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Transcortical Connection between Periosteal Ganglion and Intramedullary Ganglion Involving the Distal Ulnar Shaft: Demonstration on MRI

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Abstract

A soft tissue ganglion presents as a localized cystic mass about a joint, usually at the wrist and ankle. An uncommon intraosseous ganglion resembling a geode may be seen in a bone adjoining a normal-appearing joint. A rare periosteal ganglion with variable cortical bone erosion occurs at surface of a bone. We report an elderly woman with a palpable periosteal ganglion at the surface of her right ulnar shaft, which had perforated the underlying cortex and connected with a secondary intraosseous ganglion. MRI demonstrated a transcortical connection at the mid ulnar shaft between these two bone ganglia. Both bone lesions were treated surgically without recurrence at 1-year follow-up.

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Introduction

A Soft Tissue Ganglion (STG) arising along a tendon or ligament typically presents as a bump at dorsal surface of wrist, foot or ankle of young persons, especially women [1,2]. Rare Intraosseous Ganglion (IOG) seen incidentally on radiographs as a juxtaarticular small intramedullary lucency resembles a geode; however, without the classic radiographic features of osteoarthritis in the adjoining joint [3-8]. Even rarer is Periosteal Ganglion (PG), located at bone surface, with variable erosion of underlying bone cortex [9-13].

We are demonstrating for the first time on MRI a transcortical connection of a surface PG with an IOG in ulnar shaft of an elderly woman and review the literature.

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Case report

A 66-year-old woman complained of a palpable, mildly tender, non-movable, firm soft tissue mass of 3 weeks duration at her right dorsal mid forearm. The overlying skin was intact. The past medical history was unremarkable, and she denied prior local trauma. Radiographs of right forearm arm revealed a mildly expansile intramedullary lytic lesion with endosteal cortical scalloping in the distal ulnar shaft, but without associated periosteal reaction, cortical break or matrix mineralization (Figure 1). No localized soft tissue mass or calcification was evident. The right wrist was unremarkable. MRI revealed a mildly expansile intramedullary lesion with endosteal cortical scalloping, measuring roughly 8.0 cm in craniocaudal dimension, extended from the mid shaft to intact distal articular surface of the ulna (Figure 2). The intramedullary lesion was isointense (same signal as normal muscle) on T1 weighted image (T1WI), and homogeneously hyperintense on fat-suppressed T2 weighted image (T2WI) with a multilobular appearance due to intralesional septa (Figure. 2 and 3a,b). On post contrast fat-suppressed T1WI following intravenous administration of Gadolinium (Gd), the lesion showed only rim-enhancement (Figure 3c). MRI study also demonstrated the clinically palpable surface soft mass, measuring roughly 4.5 x 3.8 x 3.5 cm (craniocaudal x transverse x AP) in size, was in intimate contact with underlying dorsal cortex of the mid ulnar shaft. This non-enhancing well-marginated surface mass, which was homogeneously mildly hyperintense (MR signal higher than muscle due to presence of mucin) on T1WI, and homogeneously hyperintense on fat-suppressed T2WI, had perforated the cortex and established a direct communication with the medullary cavity of at the mid ulnar shaft through a small transcortical perforation (Figure 3). The right wrist was unremarkable without joint effusion, synovitis, bone erosions or geodes (Figure 2). The identical MRI features of the surface mass at the mid ulnar shaft and the intramedullary lesion in the distal ulnar shaft were consistent with benign fluid-filled cysts, connected via a well-defined small transcortical tract (Figure 3).

Because of her symptoms and risk for pathologic fracture, the patient chose to undergo surgery. Under general anesthesia, an incision was made over the dorsal surface of the distal ulnar shaft. A high-speed burr was used to create a window in the bone through which amber-colored, clear, jelly-like fluid, reminiscent of STG, flowed out under pressure. The long intramedullary lesion in the distal ulna was then curetted and bone grafted. The subperiosteal cystic lesion located at the dorsal surface of the right mid ulnar shaft also was surgically resected after excising the overlying periosteum.

Histopathology of the surgical specimens revealed both the surface and the intramedullary cystic bone lesions were ganglia surrounded by fibrous tissue and lined internally by psudosynovial cells with myxomatous degeneration and scant cellularity (Figure 4). At 1-year follow-up, the radiographs of the right forearm of the asymptomatic patient showed complete healing without recurrence of the lesions (Figure 5).

Discussion

Soft tissue ganglion (STG) filled with gelatinous fluid and surrounded by a fibrous capsule is the most common superficially located myxoid soft tissue mass that lies in close relationship with joint capsule, tendon sheath or tendon at dorsum of the wrist and hand, and the foot and ankle, of young person's [1,2].

Uncommon Intraosseous Ganglion (IOG), containing similar gelatinous fluid, is seen incidentally on radiographs as a benign-appearing, juxtaarticular, intramedullary small lytic lesion with sclerotic margin in bones of ankles and wrists [3-8]. The lesion has mild male preference in contrast with STG, which has female predilection, and is usually is seen in the 4th and 5th decades of life (range 14 years to 86 years; average age, ~50 years). Although asymptomatic, an occasional patient may complain of nagging dull pain at the adjoining joint [4,5]. An IOG can be primary or idiopathic, which occurs de novo; or secondary, which is caused by an STG penetrating underlying bone cortex (4). Previously, it was thought that a growing STG would not cause cortical erosion of adjoining bone, as it was more likely to displace more pliable adjacent soft tissues [3-5]. Despite the commonly held belief that a juxtaarticular IOG has no connection with adjacent joint, such connection between IOG and adjoining joint has been observed occasionally on arthroscopy, suggesting an alternate articular origin of IOG, similar to a geode [6,8,14]. Rarely, fluid-fluid levels in IOG, obviously from internal bleeding, have been observed on MRI [7].

Periosteal Ganglion (PG), containing similar jelly-like material of STG and IOG, is extremely rare. Originally considered as a peculiar form of periostitis, it was described first by Olliers as "*periostitis albuminosa*" (1864), and later by Poncet (1874) as "*ganglion periostale*" [9]. The well-marginated cystic surface bone lesion is located beneath, within, or on the outer surface of periosteum and causes variable pressure erosion of underlying cortex [10-13]. Long bones of lower extremities, especially tibia, are most commonly involved. Other less frequent sites are femur, radius, ulna, fibula and ilium [9-13]. The lesion, usually seen in adult men in the fourth and fifth decades of life, may rarely affect children [9,13]. The slow-growing lesion presenting as a hard nodule on bone surface ranges from a few mms to several cms in size in size, and can be mildly tender, as was seen in our patient [9,11,13,15]. Duration of symptoms varies from a few weeks to several years. Infrequently, the lesion may compress adjacent nerve or blood vessel resulting in local neuropathy or ischemia [10]. Rare bleeding from an adjoining blood vessel into PG has been reported [16]. Occasionally, PG in the region of pes anserine bursa, a common location at proximal tibia, may spontaneously rupture and empty into adjoining knee joint [10,17].

On radiographs, a small PG located on surface of a bone may be missed (as seen in our patient). Occasionally, the surface bone tumor may induce cortical scalloping, erosion or periosteal reaction mimicking a malignant surface bone tumor, such as periosteal osteosarcoma [9-13]. On CT, PG appears as a well-defined hypo- or iso-dense soft tissue surface mass in intimate contact with underlying bone cortex. Periosteal reaction or cortical bone erosions, when present, can be seen with advantage. Recently, transcortical connection between a surface PG and an IOG involving tibia was demonstrated on CT [18]. On MRI, PG is seen as a well-defined cystic mass at bone surface, while an IOG presents as an intramedullary cystic lesion. Both lesions tend to be homogeneously hypo-, iso- or mildly hyper-intense (due to presence of mucinous fluid), relative to normal muscle, on T1WI, and homogeneously hyperintense on fat-suppressed T2WI and STIR (short tau inversion recovery) images (Figure 2 and 3) [10,12,13]. On post contrast fat-suppressed T1WI following intravenous administration of Gd, these cystic bone lesions may show no or minimal rim enhancement of the surrounding

fibrous capsule (Figure 3c) [8-10,13]. Occasionally, non-enhancing septa may impart a multilobular appearance to the lesion on MRI (Figure 2b). MRI can show periosteal reaction, cortical erosion, or cortical perforation, when present (Figure 3). It can demonstrate relationship of PG with adjoining neurovascular bundle and fatty atrophy or edema in affected muscles [10]. If communication of PG with adjoining joint is suspected on MRI, radiographic or MR arthrography can be performed to delineate the connecting tract [17].

Ultrasonography (US) demonstrates PG as a well-defined, fluid-filled anechoic, cystic soft tissue mass on bone surface [8,15]. A large PG can have septations within it contrary to the prior claim that only STG can have such intralesional septa [12].

It is now universally accepted that except for their specific locations, STG, IOG and PG, filled with similar acellular mucous or gelatinous fluid, are histopathologically same lesion [1-9]. Currently, it is believed that these ganglia are formed by focal myxomatous degeneration of connective tissue due to increased fibroblastic proliferation and activity leading to increased secretion of intercellular mucin and increased hyaluronic acid secreted by the flattened connective tissue cells internally lining ganglion wall [1,4,5]. The triggering mechanisms that lead to formation of these ganglion cysts are thought to be repetitive subclinical trauma and local ischemia [4,5]. These cysts lack the synovial lining of true cysts [4].

Earlier reports have stated that a surface PG can cause variable erosion of underlying bone cortex, but it would not perforate and extend into medullary cavity [7-9,14]. However, cortical perforation with connection between surface PG and IOG in distal tibia on CT and US has been demonstrated recently [18]. We are demonstrating for the first time on MRI such a transcortical connection between surface PG and IOG involving ulna (Figure 2 and 3).

We are uncertain whether it was IOG or PG that perforated underlying bone cortex in our patient [4,5,15,17]. However, we believe in our elderly woman with osteoporosis, the small surface PG with increased intralesional pressure gradually eroded and perforated the thin cortex and established direct connection with bone marrow at the mid ulnar shaft resulting in formation of secondary IOG in the distal ulnar shaft similar to formation of IOG by a perforating STG [4]. Although we did not measure intraosseous pressure of the IOG at surgery in our patient, we did observe jelly-like fluid under pressure spurting out of the bone marrow at the excision site. Thus, we suggest that intermittent pumping of the gelatinous fluid contents of the perforating PG into the medullary cavity by muscle contraction in the forearm over time led to formation of the large secondary IOG in the distal ulnar shaft with endosteal scalloping and mild bone expansion, but without sclerotic margin typically seen with an IOG. This lack of sclerotic margin of the IOG of the distal ulna on the radiographs suggested an ominous bone lesion, such as plasmacytoma or lytic metastasis, in our elderly patient (Figure 1). It is less likely that an idiopathic IOG in the distal ulna with normal wrist grew overtime and ultimately penetrated ulnar cortex at a weak point leading to the formation of a secondary small surface PG. However, such a primary IOG in the distal ulnar shaft did not have to penetrate the bone cortex as it had ample room available to extend into and fill the unaffected medullary cavity of the proximal ulna.

Recently, a case in which a PG arising at mid tibial shaft connecting directly with a nearby knee joint through a soft tissue

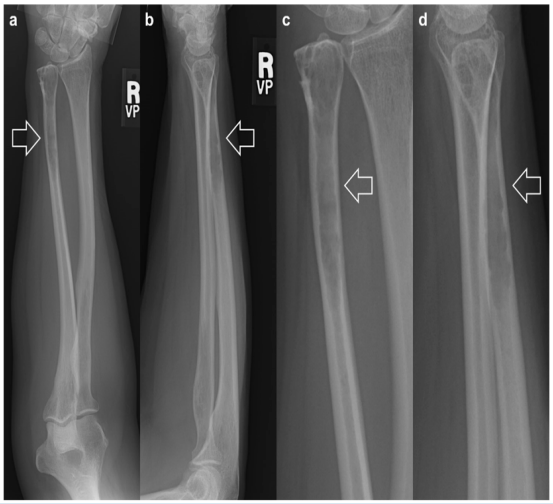

Figure 1: AP **(a)** and lateral radiographs **(b)** of right forearm show a long intramedullary lytic lesion with endosteal cortical scalloping and mild bone expansion in right distal ulnar shaft (arrows).

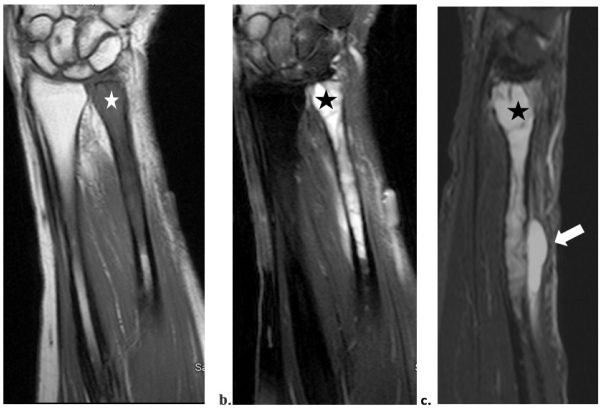

Figure 2: Coronal T1WI MR Image of right forearm shows homogeneously isointense long intramedullary lesion (white star) in a mildly expansile distal ulnar shaft **(a)**. Coronal **(b)** and sagittal **(c)** fat-suppressed T2WI of right forearm show intramedullary lesion (black stars) in distal ulna is heterogeneously hyperintense with multilobular appearance due to intralesional septa. Sagittal fat-suppressed T2WI **c** also shows a well-marginated homogeneouosly hyperintense surface soft tissue mass at dorsal cortex of right mid ulnar shaft (arrow).
Note: Absence of fluid in right wrist indicating no communication between distal ulna and wrist.

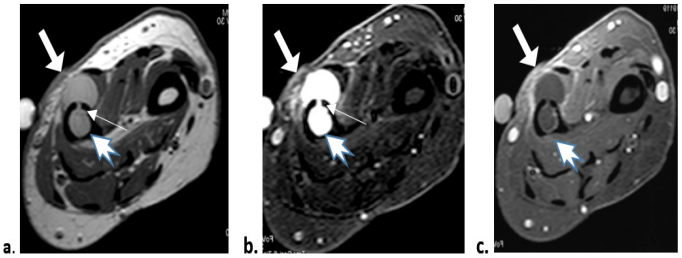

Figure 3: Axial T1W **(a)**, fat-suppressed T2W **(b)** and post-contrast fat-suppressed T1W **(c)** MR images of right forearm show dorsal surface PG (large arrow) communicates with IOG (arrow head) through a transcortical perforation (thin arrow in **a** and **b**) at mid ulnar shaft. These lesions are homogeneously mildly hyperintense to normal muscle (due to presence of mucin in ganglion fluid) on T1WI **(a)**, homogeneously hyperintense on fat-suppressed T2WI **(a)**, and show rim enhancement on post contrast fat-suppressed T1WI **(a)**.
Note: fluid-containing PG, transcortical perforation and IOG have similar MR signal characteristics.

tract in the proximal lower leg has been reported [18]. The connecting soft tissue channel extending from the PG to the joint demonstrated on delayed images of a knee arthrogram was later confirmed on arthroscopy and surgery [16].

Periosteal ganglion can be treated effectively in some cases with aspiration alone, or with corticosteroid injections; however, in most cases, surgical excision of PG is curative [10,11,14]. Excision of adjacent normal periosteum and underlying sclerotic bone is recommended to prevent recurrence [10]. Occasional recurrence of PG following excision may not necessarily be due to inadequate surgery as stimulation of fibroblastic activity and mucoid degeneration of connective tissue in the surgical site can lead to such recurrence [7,10,20]. In case of IOG, curettage and bone grafting or cementation are the preferred treatment [3-6,18,19,21]. Also, when there is an unrecognized connection of PG with an adjoining joint, recurrence following surgery will occur [8,20]. Such rare communication between PG and adjoining joint, if present, can be demonstrated with combined cystogram (of PG) and arthrogram with delayed imaging, or with MR arthrography [16,21,22]. Recently arthroscopy-assisted excision of a small IOG and arthroscopy advocated to detect communicating stalk extending into joint during excision of an IOG have been reported [23].

Conclusion

We have reported an elderly woman with a PG at the surface of her right mid ulnar shaft that had perforated and communicated with medullary cavity through a cortical defect resulting in formation of a secondary IOG in the distal ulnar shaft. We have demonstrated on MRI the transcortical connection between these two bone ganglia, speculated on their pathogenesis and reviewed the literature on ganglion cysts.

Declaration

Informed consent: Informed consent was obtained from the patient.

Conflicts of Interest: The authors declare that they have no conflicts of interest.

Statement of authors' contribution: We certify that all authors have contributed equally and substantially in preparation of this manuscript.

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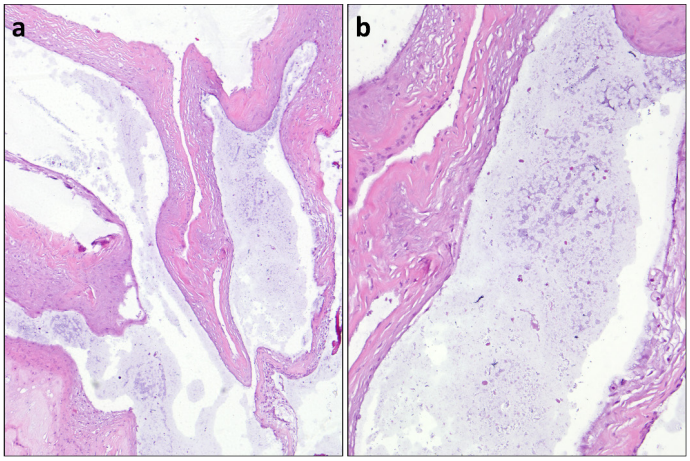

Figure 4: Photomicrographs of periosteal ganglion show a pseudocyst consisting of bland fibrous soft tissue wall and containing myxoid material (**a**, x100; **b**, x200).

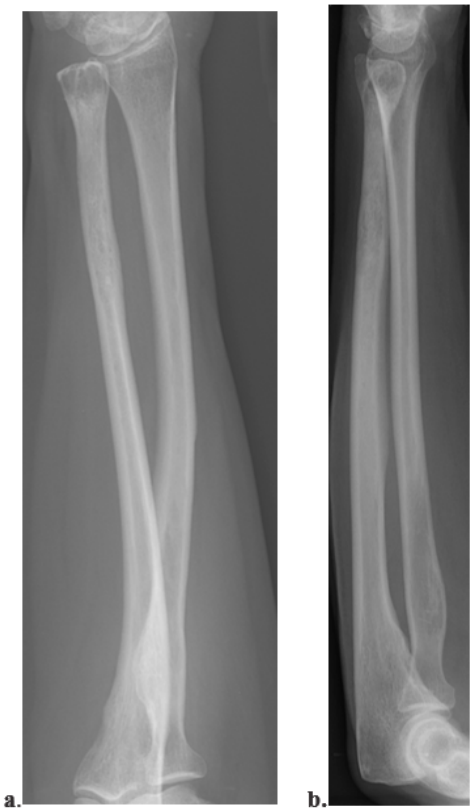

Figure 5: 1-year postoperative follow-up in asymptomatic patient. AP **(a)** and lateral **(b)** radiographs of right forearm show healed surgically treated communicating PG and IOG changes in right ulna without recurrence.

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