The effects of hormonal therapies on tumour growth should be well known by the readers. A significant tumour regression can be expected after discontinuation of progesterone agonist treatments, which can modify the initial surgical strategy.

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Interdural hematomas: an update of literature

Dear Editor,

Interdural hematoma (IDH) is an extremely rare entity. To our knowledge, to date, only 6 cases of interdural hematomas have been reported with varying etiologies, such as iatrogenic (postoperative) hemorrhage, aneurysm rupture, intrauterine asphyxia, and mechanical injury.[1-6] Interdural hematomas can be misdiagnosed as extradural hematoma (EDH) because of their similar clinical presentation and radiologic features.[1] The clinical data of these six cases are displayed in Table I.[1-6]

Calcium deposits may be seeded in cranial hematomas secondary to trauma, subdural effusion, meningitis, or as a sequel of VP shunting.[7] Radiologically demonstrable calcification of chronic subdural hematomas has been reported to range from 0.3% to 2.7%.[8] The term "armored brain" has been used to describe a unilateral or bilateral calcification of chronic subdural hematomas.[7, 9] The patients usually present with headaches, seizure or a visible skull deformity.[8, 10] There are few reports of calcified subdural or epidural hematomas operated upon successfully.[7, 8, 10] However, to our knowledge, there have been no reports of calcification of interdural hematomas in the English literature. This study includes a case of an ossified interdural hematoma undergoing osseous metaplasia with histological confirmation.

A 74-year-old man (case 1) was admitted to emergency service with seizure and right-sided weakness. The patient had been using warfarin due to a previously diagnosed atrial fibrillation. No history of head trauma or hypertension was reported. On the neurological examination, a lethargic appearance as well as a dysarthria with accompanying right hemiparesis was noted. Blood pressure was 130/80 mmHg and had an INR value of 1.38. A Cranial computed tomography (CT) showed a biconvexly shaped extra-axial, hyperdense lesion located on left fronto-parietal convexity (Figure 1A). Epidural or subdural hematomas as well as a hemorrhagic meningioma were considered in the differential diagnosis. Cranial MRI revealed an oval extra axial lesion, which was 45 x 46 mm in diameter and hypointense on all sequences. Diffusion MRI revealed no sign of ischemia ruling out vascular stroke as the cause of the patient's dysarthria. A calcified meningioma was considered as the most likely diagnosis (Figure 1B-D). Surgical treatment was planned and a left frontoparietal craniotomy was performed. Intraoperatively, the lesion was found under a purple colored, smooth and swollen dura mater. The dura was circularly cut around the lesion. The inner dural layer was intact and the lesion was packed between the two dural layers. An indentation of the underlying cortex was noted due to the mass effect of the lesion (Figure 2A). When the dural pack was incised, only a coagulum was found inside. No tumoral tissue was found and a diagnosis of interdural hematoma was made (Figure 2B, C). Postoperative course was uneventful and the patient's hemiparesis recovered after surgery. Histopathological examination was reported as IDH without any vascular malformation or neoplastic process.

A 53-year-old patient (case 2) presented at our outpatient clinic complaining of headache and focal seizures that started three years ago. Epileptic attacks were confined to the face and left arm without loss of consciousness and had become more frequent in the recent months. The patient was started on levetiracetam 500 mg daily 3 months ago by a neurologist. A computed tomography (CT) of the brain was obtained and the patient was referred to our clinic. He reported no visual abnormalities and no nausea or vomiting. Medical history revealed no major trauma or any past history of meningitis or neurosurgical interventions. Neurological examination revealed no abnormality. Head CT revealed an irregularly shaped hyperdense extra-axial mass over the right frontoparietal

TABLE I.—*A summary of the previously reported interdural hematomas.*

	Year	Author	Age	Sex	Etiology	Location	Clinic presentation	Treatment
1	1996	Ragland *et al*.[1]	0	N/A	Intrauterine asphyxia or mechanical injury	Falx cerebri	Normal physical examination	Conservative
2	2004	Miyajima *et al*.[2]	79	M	Idiopathic	Right parietal	Headache	Removal of hematoma with both involved dural layers
3	2009	Eom *et al*.[3]	78	M	Iatrogenic	Right frontoparietal	Headache	Evacuation of hematoma
4	2010	Brock *et al*.[4]	42	F	Aneurysm rupture	Posterior fossa	Headache	Aneurysm clipping
5	2011	Bartoli *et al*.[5]	35	F	Aneurysm rupture	Falx cerebri and right tentorium	Headache, neck stiffness, bilateral abducens palsy	Coiling of aneurysms
6	2012	Baharvahdat *et al*.[6]	51	M	Iatrogenic	Left parietal	Loss of consciousness	Evacuation of hematomas
7	2014	Present Case 1	7th decade	N/A	Idiopathic	Left parietal	Seizure and right hemiparesis	Removal of hematoma with both involved dural layers
8	2014	Present Case 2	5th decade	N/A	Idiopathic	Right frontoparietal	Headache and seizure with accompanying congenital anomalies.	Removal of hematoma and the adhered inner layer of dura

area, which was attributed to a calcified en-plaque mass lesion stemming from the dura (Figure 3C, D). Plain film radiograph of the skull revealed a large patch of calcification extending from the frontal to parietal region on the right side (Figure 3A, B). Both on the radiographs and the CT scan, a slight degree of skull asymmetry was noted which was not conspicuous on physical examination. A cranial MRI was performed. The lesion was isointense on T1 and hyperintense on T2 weighted images (Figure 3E, F). On both sequences however, a stark hypointense halo was visible around the lesion, which was attributed to calcification. Signs of compression on frontal lobe were noted. Coincidentally, the cranial asymmetry was observed to involve the cerebral hemispheres as well. A smaller right ventricle with effacement of the gyri (pachygyria) beneath the lesion was noted. The pachygyria also involved the medial aspect of the frontal lobe (Figure 3F). As there was no sign of brain edema on the MRI or increased intracranial pressure on physical examination, the abnormal cerebral findings were considered to be congenital anomalies. Meningioma, chronic subdural hematoma or metastases were considered in the differential diagnosis. Because of the increasing frequency of seizures and signs of mass effect of the lesion, surgical treatment was planned. A right-sided frontoparietal craniotomy was performed. After removal of the bone flap, the dura was noted to have stone-hard rigidity (Figure 4A-C). The outer layer of the dura was cut and a bony mass was exposed. The mass had no vascularity. It was cut into smaller pieces using high-power pneumatic drill. The fractioned mass had a smooth and glistening surface. It was organized in ossified fine layers and had a white-to-yellow coloration. The ossified layers left the impression of a gradual sedimentary accumulation. The mass was strictly adhered to the inner layer of the dura (Figure 4B, C). However, the arachnoid membrane over the brain was intact and was not adhered to the mass. Therefore the inner layer of the dura was removed along with the mass (Figure 4C). The remaining outer layer was used for dural repair during closure. The recovery of the patient was uneventful. Postop MRI revealed total removal of the mass and the patient was advised to continue levetiracetam.

Histopathological study (Figure 5) of serial sections acquired from the removed dural mass revealed three consecutive zones, which could be designated as organizing (Figure 5A), calcifying (Figure 5B) and ossifying zones (Figure 5C), respectively. A rich vascular network embedded in a collagen rich matrix characterized all three individual zones. Homogenous mineralization was a conspicuous feature of the calcifying zone. An even more interesting finding was the tiny island of mature adipose tissue ensheathed by a segment of calcified spongiotic bone tissue (Figure 5C). There were no perceptible neoplastic features within the specimen and the findings were interpreted to be a mesenchymal metaplasia, specifically osseous metaplasia. Therefore, in light of the intraoperative and pathological findings, the mass was diagnosed as a calcified chronic interdural hematoma with osseous metaplasia.

Cranial dura mater has two layers: outer (periosteal) layer and inner (meningeal) layer.[1-4] Although these two layers are attached to each other, they are separated around the dural sinuses, Meckel's cave, and sella turcica.[1-3] Interdural hematomas and tumors are known to occupy these interspaces.[1, 2] However, in regions where the two layers are attached, IDHs are extremely rare. To our knowledge, there are only six reported cases in the literature.[1-6]

Several etiologies have been implicated. Ragland *et al.* reported the only pediatric case of IDH at falx cerebri, which was possibly due to an intrauterine asphyxia or mechanical injury.[1] Aneurysm rupture has been associated with IDH in two cases reported by Brock *et al.*[4] and Bartoli *et al*.[5] Also there are two reported cases of IDH, which occurred after a previous cranial surgery.[3, 6] However, Eom *et al.* could not show a definite reason for a contralateral IDH developing after a previous surgery.[3]

Even though the patient was on anticoagulation therapy in our first case, it was not clear how the IDH developed, as the patient's INR value was 1.38. The patient also had no history of had trauma. Similarly, there was no trauma history or anticoagulation in the second case. Miyajima *et al*. suggested that a high amount of fibroblast involvement especially in inner layer of dura may cause inflammatory process, which may lead to hemorrhage.[2] Eom *et al* suggested that IDH could occur in elderly patients because of strict attachment of periosteal dura to the bone which makes it easier for the bleeding to reach the interdural space.[3] The latter explanation seems rational for both of our presented cases.

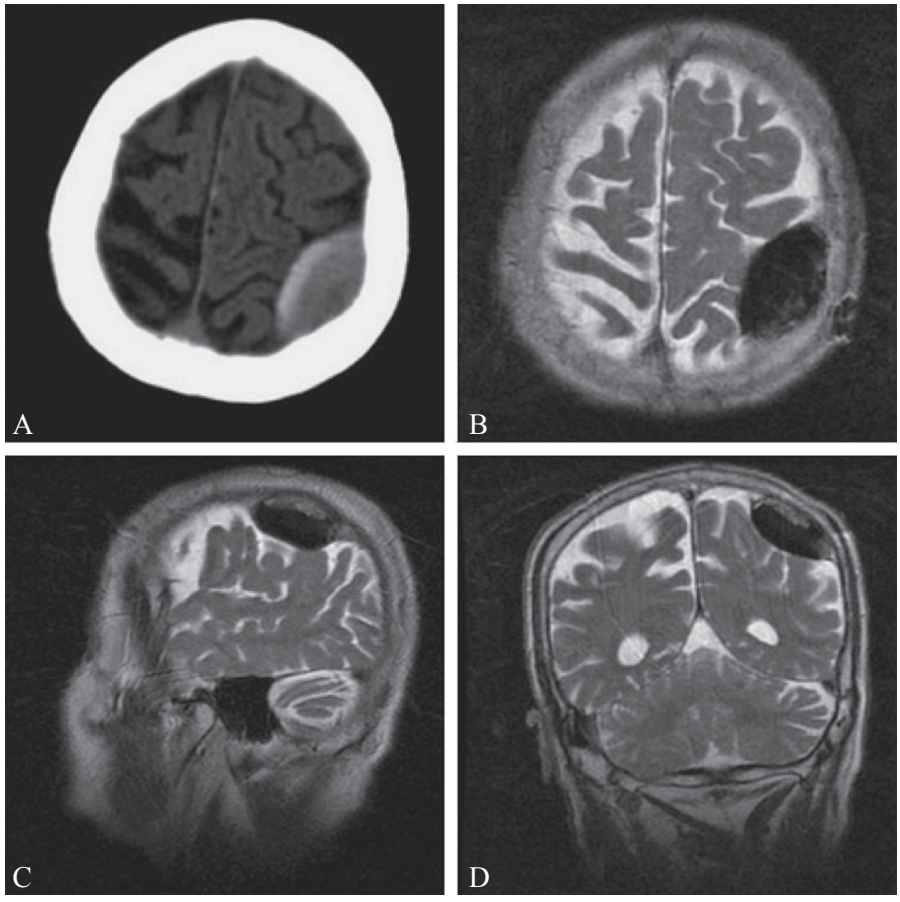

Figure 1.—Case 1. CT scan showed a biconvexly shaped hyperdensity (A) and MRI showed an interdural hematoma, the "Chinese Dumpling" view (B-D).

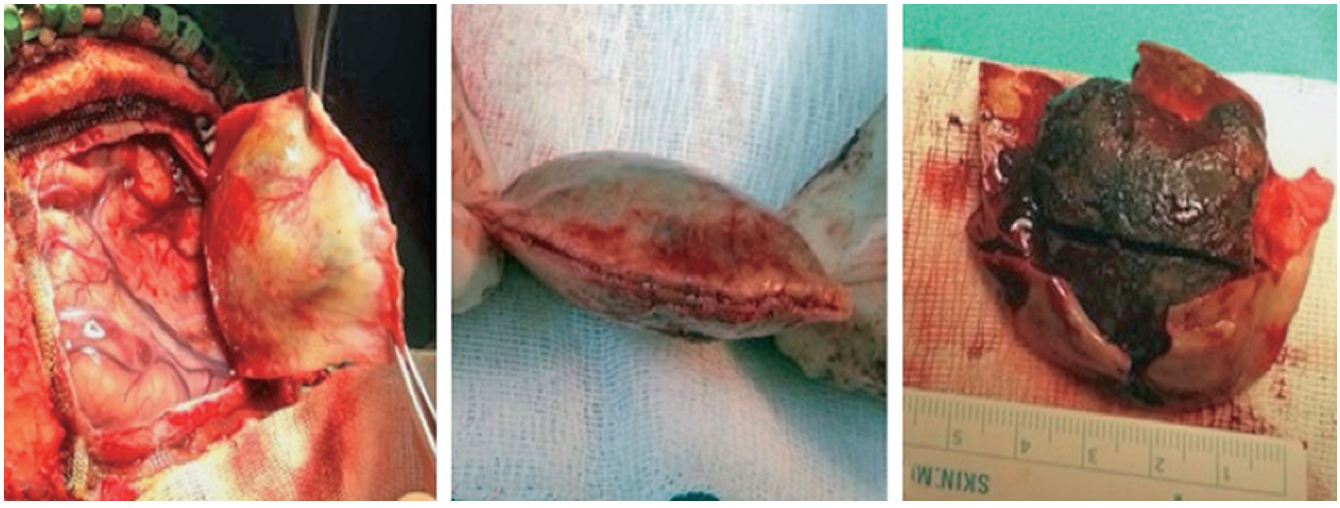

Figure 2.—Case 1. Perioperative view of IDH.

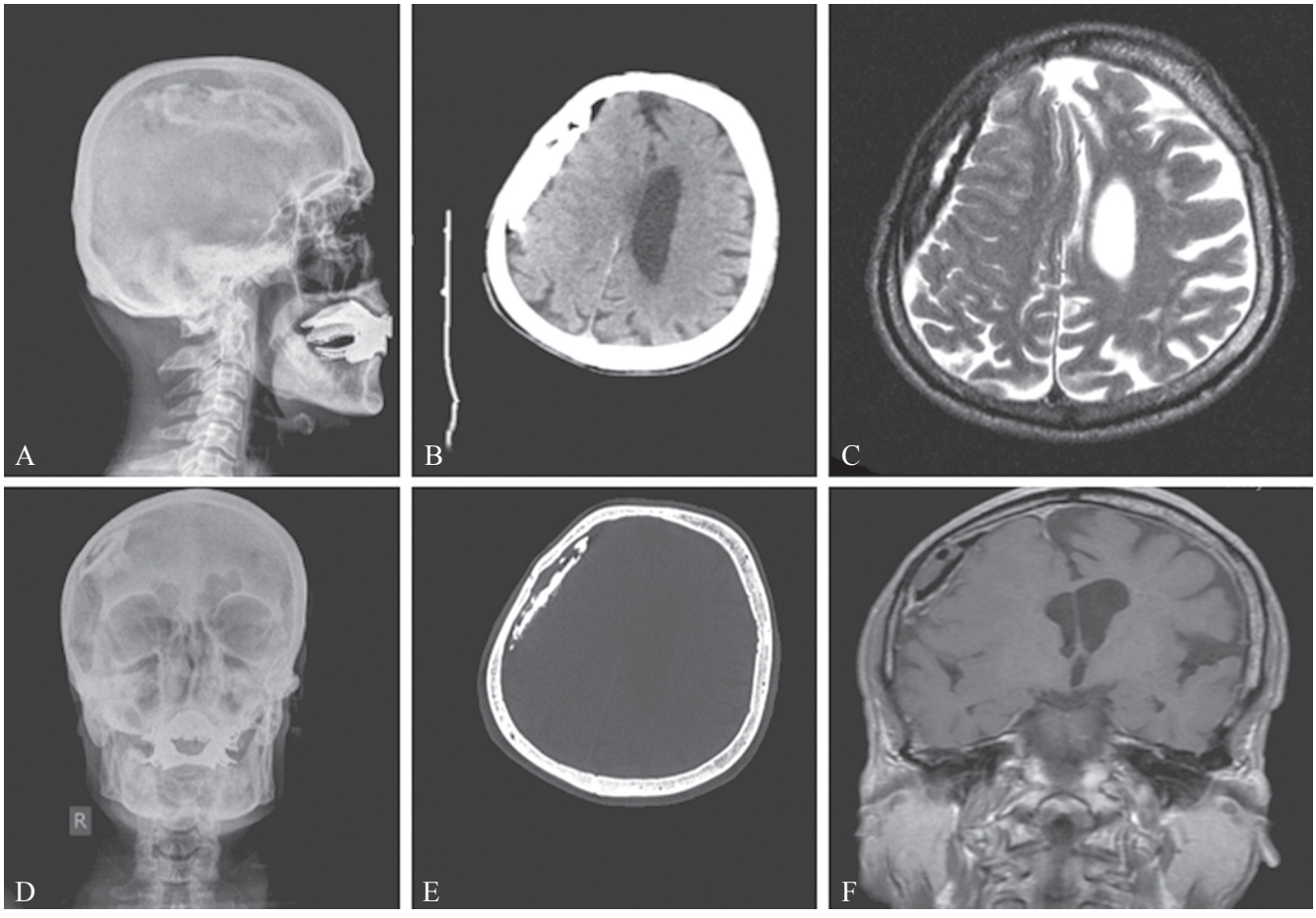

Figure 3.—Case 2. Plain film radiograph showed calcification at the right frontoparietal region (A, B). CT scan showed a hyperdense extra-axial mass (C, D). Brain MRI showed that the lesion was isointense on T1 and hyperintense T2 weighted images (E, F).

Clinical presentation varies from simple headache to loss of consciousness.[1-6]However, neck stiffness and ophtalmoplegia have been reported with associated aneurysmal ruptures.[4, 5] The main symptoms in our cases were hemiparesis and focal seizures. The ossified hematoma in case 2 was accompanied by congenital developmental cerebral abnormalities.

Similar to IDHs, a biconvexly (lenticular) shaped hyperdensity adjacent to the skull is the classic CT scan appearance of EDH.[1, 2] Therefore CT scans are mostly unsatisfactory to differentiate IDH from EDH.[1] However, MRI can readily display a bleeding between the two layers of dura. The first case displayed the classical MRI appearance, which has been previously described as a "Chinese dumpling".[2]

MRI study of the second case also revealed accompanying congenital anomalies including cranial and ventricular asymmetry with gyral effacement in the frontal lobe on the lesion side. Ragland *et al*. also reported the use of ultrasound imaging for the diagnosis of a falx interdural hematoma discovered during a routine obstetrical ultrasonography.[1]

Calcified chronic subdural hematomas have been reported in both pediatric and adult patients.[11, 12] In pediatric cases calcified subdural hematomas are frequently associated with previous ventriculoperitoneal shunt surgeries, and the term "armored brain" has been used to describe them.[12] Calcifications can be unilateral, bilateral, partial or complete. Both inner and outer layers may be calcified. Thickness of calcification can also vary.[11] The calcifications have been reported mostly along the inner surface of dura mater.[7] An underlying metabolic abnormality or poor circulation with delayed resorption of the hematoma fluid may cause microscopic seeding of calcium deposits within the membranes of the hematoma.[11] Calcium deposition may proceed to extensive calcification and in further stages, ossifications may occur. Similarly, in our case 2, the base of the hematoma displayed ossification with gradual transition to the calcified and organized layers above. These findings may indicate that calcium deposition (mineralization) had started in the inner layer and gradually transformed into ossification by means of osseous metaplasia.

The precise mechanism of mesenchymal metaplasia with bone

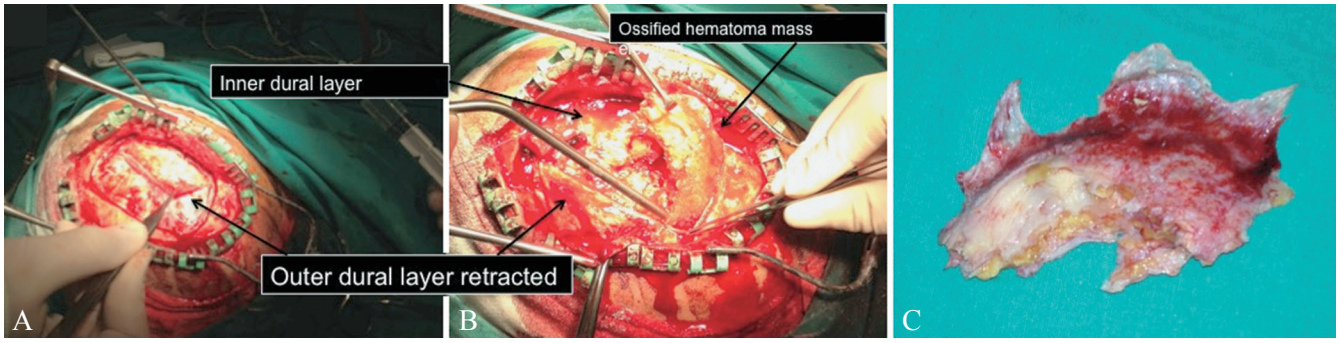

Figure 4.—Case 2. Perioperative view of ossified IDH. The ossified mass was lodged between the inner and outer layers of the dura (A, B). Please note the adipose tissue at the base and the overlying sedimentary layers of ossification.

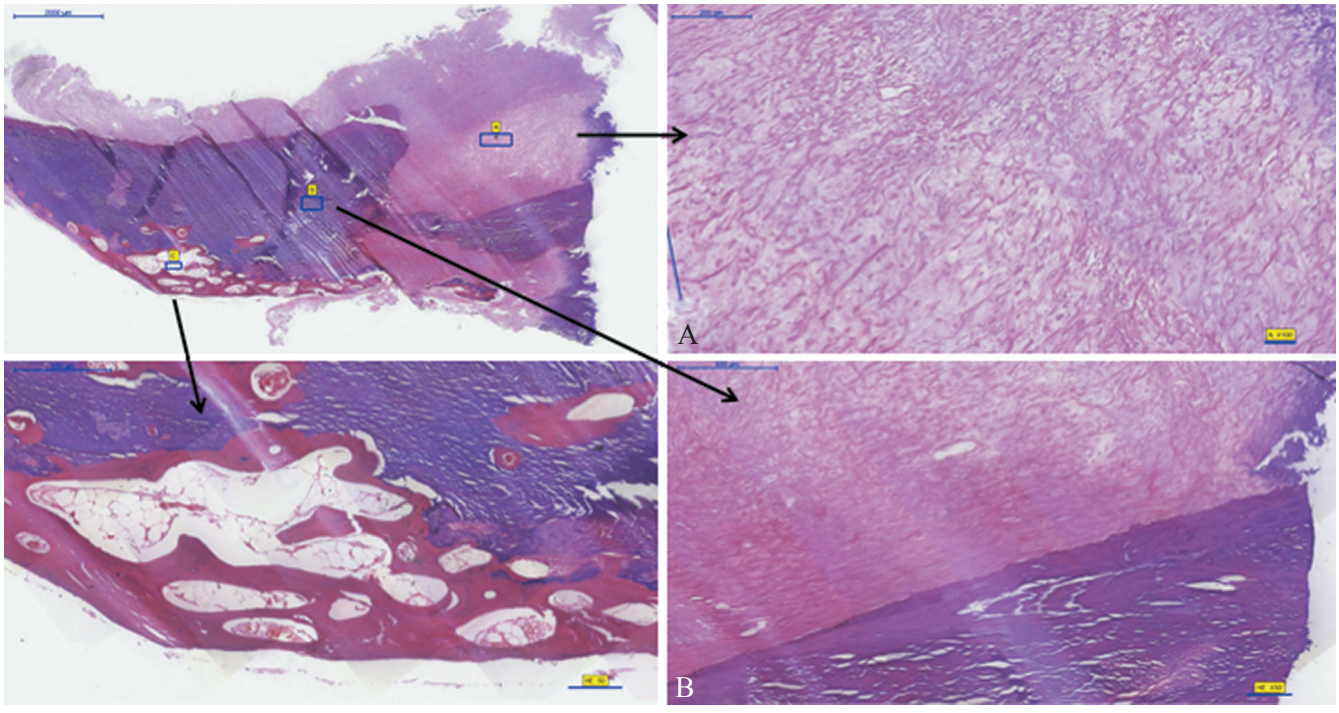

Figure 5.—Case 2. Histological examination of the removed hematoma mass revealed three distinct zones. All three zones are encoded as: A) organizing zone; B) calcifying zone; C) ossifying zone (Hematoxylin-eosin, x40 original magnification). A) Higher magnification of a highly vascular network embedded in collagen rich matrix (Hematoxylin-eosin, x100 original magnification); B) calcifying zone adjacent to the vascularized zone in chronic hematoma. (Hematoxylin-eosin, x50 original magnification); C) spongiotic bone tissue containing mature adipose tissue was found within the chronic hematoma, which is a stigma of osseous metaplasia (Hematoxylin-eosin, x50 original magnification).

formation is essentially still unknown. However, mesenchymal metaplasia is not an uncommon event and is mostly detected in extensive soft tissue hematomas. Metaplasia is a reversible change in which one differentiated cell type (epithelial or mesenchymal) is replaced by another cell type. It represents an adaptive transformation of a vulnerable cell type to better withstand an adverse environment. In soft tissues, osseous metaplasia occurs after extravasation of blood cells, which in turn trigger a local inflammatory process resulting from a series of mediator release. Then, overt angiogenesis accompanied by an exuberant fibroblastic proliferation may occur. Rarely, this astounding fibroblastic proliferation, *i.e.*, metaplasia, is transformed into newly formed mature connective tissue elements, such as cartilage, bone or bone marrow. Osseous metaplasia can be conjectured as the end result of the innate tendency of mesenchymal cells to differentiate in response to chronic inflammatory reaction.[13] Similar to our patients, two of the six cas-

es reported previously, underwent surgical treatment due to signs of brain compression. Two IDH cases with aneurysm rupture were treated via coiling and clipping as there was no need for hematoma evacuation. For ossified subdural hematomas, surgical intervention is not recommended for asymptomatic cases. However, successful surgical removal has also been reported for symptomatic cases.[11]

In conclusion, IDH is an extremely rare situation. Differentiation of IDH from EDH may be difficult because of their similar clinical and radiologic features. However MRI may be more beneficial in determining the exact location of the hematoma and during the differential diagnosis including meningiomas. Ossification of IDH is possible. This report documents unequivocal features of ossification in an intradural hematoma resulting from mesenchymal metaplasia.

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Synovial-type giant cell tumors of the axial spine

Dear Editor,

Synovial-type giant cell tumors (SGCTs) arise from synovial joints and tendon sheaths and go by many names, including giant cell tumors of the tendon sheath, tenosynovial giant cell tumors, or pigmented villonodular synovitis depending on anatomic location and histologic pattern. Two histologic subtypes are described; localized SGCTs are typically more circumscribed and less likely to recur after treatment, while the diffuse form of SGCT displays a more aggressive growth pattern. Regardless of subtype, these tumors generally arise in appendicular joints, most commonly in the hand, and rarely occur in the spine. When presenting in the spine, they typically localize to the cervical spine of younger- to middle-aged individuals. Here we present an unusual case of a symptomatic SGCT in an elderly individual that was successfully treated with surgical *en-bloc* resection.

A 79-year-old female with a history of breast cancer, previously treated with lumpectomy and radiation presented to her oncologist with symptoms of weight loss, anorexia, poor mood, and back pain. Positron emission tomography-computed tomography (PET/CT) was performed as part of the oncology work up and demonstrated a focal area of increased metabolic activity in the lumbar spine concerning for metastasis.

Subsequent magnetic resonance imaging (MRI) of the lumbar spine was performed revealing a homogenously enhancing 12×16 mm lesion arising from the right L3-4 facet joint (Figure 1A-C), mild lumbar stenosis, and a Grade I spondylolisthesis. The patient's spine-related symptoms were mild, intermittent low back pain and she denied symptoms of neurogenic claudication or radiculopathy. Her neurological exam was normal with a full range of motion of the lumbar spine.

Given her cancer history, the decision was made to proceed with operative biopsy and resection of the lesion. Under general