

## Concurrence of a solitary vertebral osteochondroma and spina bifida occulta: Case report

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### Abstract

Osteochondromas are the most common benign tumor of the bone. They occur in two forms as solitary and hereditary multiple form. Osteochondromas are generally located on the metaphysis of the long bones.

Spinal osteochondromas are rare and seldom cause cord and root compression and also be asymptomatic. In the diagnosis of osteochondromas, radiological methods are very important. In this case report, we study the imaging findings in a patient with a solitary vertebral osteochondroma and spina bifida occulta.

**Keywords:** Spinal osteochondroma, spina bifida occulta

### Introduction

Osteochondroma, also termed osteocartilaginous exostoses, are benign lesions, often considered to be the most common bone tumor, which represent a developmental physical growth defect [1]. They are thought to form when fragments of epiphyseal growth plates or cartilage herniate through the periosteal covering of bones. As these cartilaginous fragments develop, they undergo ossification and maturation, during which these lesions expand [2].

They account for 10±15% of all bone tumors and apparently occur in 3% of the population overall [3]. It typically occurs in young adolescent patients and typically does not develop after skeletal maturity [4]. Osteochondroma of the spine is rare and comprises only 1.3–4.1% of all osteochondromas [4]. Only 0.5–1% of spinal osteochondromas may develop insidious but progressive symptoms of myelopathy, radiculopathy, or both, resulting in serious neurological sequelae if not diagnosed and treated early [4]. Although they are rare, when they do arise in the vertebral column, they most commonly present in the cervical vertebrae and almost always occur in the posterior elements of the vertebrae [5].

Solitary osteochondromas show a predilection for the metaphyses of the long tubular bones, especially the femur, humerus, and tibia, but have been described in virtually every part of the skeleton [3]. Multiple osteochondromas occur as part of a rare familial syndrome with autosomal dominant inheritance, termed hereditary multiple exostoses [3]. Spinal osteochondromas rarely cause neurological symptoms [3]. We report a solitary intraspinal vertebral osteochondroma presenting with no neurological symptoms.

### Case Summary

A 17-year-old male presented to our radiology department on 23.05.2025, with a 3-year history of a solitary painless mass at the mid back which was increasing in size since past 2 months. There was no history of trauma, fever, neurological defects or previous surgeries. The patient had no significant past medical history or family history of bone tumors.

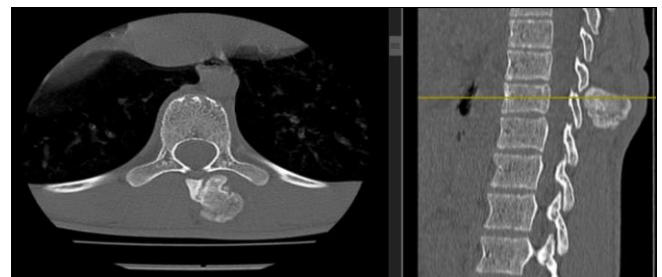
On presentation, general examination was unremarkable with vital signs within normal limits.



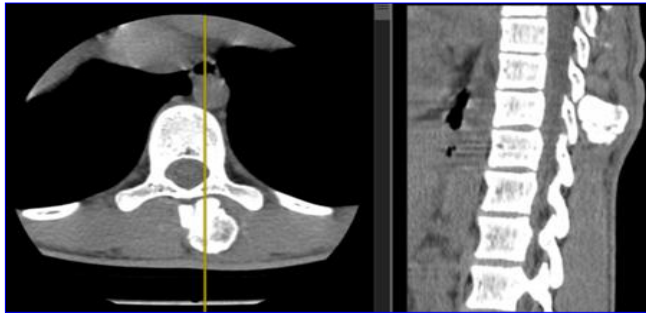
**Fig 1:** Pre-operative Lateral radiograph of thoracolumbar spine showed an ill-defined radiodensity present at the back at the level of D10 vertebral body (blue arrows)

Initial radiograph of (Fig. 1) showed evidence of an ill-defined radiodensity present in the back posterior to the D9 vertebral body.

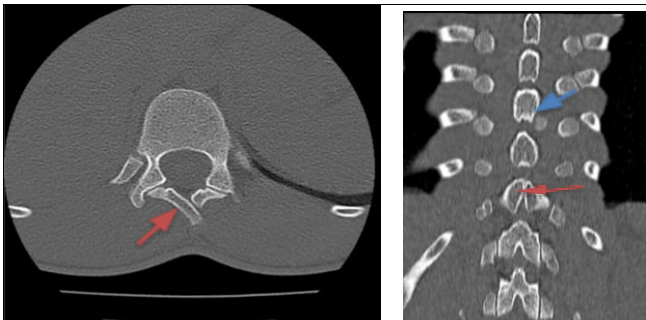
For further characterization of the lesion, the patient underwent a Non-Contrast CT and a Contrast Enhanced MRI of the thoracolumbar spine.



**Fig 2:** NCCT of Dorsolumbar spine axial section at the level of D10 vertebral body and sagittal reformatted image in bone window depicting the nature and extent of tumor. It appears as a well-defined bone density mass with a cortex and medulla arising from the tip of spinous process of D9 vertebra (red arrow).



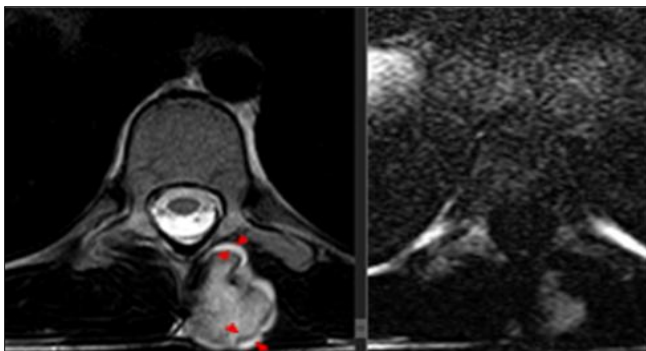
**Fig 3:** NCCT of Dorsolumbar spine axial section at the level of D10 vertebral body and sagittal reformatted image in soft tissue window confirming bone window findings. The lesion appeared to be epicentered in the intermuscular plane



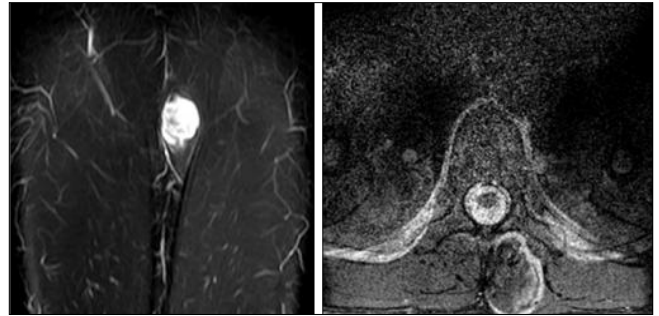
**Fig 4:** NCCT of Dorsolumbar spine axial section at the level of D11 vertebral body and coronal reformatted image in bone window showing non-fusion of left lamina with spinous process of D11 vertebra – paraspinous cleft (red arrow). Blue arrow points at the posterior most aspect of the tumor



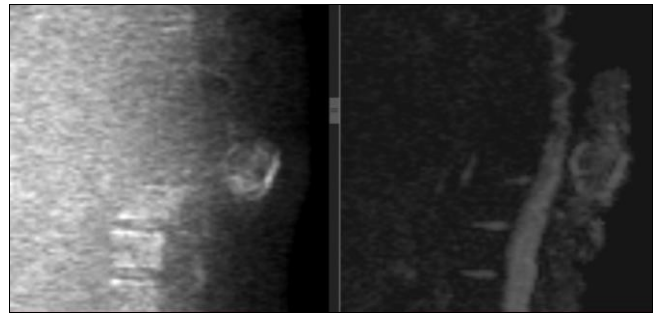
**Fig 5:** The above-mentioned lesion appears heterogeneously hyperintense on Sagittal MR T1WI and T2WI sequences. A thin hypointense chondral cap is noted around the lesion (characteristic of osteochondroma)



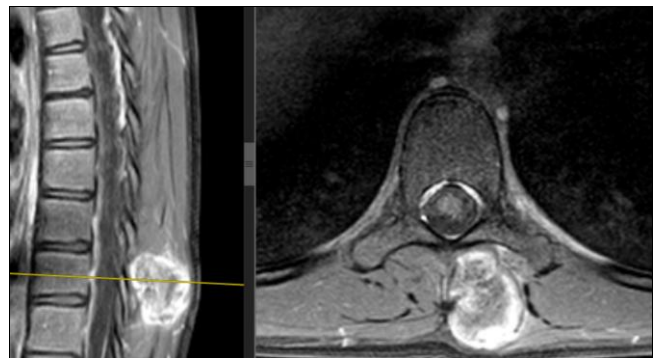
**Fig 6:** Axial MR T2WI and T1WI sequences highlighting the osteocartilaginous cap covering (red arrowheads) the margins of tumors evenly on all sides. In this case, the cap thickness was measured to be ~3.5 mm



**Fig 7:** The tumor appears heterogeneously hyperintense on STIR sequences and shows foci of blooming on GRE sequences



**Fig 8:** DWI sequences & ADC images confirmed patchy areas of facilitated diffusion within the tumor substance. Thus, ruling out malignant etiology



**Fig 9:** On contrast enhanced MRI the tumor showed peripheral enhancement with enhancement in the central bony part



**Fig 10:** Post-operative Lateral radiograph of thoracolumbar spine shows no tumor in the previously documented site confirming en masse excision of the tumor

CT scan revealed a mass with exostosis configuration arising from the tip of spinous process of D9 vertebral body, extending to left paraspinous area (Fig. 2-4).

Magnetic resonance imaging (MRI) revealed that on the tip of the spinous D9 vertebrae, there was a multilobular lesion 3.4 x 2.5 x 4.2 cm with a thin chondral cap measuring 3.5 mm in maximum thickness, not compressing to the spinal canal, showing posterior expansion and extending from D7 to D10 vertebrae and appearing hyperintense on T1WI, T2WI, STIR sequences with central areas showing blooming on SWI and no restriction on DWI sequences, thus confirming the benign nature of the tumor with no sign for neoplastic transformation. (Fig. 5 –7).

No evidence of cortical destruction, lucent lesions and thickened cartilaginous cap ruled out malignant degeneration<sup>[2]</sup>.

Also, there was evidence of non-fusion of left lamina with spinous process of D11 vertebra – paraspinous cleft – suggesting a posterior vertebral fusion anomaly (spina bifida occulta).

Ligamentum flavum, bilateral sacroiliac joints and spinal cord was normal.

En masse excision of the tumor was performed and specimen sent for HPE.

## Discussion

Spinal osteochondroma, an uncommon entity, is thought to arise from excessive cartilaginous tissue of secondary ossification centers present in the posterior elements of the spine, most commonly the tip of the spinous or transverse process<sup>[4]</sup>. Though any part of the vertebrae can be involved, they're generally seen at the cervical and thoracic regions with the posterior arch elements being most commonly involved<sup>[4, 7]</sup>.

Exact pathogenesis is unknown but there have been studies showing genetic mutations in the exostosis gene, causing accumulation of heparan sulfate proteoglycans within the cytoplasm, which prevent them from participating in the normal diffusion of Indian hedgehog ligands in the extracellular space. This results in loss of normal polarity, causing chondrocytes in the growth plate to grow in the wrong direction<sup>[9]</sup>. Osteochondromas have a varying structure and may appear in sessile forms, with a broad base of attachment or present as a pedunculate mass, with a narrow attachment<sup>[2]</sup>.

The incidence of spinal involvement in HME is about 7–9%, whereas that in solitary osteochondroma is about 1–4%.<sup>4</sup> SOC affects male patients more frequently than female patients, with a sex ratio of 2.5:1<sup>[7]</sup>.

They are most commonly sporadic, but can be associated with syndromes like Hereditary Multiple Exostoses. Sometimes they also arise after fractures, trauma, radiation and hematopoietic stem cell transplantation<sup>[9]</sup>. There have been very few cases in the literature that have shown any association or correlation between spinal osteochondroma and spinal bifida defects. In the first and second decades of life, osteochondromas can develop during the growing period of the skeleton system<sup>[3]</sup>.

Osteochondromas do not grow after skeletal maturity, and they usually manifest clinically in young adolescent patients<sup>[4]</sup>. Osteochondromas enlarge from growth at the cartilage cap, identical to a normal physal plate.

Osteochondromas producing clinical symptoms late in adult life are extremely rare. Complications like spinal cord

compression is unusual because the majority of these lesions grow out of the spinal canal.

Symptomatic lesions need to be treated surgically, and good results following total resection have been reported<sup>[4]</sup>.

The recurrence rate is extremely low but rarely regrowth after subtotal excision has been reported<sup>[6]</sup>.

Sudden growth of an osteochondroma may indicate malignant transformation, which occurs in 1% of solitary cases<sup>[6]</sup>.

**Malignant Degeneration:** The risk of malignant transformation of multiple osseocartilaginous exostoses is well known. In contrast, a malignant tumor developing from a SOC is observed much more rarely. The frequency of degeneration is estimated at about 1% in SOC and 10 to 15% in multiple exostosis for all tumor locations<sup>[7]</sup>.

Osteochondromas are commonly imaged using modalities such as radiographs, CT, and MRI. For spinal osteochondromas (SOC), radiographs are typically of low diagnostic yield.

Radiographs may detect large, heavily calcified lesions, but smaller lesions may be easily missed due to the complexity of overlapping structures on a typical spine radiograph. Albrecht *et al.* suggested only 21% of osteochondromas were considered diagnostic with radiographs. Not much information about the relationship between the tumor and other structures are given by the radiographs<sup>[8]</sup>.

On CT, osteochondromas demonstrate classic cortical and medullary continuity with underlying bone.

The calcified cartilage is high density at CT, with low-density yellow marrow and an intermediate density cartilage cap<sup>[2]</sup>. Although, typically the cartilage cap is difficult to appreciate on CT.

The detection of symmetric calcification with CT scanning is important in preoperative diagnosis<sup>[8]</sup>.

On MRI, osteochondromas can present with varying signal characteristics, depending on the size of the lesion, the amount of marrow, and degree of cartilage calcification. The medullary and cortical components of the osteochondroma mimic normal bone on MRI<sup>[2]</sup>.

Marrow usually demonstrates high T1 signal intensity with intermediate intensity on T2-weighted imaging. The cortex is typically low signal on all sequences. Signal from the cartilage cap differs depending on degree of cap mineralization<sup>[2]</sup>.

Typically, intermediate to low T2 signal is seen in areas of the cartilage cap, and high T2 signal is observed in nonmineralized cartilage. T1 signal is intermediate to low intensity within parts of the cartilaginous cap. MRI is well suited to evaluating the effect of an osteochondroma on surrounding structures, especially the spinal cord and nerve roots.

With intravenous contrast medium, peripheral and septal contrast enhancement is seen<sup>[2]</sup>.

All above findings were appreciable in our patient, who was having a lesion with similar imaging characteristics as above.

Furthermore, in our case, there was also presence of left paraspinous left in the left lamina of D11 vertebral body, suggesting a spina bifida occulta<sup>[10]</sup>. There have been very few reports of co occurrence of a spina bifida spectrum defect and vertebral osteochondroma in literature.

In our case, the tumor was removed and Histopathological examination of excision biopsy (both performed at our

healthcare setup) proved the diagnosis of osteochondroma. The paraspinal defect was not operated on.

### Conclusion

Spinal osteochondromas are rare entities and clinical manifestations due to spinal cord compression by the tumor are rarely seen. Total removal of the tumor is the choice of treatment if there is neurological compromise or quality of life is affected. Thorough examination and relevant imaging must be carried to diagnose its complications or other concurring pathologies.

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