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REVIEW

MRI signs for intraneural ganglion cysts: a roadmap revealing the pathoanatomic and pathophysiologic principles underlying the unifying articular theory

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Abstract
Intraneural ganglion cysts (IGCs) are benign, mucinous lesions that are often found within the epineurium of peripheral nerves, causing mononeuropathy due to nerve compression. These cysts arise from synovial fluid tracking from an adjacent synovial joint along an articular nerve branch to the parent nerve, as proposed by the unifying articular theory. This theory has transformed the understanding of IGCs from their being considered as isolated nerve lesions but as joint-related pathologies. The emphasis of identifying the cyst's origin and its connection to the neighboring joint has shifted the surgical strategy from evacuation of the cyst to addressing the origin of the cyst and its articular branch connection. The role of high resolution imaging in both detection and surgical planning is thereby critical, because in some cysts (i.e. affecting the peroneal and tibial nerves in the knee region), the cyst may be intraneural or extraneural and arise from different joints. In this article, we summarize the magnetic resonance imaging (MRI) signs for IGCs and introduce the concept of phase-based signs which are all explained by pathoanatomic and pathophysiologic principles underlying the unifying articular theory. These signs, in turn, thave expanded our understanding, changed our clinical practice, and advanced the field.

Keywords Intraneural ganglion cyst · MRI signs · Peroneal nerve ganglion cyst

Introduction

Intraneural ganglion cysts (IGCs) are benign, mucinous lesions that are found within the epineurium and/or paraneurium of peripheral nerves, often causing mononeuropathy due to nerve compression [8]. These cysts arise from synovial fluid arising from an adjacent synovial joint and track along an articular nerve branch to the parent nerve, as proposed by the unifying articular theory [42]. This theory has shifted the understanding of IGCs from isolated nerve lesions to joint-related pathologies, emphasizing the critical role of imaging in identifying the cyst's origin and its connection to the neighboring joint [15]. It is important not only for the detection of these cysts, but also for surgical planning.

A significant amount of research has been conducted over the past 20 years attempting to understand the pathophysiology and pathoanatomy of IGC formation and propagation by our groups and others. Over this time, different MRI signs have been introduced which are useful in establishing a radiologic diagnosis, demonstrating a joint connection and underlying principles of cyst formation and guiding treatment. This comprehensive review summarizes a vast literature and focuses on the MRI signs of IGCs based on the articular theory, exploring their diagnostic utility and their impact on improving surgical outcomes. We believe these radiologic signs on MRI provide a roadmap for radiologists and surgeons to understand the formation and treatment of IGCs.

The articular theory

Most of the work towards validating the unified articular theory has been based on the most commonly involved site for an IGC- the common peroneal nerve (CPN) at the fibular

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neck arising from the superior tibiofibular joint (STFJ) [15]. The articular theory provides a robust anatomical explanation of IGC development and pathophysiology, which has been supported by many groups [4, 24, 33]. This theory explains not only the most common cases: peroneal in the lower limb and ulnar nerve in the upper limb [34] but also apparent outliers including examples in suprascapular [26], plantar, [11], tibial [4, 39], digital and other nerves [27]. IGCs are connected to the joint through an articular branch of the nerve. For IGCs, the articular theory focuses targeted treatment to the articular branch connection and to the joint of origin. Of note, articular connections have been identified in many cases where the joint connection was refuted. [15, 30] Since the inception of the articular theory, outcomes have improved and intraneural recurrences have decreased. This articular theory also explains extraneural (soft tissue) ganglion cysts [19, 45]. Extraneural cysts occur when fluid is extravasated from a synovial surface, such as a joint and create a cyst which is connected to the joint via a non-neural pedicle. It also explains an analogous, equally controversial entity- adventitial cysts in vessels-which also originate from joints [13, 23].

There are 3 major principles of the unifying theory for IGCs:

1. An articular branch connection to a synovial joint, consistent with Hilton's Law [10], serves as the pathway through which synovial fluid from a neighboring joint can egress through a capsular rent/defect. These joints commonly have degenerative changes.
2. Cyst fluid extends following the path of least resistance.
3. Pressures and pressure fluxes can explain the most extensive type of cyst with extreme distribution in the subparaneurial compartment [6]. ICGs have dynamic features, and can vary from these extreme cysts to nearly invisible cysts and/or nearly invisible joint connections [18, 44]. Several snapshots in time have captured dramatic fluctuations in the size and morphology of IGCs, taking on the course of a roller coaster and highlighting their dynamic nature [43].

Prototypes

Peroneal/tibial IGCs in the knee region

While originally thought to be quite rare, IGCs are becoming increasingly recognized, especially with improved imaging. For example, ultrasound (US) is nowadays frequently used to diagnose peroneal neuropathy which leads to increasing detection of ICGs [3]. Of the > 1100 IGC cases in the literature, more than 600 have involved the peroneal nerve. As recently found, the actual prevalence of peroneal IGCs in the setting of peroneal mononeuropathy is about 18% [41]. We believe and have shown that an articular connection can always be identified in the cases of IGCs. The joint origin of these peroneal cysts needs to be addressed at the time of surgery. Patients with peroneal IGC typically present with predominant deep peroneal nerve (DPN) loss.

Anatomy

Just proximal to the popliteal fossa, the sciatic nerve bifurcates as the CPN and tibial nerve. In the popliteal fossa, there are variable communicating branches of the CPN and tibial nerves to form the sural nerve. After passing obliquely beneath the biceps femoris muscle, the CPN branches within the fibular tunnel into the superficial peroneal nerve (SPN), the DPN and the articular trunk which gives rise to a tibialis anterior branch and the important recurrent articular branch to the anterior portion of the STFJ. The articular trunk anatomy involves a descending, transverse and ascending limb taking a U-shape across the fibular neck [28]. (Fig. 1) Understanding the intraneural topography of the articular branch explains its location within the DPN fascicular group. Knowledge of the different layers of the nerve, including the epineurium and the paraneurial layers surrounding the epineurium is of utmost importance in order to explain the pathophysiological development of IGCs which initially develop in the subepineurial and can later extend into the subparaneurial compartment [7, 22]. Of note, ganglia can be intraneural, extraneural or combined intra/extraneural as shown in Fig. 1.

Pathoanatomy

Based on the anatomy and principles of the articular theory, stages and phases have been put forth to explain the formation and propagation of IGCs. For peroneal IGCs arising from the STFJ, they form within the anterior aspect of the STFJ (Stage 0). They extend along the articular branch (Stage I), and within the DPN portion of the CPN (Stage II), the CPN (Stage III), and the sciatic nerve (Stage IV) (Fig. 2). Phase I (ASCENT) corresponds to articular cyst fluid from the STFJ passing along the articular branch into the CPN below the epineurium. Phase II (CROSSOVER) occurs when the IGC has reached the sciatic bifurcation and the cyst can pass through an epineurial opening into the subparaneurial space [6]. The subepineurial cyst can be compared to a geyser channel below the earth. When the pressure increases and reaches a specific threshold, the cyst will "erupt" and cross over from the subepineurial to the subparaneurial compartment. This latter compartment is a potential space and can expand significantly [6]. After cyst expansion, redistribution of cystic fluid occurs within the different compartments following the path of least resistance.

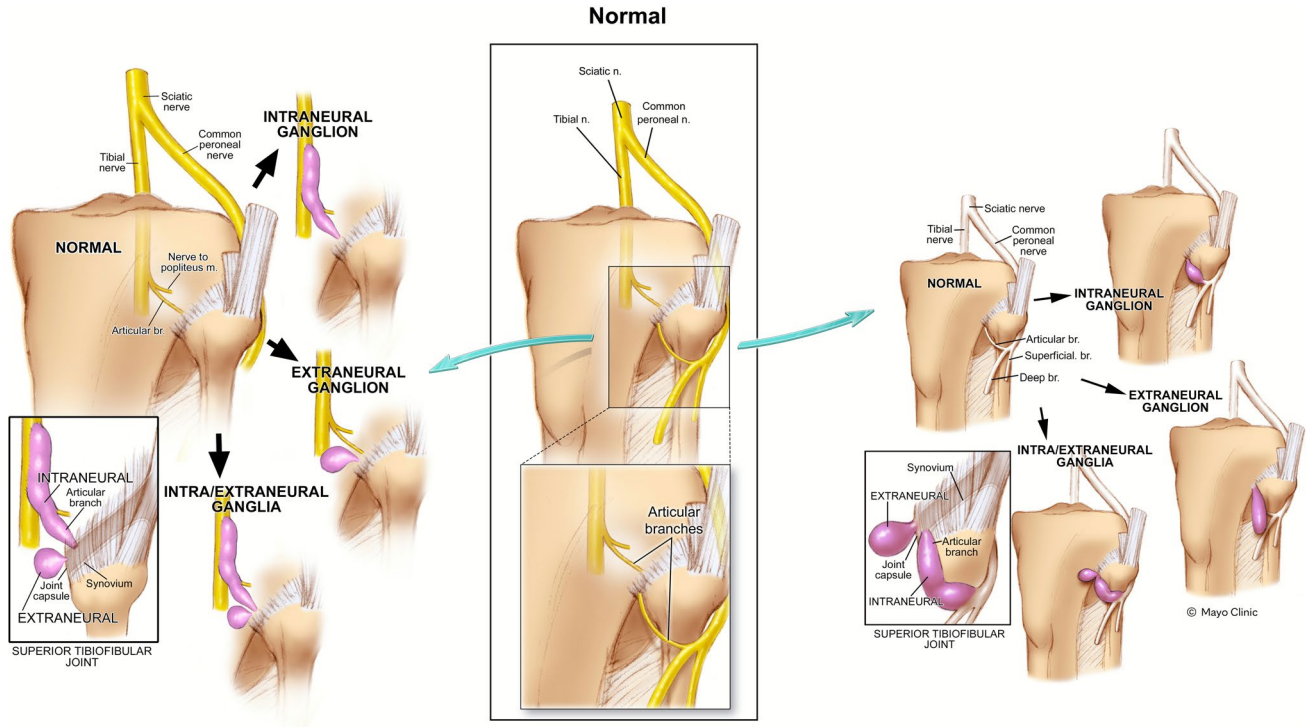

Fig. 1 Normal anatomy of the sciatic, tibial, common peroneal nerves along with the articular branches to the superior tibiofibular joint (anterior joint-peroneal, posterior joint-tibial). Intraneural and/or extraneural ganglion cysts involving the tibial (left of figure) and peroneal nerves (right of figure) cysts can arise from the STFJ. (From: Spinner RJ, Mokhtarzadeh A, Schiefer TK, Krishnan KG, Kliot M, Amrami KK. The clinico-anatomic explanation for tibial intraneural ganglion cysts arising from the superior tibiofibular joint. Skeletal Radiol. 2007 Apr;36(4):281–92. https://doi.org/10.1007/s00256-006-0213-2. Epub 2006 Dec 23. PMID: 17,187,290, published with permission)

Phase III (SUBPARANEURIAL ASCENT/DESCENT) is a result of rapid filling of the subparaneurial space with cystic fluid due to the high intraneural pressure with a combination of low compliance of the subparaneurial space and gravity favoring downward extension (Fig. 3). This descent follows the subparaneurial compartments around the tibial and CPN, including the distal branches: the DPN, SPN and sural nerves [40]. The extent can be proximal or distal over long-segments leading to extreme intraneural and extreme subparaneurial ganglion cysts [7, 31]. Thus, an extreme IGC involving the peroneal nerve arising from the STFJ can lead to extreme longitudinal involvement in the sciatic nerve in the thigh or lower buttock and explain apparent clinical outliers [31]. Extraneural rupture of the cyst or spontaneous regression (absorption) can follow the subparaneurial distribution, which can be explained by openings in the outermost paraneurial layers [6, 7]. Peroneal IGCs may also rarely develop from the knee joint and propagate similarly.

Tibial IGC counterpart

Tibial IGCs arising from the STFJ provide additional support for the unifying articular theory [10, 39]. Similar to the development of peroneal IGCs from the anterior portion of the STFJ, cystic fluid passes through an articular branch supplying the popliteus branch [39]. Cyst formation and propagation follows similar stages/phases as do peroneal IGCs with extension into the sciatic nerve and cross-over to the CPN through an epineurial opening. Tibial IGCs form within the posterior aspect of the STFJ (Stage 0), and extend along the articular branch (Stage I), and the branch to the popliteus (Stage II), followed by the main tibial (Stage III), and the sciatic nerve (Stage IV) (Fig. 2). They may also undergo the crossover phenomenon (Fig. 3). Tibial IGCs may be found within the popliteal fossa originating from the tibiofemoral joint and extending via articular branches to the knee [20]. Another important example of tibial IGC are those found in the foot/ankle region (tarsal tunnel) with subtalar or tibiotalar joint connections [36].

Radiologic imaging

Radiologic imaging, by means of US and particularly magnetic resonance imaging (MRI) has become indispensable in diagnosing IGCs and characterizing them.

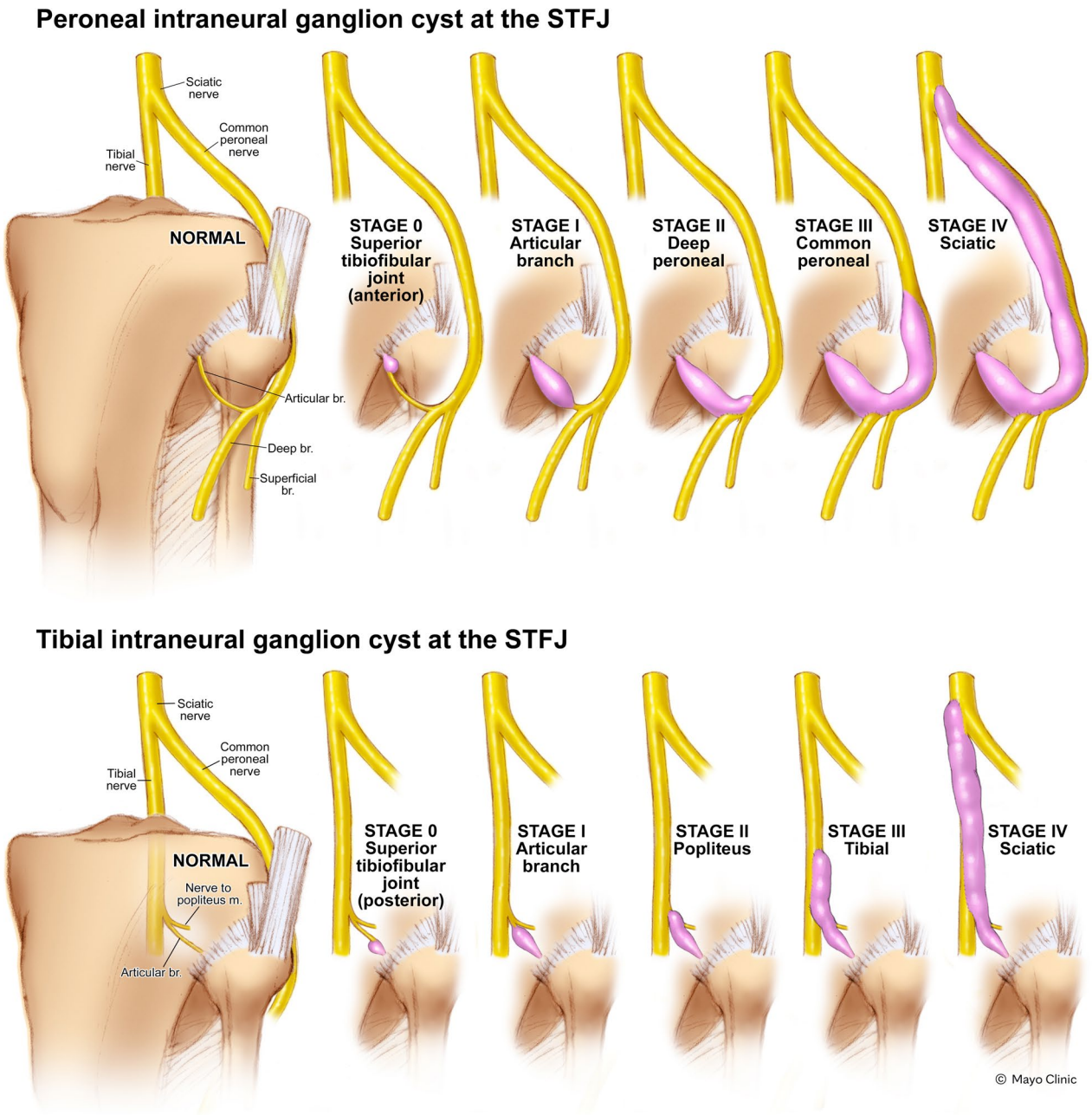

Fig. 2 Various stages of intraneural ganglion cyst (IGC) development. Peroneal IGCs develop from the anterior portion of superior tibiofibular joint (Stage 0) proximally to the articular branch (Stage I), the DPN (Stage II), the CPN (Stage III), and the sciatic nerve (Stage IV). Tibial IGC develop from the posterior portion of superior tibiofibular joint (Stage 0) proximally to the articular branch (Stage I), the popliteus branch (Stage II), the tibial (Stage III), and the sciatic-nerves (Stage IV). (From: Spinner RJ, Mokhtarzadeh A, Schiefer TK, Krishnan KG, Kliot M, Amrami KK. The clinico-anatomic explanation for tibial intraneural ganglion cysts arising from the superior tibiofibular joint. Skeletal Radiol. 2007 Apr;36(4):281–92. https://doi.org/10.1007/s00256-006-0213-2. Epub 2006 Dec 23. PMID: 17,187,290., Used with permission of Mayo Foundation for Medical Education and Research, all rights reserved.)

They can reveal characteristic signs that distinguish IGCs from other cystic lesions (including extraneural ganglion cysts) and can help guide surgical management to prevent recurrence [37].

While our preference at our institution has been high resolution MRI to image IGCs [1, 18, 43], we recognize the utility of high resolution US as well [16]. Other groups

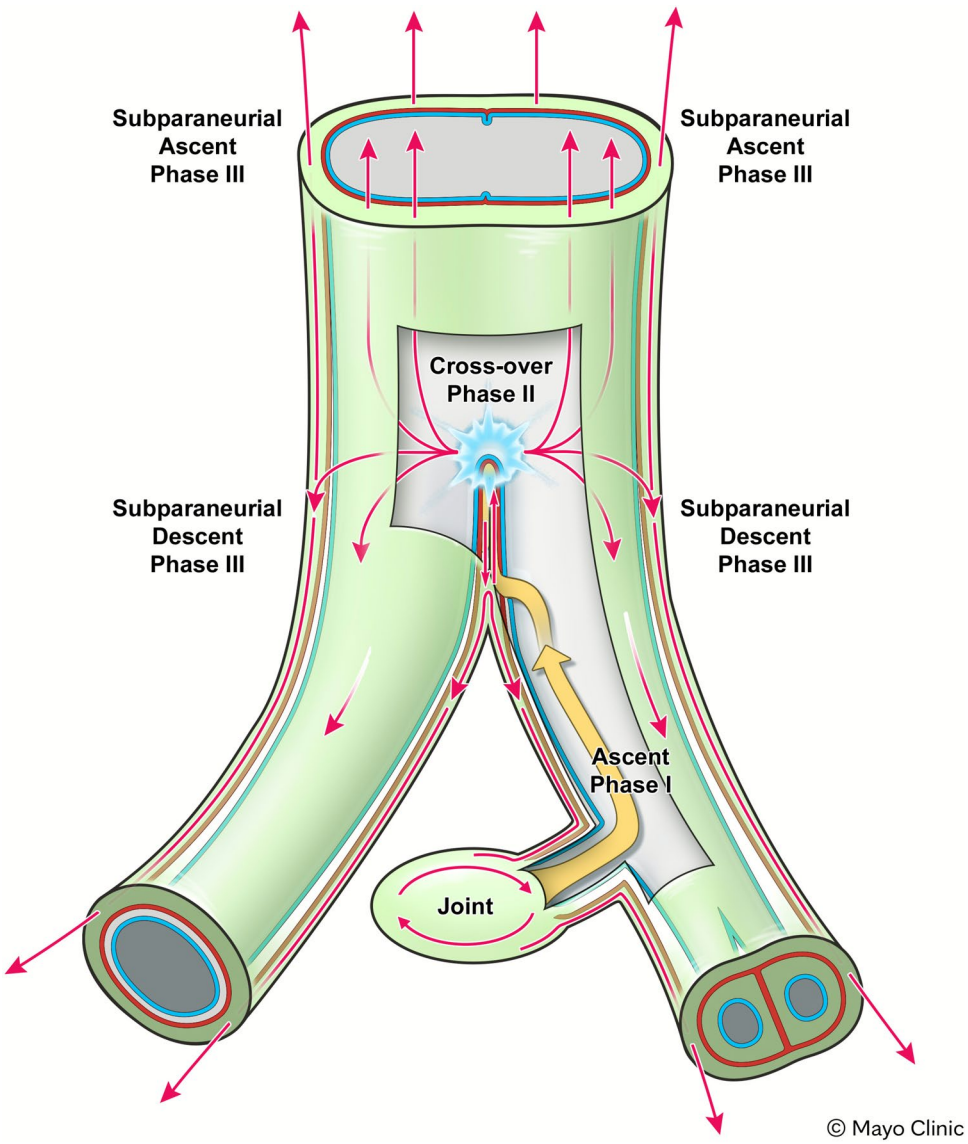

Fig. 3 Various phases of intraneural ganglion cyst (IGC) progression. Peroneal and tibial epineurium is light blue, peroneal and tibial paraneurium is red and becomes sciatic epineurium, extra paraneurium is black. Phase I (ASCENT): Articular cyst fluid passes from the superior tibiofibular joint along the articular branch into the common peroneal nerve beneath the epineurium (light blue line). Phase II (CROSSOVER): Cyst fluid crosses from the subepineurial (has to cross through both peroneal and sciatic epineurial layers) to the subparaneurial space, expanding within this potential compartment. Pressure fluxes similar to a geyser will lead to Phase III (DESCENT IIIA-parent nerve-/IIIB-secondary nerve- or ASCENT): Rapid filling of the subparaneurial space occurs under high intraneural pressure, both downwards and upwards. Used with permission of Mayo Foundation for Medical Education and Research, all rights reserved

have preferred to use high resolution US [14, 21]. US can detect cystic lesions and can raise concerns for an IGC but it is operator-dependent and the articular branch connection is often challenging to visualize [12]. A recent study showed that MRI was statistically more likely to detect a joint connection compared to US ($p < 0.01$) [15]. In this study, MRI found the joint connection in 62% of cases with US only detecting the connection in 16% of the cases [15]. MRI provides additional details and can help identify the different nerve compartments involved as well as identify the articular branch connection. On MRI, IGCs are hypointense on T1 and hyperintense on T2-weighted sequences and follow the course of their parent nerves, with or without extension into their branches [8]. In T1 contrast enhanced sequences, IGCs can show wall enhancement [15]. The joint connection can be followed best on thin axial slices to identify the articular branch and involved joint. Various signs can be seen in different planes (axial, sagittal, coronal) which are discussed in detail below.

As imaging technology has advanced, the detection of IGCs has increased not only in patients with typical presentations of peroneal mononeuropathy but also in patients with atypical entrapment syndromes at unusual anatomical sites [29]. The ability to identify joint connections with MRI has transformed the management paradigm, moving away from isolated cyst resection or decompression toward addressing the underlying joint pathology [37].

Radiologic signs of IGCs

All MRI signs were introduced based on pathoanatomic and pathophysiologic principles which are described below. Despite variability and limitations between resolution of different imaging parameters, these signs are robust and sensitive. We will focus on the well described peroneal and tibial IGCs arising from the STFJ. The articular branches are very small (even when involved by cyst) and multiple thin slices are required to identify them especially when the cyst does not expand them as in the case of nearly invisible joint connections [17, 44]. In the case of peroneal IGCs at the STFJ, disproportionate denervation changes or atrophy in the tibialis anterior muscle can be seen, previously reported at 57% of cases [44]. Denervation changes can then more extensively involve the anterior followed by the lateral compartment of the leg. Degeneration of the STFJ was found in 100% of cases [44]. Denervation changes of the popliteus muscle is often seen with tibial IGCs. Figure 4 summarizes the signs that occur during Phase I while Fig. 5 illustrates those during Phase II and III. This concept of phase-based MRI helps with the understanding of pathoanatomy of IGC formation putting into perspective the temporal component of its formation as well as its natural history when MRI comparisons are available.

1. Tail sign (Phase I)

   This sign assesses for a joint connection. This connection occurs at the articular branch of the nerve in IGCs or non-neural pedicles in the case of extraneural ganglia. In the case of IGC, the tail sign represents the

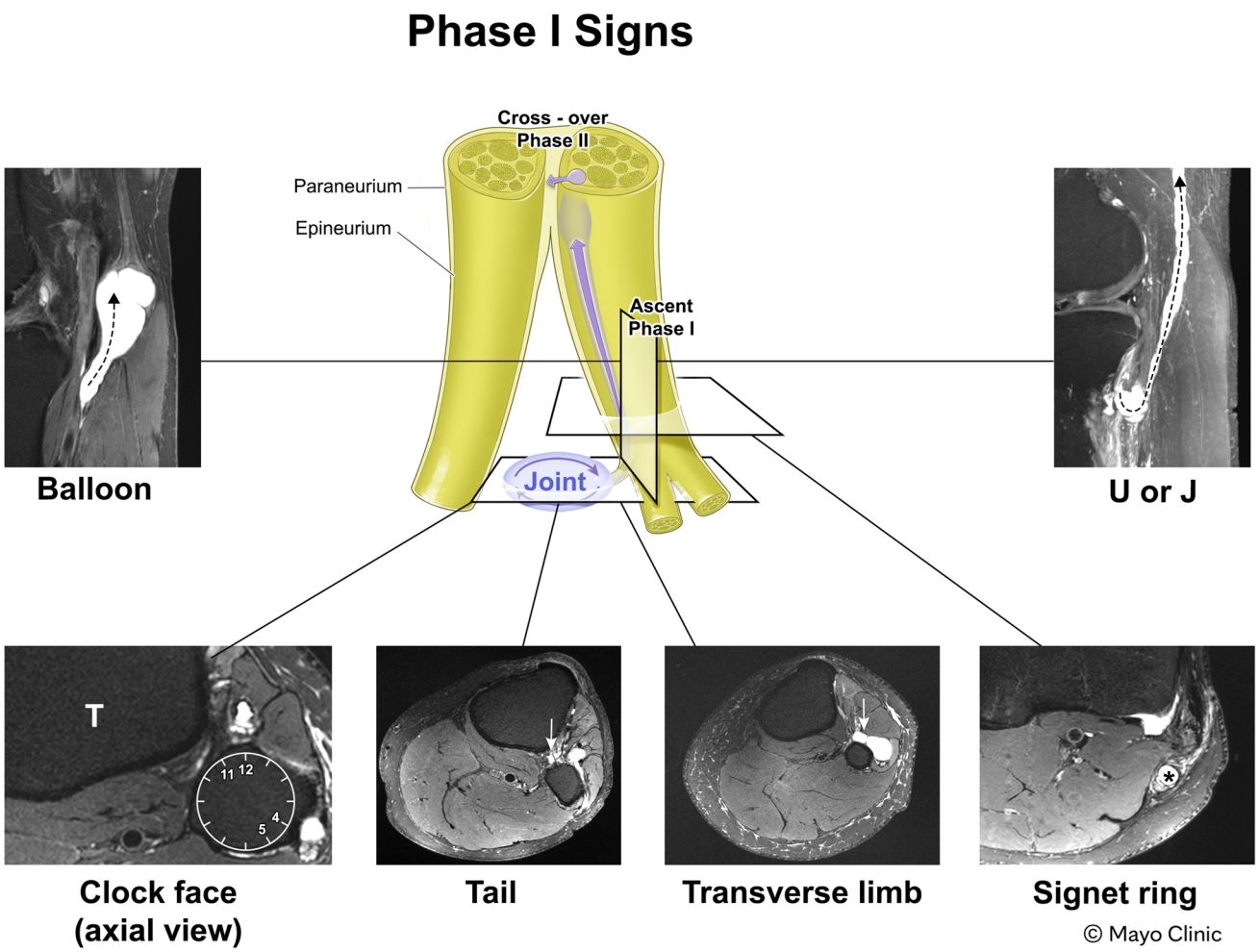

Fig. 4 MRI signs of intraneural ganglion cysts, organized by pathoanatomy and their corresponding sites of detection during Phase I. Top panel: tibial intraneural ganglion cysts originating from the superior tibiofibular joint (STFJ) (shown to generalize signs); bottom panel: peroneal intraneural ganglion cysts from STFJ. All MRIs obtained at 7T. Used with permission of Mayo Foundation for Medical Education and Research, all rights reserved

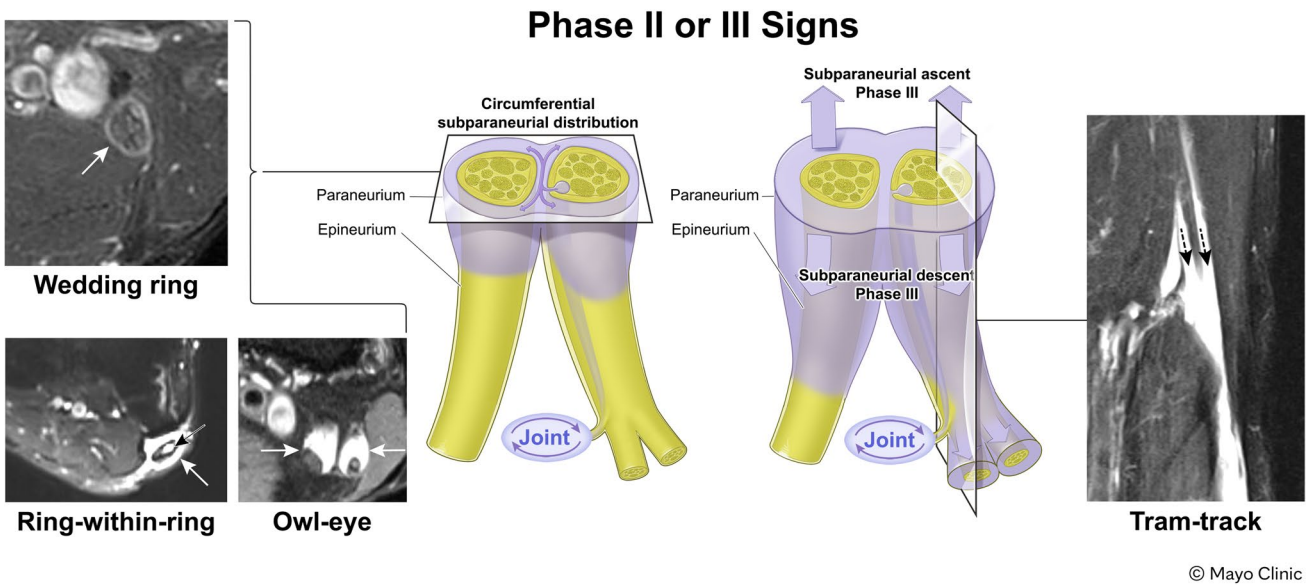

Fig. 5 MRI signs of intraneural ganglion cysts, organized by pathoanatomy and their corresponding sites of detection during Phase II or Phase III. Used with permission of Mayo Foundation for Medical Education and Research, all rights reserved

ascending limb of the articular branch which is the first segment of the "U or J sign" (will be discussed later) [28]. The tail sign is commonly best seen on thin sliced axial and sagittal T2-weighted fat suppressed images focusing on the joint.

2. Transverse limb sign (Phase I)

   This sign represents intraneural cyst extension into the transverse limb of the articular branch of the peroneal nerve [28]. The transverse limb sign is seen best on a sagittal T2 image along the course of the articular branch as it crosses right anterior to the neck of the fibula. It can also be seen on axial and coronal cuts. This is usually not seen in extraneural ganglion cysts.

3. Signet Ring sign (Phase I)

   The signet ring sign is present in cysts located within the epineurium. It shows the eccentric nature of the IGC and highlights the displacement of the fascicles within the nerve when the cyst extends proximal enough into the larger common peroneal or other major nerves [28]. This sign is typically seen in axial T1 or T2-weighted sequences. The nerve fascicles appear as crescent-shaped structures surrounding a low-signal cystic area, with interspersed fat highlighting their appearance on T1-weighted sequences. On T2-weighted images, the fascicles are similarly displaced in a crescentic configuration around the bright signal of the intraneural cyst. The signet ring sign has near perfect sensitivity but relatively lower specificity [28]. One reason why this sign is absent would be that the IGC has less proximal extension along the nerve or the intraepineurial cyst has deflated from altered pressures leading to subparaneurial extension. Additionally, it helps differentiate it from extraneural ganglia, which may displace the nerve or flatten the fascicles without showing any cystic component within the nerve. In these cases, a pseudo-signet ring sign is described where the nerve itself is normal with a preserved fat plane between the nerve and the cyst.

4. U or J sign (Phase I)

   This sign represents the anatomical configuration of the articular branch of the peroneal nerve [28]. When this is affected by the IGC, one can observe the U or J-type shape on a coronal T2-weighted MRI where the cyst involves both the descending and ascending limbs of the articular branch (U sign) vs extension even more proximal toward the parent nerve (J sign). J sign can be observed in tibial origin cysts as well.

5. Clock face sign (Phase I)

   The clock face sign is seen in T2 axial fat suppressed images. At the mid-fibular head level (where its diameter is maximal) its shape is circular whereas the tibia appears triangular. For example, with a clock face over the left fibula, the signet ring is usually seen between the 4 and 5 o'clock position, while the tail sign is present at the 11 to 12 o'clock position. A few slices distal to that point, at the fibular neck, the previously described transverse limb sign can be seen between the 12 and 2 o'clock position [30, 35]. For tibial IGCs at the STFJ, the clock face shows a connection at the 9 o'clock position which supports the anatomic location of the articular branch relative to the STFJ and the cyst [35].

In the case of extraneural ganglia, the joint connection is not in a consistent position and varies according to the location of the capsular defect. The connection is usually more superior compared to IGCs (usually 12-2 o'clock position). The cyst location is also inconsistent and can pass through different planes following the path of least resistance (intramuscular, subcutaneous, in vessels or intraosseous) [30, 35].

6. Balloon sign (Phase I)

   This sign represents the proximal expansion of an IGC with a tapered distal neck which is similar to a party balloon [25, 32]. Explained by the same mechanism, the cyst fills under pressure creating the balloon through a thin neck (articular branch) covered by epineurium. The balloon (cyst) then enlarges in a proximal direction within the nerve (in cases without cyst rupture) which signifies directionality which, in turn, can help identify the joint of interest in atypical IGC cases in the hand or foot and assist in surgical planning [32].

7. Wedding ring sign (Phase II or III)

   Peroneal ICGs can extend proximally to the level of the sciatic bifurcation. In that location, the tibial and CPN paraneurium becomes the epineurium of the sciatic nerve and the outermost circumneurium becomes the sciatic paraneurium [6]. If the cyst extends to that level and crosses the epineurium and subsequently the paraneurium of the CPN (crossover), it may surround the sciatic nerve either partially or circumferentially in the subparaneurial space [40]. This potential space is filled with cystic, mucinous fluid and creates the wedding ring sign which is a sign of crossover from the peroneal to the tibial nerve and vice versa [6, 7]. It may be present in the CPN, sciatic or tibial nerves. The wedding ring sign may be subtle because of dynamic features of cyst development.

8. Ring within ring sign (Phase II or III)

   This sign is a combination of a subepineurial cyst creating the "signet ring" sign at a location where subparaneurial extension has occurred which subsequently creates a subparaneurial cyst surrounding the signet ring; thus the name "ring within ring" [6, 7]. This sign demonstrates a cyst-within-cyst at two different compartments of the nerve and is best seen on a single axial T2-weighted slice.

9. Owl-eye sign (Phase II or III)

   This sign is simply a more prominent wedding ring sign, signifying a higher degree of subparaneurial extension which assumes an owl-eye appearance. The complete owl sign is best seen on axial T2 fat suppression sequence initially with circumferential subparaneurial cyst around the epineurium of a nerve (CPN, tibial, sural) or later to distal branches during the subparaneurial ascent or descent of Phase III [6, 7].

10. Tram track sign (Phase III)

    The tram track sign signifies the occurrence of extreme subparaneurial extension longitudinally either during the ascent or descent phase [6, 7]. On coronal or sagittal T2-weighted MRI, the hyperintense cystic fluid in the subparaneurial space is reminiscent of tram-tracks. These can extend all the way from the sciatic down to the sural nerve creating this sign.

Differential diagnosis

The main differential diagnosis of cystic nerve lesions includes IGC, extraneural ganglion cysts (or paralabral cyst for example), cystic schwannoma, nerve abscess, and Hansen's disease (leprosy). Clinical exam and detailed history are indispensable in narrowing down the differential diagnosis. Extraneural ganglion cysts are characterized by the same quality cystic fluid which remains extraneural and can even be seen in the intramuscular, adventitial or intraosseous spaces. Some of the signs discussed below will help differentiate between intra-and extraneural ganglions.

Extraneural ganglia occur when the capsular defect leads to joint fluid leaking in the neighboring tissues but not in the nerve. These cysts are more globular in appearance, compared to IGCs which have a more tubular configuration. They are commonly found intramuscular. A unique type of extraneural ganglion cyst is a paralabral cyst, which is a fluid-filled cyst that develops near the labrum typically after a labral tear, allowing articular fluid to leak and form a cyst. They have decreased signal intensity on TI-weighted images and increased signal intensity on T2-weighted images. Their location is most commonly at the shoulder or hip which are atypical for IGC, although they can happen [26, 38]. These cysts at the glenohumeral joint are connected to the labrum and can cause mechanical compression of the neighboring suprascapular nerve similar to an extraneural ganglion cyst. However, depending on the site of the capsular rent and articular branch, cystic fluid can enter the nerve and lead to a suprascapular IGC [38].

Cystic schwannomas are common subtypes of schwannomas. Schwannomas are more commonly solid or mixed solid/cystic. In cases of cystic schwannomas, an articular connection is not identified on imaging. Enhancement of the capsule is typically seen and can be thick. Cystic contents are often heterogeneous [5].

Nerve abscess can be a very rare mimic of IGC and often occurs in the setting of Hansen's disease (leprosy) in endemic areas. It is characterized by a peripherally enhancing lesion, T1 hypointense and T2 hyperintense, inside a T2 hyperintense nerve [2]. Often times, there is a central necrotic core that does not enhance similar to abscesses elsewhere in the body [9].

Additional, uncommon diagnoses that could be considered and for which the MRI signs can help differentiate are *synovial sarcoma* of the nerve [2, 25], de Quervain *tenosynovitis* for IGC of the superficial radial nerve [13], and *malignant peripheral nerve sheath tumor* [26].

Conclusion

The hallmark radiologic features of IGCs provide a window into their unique pathoanatomy and pathophysiology aiding in precise diagnosis. On MRI, IGCs typically present with hypointensity on T1-weighted sequences and hyperintensity on T2-weighted sequences, reflecting their mucinous fluid nature, with enhancement of the cyst wall following gadolinium administration. When evaluating IGCs on MRI, it is important to include the entire nerve that is involved (sometimes as proximal as the sciatic at the buttocks and as distal as the sural nerve at the ankle) as in cases of extreme subparaneurial cysts. Thin axial slices as well as coronal and sagittal planes, all give distinct information and can aid in the diagnosis with the signs discussed. These signs offer diagnostic specificity by highlighting the cyst's relationship with the parent nerve and its articular branch. They do not only confirm the diagnosis, but they have expanded our understanding of IGC pathophysiology and natural history with different signs emanating during the various phases of IGCs. They finally changed the clinical practice pointing to the unifying articular theory and pushing the field towards a surgical disconnection of the articular branch. We anticipate that with our improved understanding of IGCs as well as higher quality imaging, new radiologic signs will be discovered in the future.

Author contributions PT, GDR and RJS drafted the manuscript. TM and KA critically revised the manuscript and approved the final version before submission.

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Data availability No datasets were generated or analysed during the current study.

Declarations

Competing interest The authors declare no competing interests.

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