which may expand options in number of cases where splenic preservation is desired [45]. Early small case series reports favourable short-term outcomes showing complication rates below 10% although evidence still remains limited and long-term outcomes beyond five years remain insufficiently studied, cost considerations restricts widespread adoption [7,46]. Recent literature has also highlighted the potential role of advanced imaging technologies which are inclusive of CEUS, dynamic contrast-enhanced MRI protocols for evaluation of splenic perfusion and vascular pedicle orientation which may assist in determining splenic viability before or after detorsion and help guide surgical decision-making in complex cases [33,47]. CEUS in particular allows real-time assessment of splenic microvascular perfusion without exposure of radiation which can be especially beneficial in paediatric patients and during postoperative follow-up [33]. Surgical and Non surgical Management Strategies for WS are described in [Table/Fig-8] [7,38-46].

Type of management	Indication	Procedure/technique	Key decision criteria/ technical notes	Outcomes (recurrence/ complications/long-term results)	References
Splenopexy (Preferred when spleen is viable)	Viable, non infarcted spleen; no irreversible ischaemia or necrosis	Laparoscopic splenopexy using mesh fixation (sandwich/keyhole composite-mesh techniques); creation of extraperitoneal or omental pouch; suture fixation to diaphragm or abdominal wall	Viable spleen defined by preserved or restored perfusion on Doppler/CT after detorsion and absence of extensive infarction. Sandwich technique stabilises spleen between two mesh layers, whereas keyhole mesh allows pedicle passage without compression. Mesh selection depends on splenic size, pedicle length and surgeon preference.	Success rate 90–100%; recurrence generally <5-10%; perioperative complication rate ~5-15%; preservation of splenic immune function.	[38,39] [38-41]

Open splenopexy	Large spleen or complex anatomy unsuitable for laparoscopy	Fixation via retroperitoneal pocket or sutures to adjacent structures	Preferred when splenomegaly, adhesions, or limited laparoscopic access present.	Recurrence usually <10-15%; slightly higher morbidity than laparoscopic approaches.	[39,40]
Splenectomy (Total)	Non viable spleen due to torsion, infarction, necrosis, thrombosed pedicle, or sepsis	Surgical removal of spleen following intraoperative assessment post-detorsion	Indicated when perfusion fails to recover after detorsion or when extensive infarction or necrosis is present	Definitive management preventing recurrent torsion but associated with risk of postsplenectomy infections requiring vaccination	[31,32,42,43]
Partial splenectomy with splenopexy	Partial viability of spleen with salvageable tissue	Resection of infarcted portion with fixation of remaining viable spleen	Considered when segmental infarction occurs but adequate vascularised splenic tissue remains	Allows preservation of partial immunologic function; limited but favorable outcomes reported	[39,42]
Non operative management	Selected asymptomatic or high-risk surgical patients	Observation with regular imaging and clinical follow-up	Reserved for minimal symptoms or high surgical risk; requires counseling regarding torsion risk.	Natural history uncertain; delayed intervention associated with higher splenectomy risk	[32,43,44]
Robotic-assisted surgery	Benign splenic disease or technically difficult splenopexy cases	Robot-assisted partial splenectomy or robotic splenopexy	Provides improved dexterity and visualisation in complex anatomy; learning curve and cost limitations.	Early reports show complication rates <10%; long-term comparative data still limited.	[7,45,46]

[Table/Fig-8]: Surgical and non surgical management strategies for Wandering Spleen (WS) [7,38-46].

CONCLUSION(S)

The WS is a rare, although clinically very important disease which presents as hypermobility of the spleen through either congenital ligamentous defects or acquired laxity. It can be presented with asymptomatic abdominal masses to acute abdomen due to torsion and infarction, which is hard to diagnose in a timely manner. Early recognition is an essential aspect because pooled clinical series suggest that up to 60 to 65% of untreated WS cases can further develop splenic torsion thus supporting timely surgical evaluation of patients. Ultrasound, CT, dynamic MRI and CEUS are imaging modalities that are of key importance in the identification of spleen location, vascular status and mobility, with CT currently considered the most reliable modality for confirming ectopic splenic location and evaluating pedicle torsion and splenic perfusion.

The management is focused usually on preserving spleen with usage of splenopexy whereas in cases of the splenic non viability, splenectomy becomes only alternative. Laparoscopic splenopexy has shown high success rates (which are approximately 90-100%) with recurrence rates generally below 5-10% thereby making it the preferred treatment when splenic viability is preserved. In patients who are undergoing splenectomy, appropriate post-splenectomy care which is inclusive of vaccination, consideration of antibiotic prophylaxis and long-term clinical follow-up is essential to reduce the risk of overwhelming post-splenectomy infection. Reported postoperative complication rates are usually below 10 to 15% while mortality remains rare when timely intervention is performed in patients.

New minimally invasive and robotic procedures also improve outcomes, reduce morbidity in patients. Robotic-assisted techniques can further enhance precision in splenic preservation procedures although current evidence remains limited to small case series as well as it requires long-term outcome evaluation. Future research must focus on developing standardised diagnosis algorithms, long-term follow-up protocols as well as comparative effectiveness studies of different splenopexy techniques in WS cases.

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