Incidental vertebral lesions

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Incidental vertebral lesions on imaging of the spine are commonly encountered in clinical practice. Contributing factors include the aging population, the increasing prevalence of back pain, and increased usage of MR imaging. Additionally, refinements in CT and MR imaging have increased the number of demonstrable lesions. The management of incidental findings varies among practitioners and commonly depends more on practice style than on data or guidelines. In this article we review incidental findings within the vertebral column and review management of these lesions, based on available Class III data. (DOI: 10.3171/2011.9.FOCUS11207)

Key Words • incidental finding • vertebra • congenital • os odontoideum

Incidental lesions are commonly detected with imaging of the spine in asymptomatic individuals.14,31 The advent or refinement of new imaging techniques carries with it the challenge of discerning these incidental findings from pathological lesions. Magnetic resonance images of the spine are obtained with increasing frequency, possibly because the incidence of back pain is increasing20 and the population is aging.10 Trauma patients are also increasingly undergoing evaluation with CT scanning, effectively replacing plain radiography.7,11,29 Axial imaging also increases the amount of data to review and the probability of encountering an incidental lesion.43 For the purpose of this review, incidental findings are defined as unexpected lesions that are unrelated to the original reason for obtaining the scan. There are two diagnostic pitfalls when dealing with incidental findings. At one end of the spectrum, failure to recognize a serious condition masquerading as an incidental finding can lead to a diagnostic delay. Also problematic is the failure to recognize a process as benign, and in the process exposing a patient to an unnecessary intervention such as a biopsy. As with any screening test, the lower the pretest probability, the higher the likelihood of a false positive finding. With the increasing use of spinal imaging, it is important to have an algorithm for managing incidental spine lesions.

In this article, we review the imaging characteristics and management of incidental findings in the osseous spine. Some incidental findings such as small hemangiomas and enostoses are asymptomatic and clinically insignificant. Others are potentially more serious than the original diagnosis prompting the study. The finding of anemia, or osteoporosis, for example, is incidental only in the sense that it may not relate to the indications for obtaining the scan in the first place. The term incidental should not be used to suggest that the findings are any less important.

The Normal Vertebral Body

Understanding the nuances of normal vertebral body anatomy is necessary to prevent mistaking normal anatomy as lesional. The normal vertebral body cancellous bone has the T1 and T2 signal characteristics of bone elsewhere. Cortical bone is hypointense to cancellous bone on T1- and T2-weighted imaging. The cartilage endplate is hypointense to marrow on T1- and T2-weighted imaging. With the normal aging process, bone marrow undergoes an increase in T1-weighted signal intensity, corresponding to an increase in marrow fat content. While this can certainly be accelerated by factors such as radiation treatment, the process in and of itself falls within the confines of normal aging.

Additionally, venous anatomy must not be mistaken for a pathological process. Embryologically, the vertebral body is composed of a fusion of a rostral and caudal sclerotome, and a midvertebral structure can persist. There is, for example, a midvertebral body vein that, when large, can be mistaken for a vascular abnormality or even a fracture (Fig. 1).

Congenital Malformations

Congenital malformations of the spine are commonly encountered in clinical practice. These are generally benign, but on occasion can be associated with severe problems such as spinal cord injury, in the case of os odontoideum. It is convenient to think of congenital vertebral body variants in terms of their corresponding embryological dysfunction, such as failure of ossification or abnormal segmentation. Although this approach may not suffice to encompass the myriad of complex congenital malformations, it applies to more common, straightforward incidental malformations.
Abnormal Segmentation

Block Vertebra. Two or more vertebrae can fail to segment, resulting in an immobile segment of the spine (Fig. 2). This tends to occur at upper cervical levels. The clinical significance of this malformation remains uncertain. This condition is commonly observed in Klippel-Feil syndrome, in which there is a congenital fusion of 2 or more cervical vertebrae. It is possible that an increase in either intradiscal pressure or movement adjacent to a block vertebra might contribute to accelerated degeneration of adjacent intervertebral discs. However, the effect may not be large or clinically significant, and the natural history is not well defined. Figure 3 illustrates a case of mild central canal stenosis caudal to an unsegmented upper cervical spine. In a radiographic study of 25 patients with a block vertebra, there was a significant decrease in the height caudal to the block vertebra, but not to the disc height rostrally. A kinetic analysis of the adjacent levels demonstrated no hypermobility in rotation or translation. The effects of a congenitally fused vertebra on the adjacent spine may differ from those of a surgical fusion. In surgical fusion, the adjacent levels may already be degenerated, and surgery itself may have an effect on adjacent levels. Thus, it appears reasonable to inform patients with congenital fusion about the possibility of premature degeneration of adjacent levels, but the effect is largely unknown and is likely minor. Persons with Klippel-Feil syndrome and cervical stenosis may be at increased risk for spinal cord injury after minor trauma as a result of hypermobility of the various cervical segments. Activity modification should be considered in patients Klippel-Feil syndrome who are at high risk for neurological compromise, such as those with severe central canal stenosis or stenosis is unknown.
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steno…s or myelopathy. Discovery of congenitally fused segments should prompt a systematic investigation because persons with Klippel-Feil Syndrome often have cardiac and/or genitourinary congenital anomalies as well.39

Aberrated Number of Vertebra. Approximately 20% of the population has an aberrant number of lumbar vertebral bodies.28 This has a significant implication in clinical practice, with the most obvious being the accuracy of using numbered vertebral levels for surgical localization. When the number of vertebral segments is variant or even indeterminate, surgical localization has the potential to be misleading and not consistent between practitioners. In a large series of patients undergoing whole spine MR imaging, the number of lumbar vertebral bodies varied between 3 and 6.28 While these findings are incidental, they can have profound consequences if care is not taken to accurately and consistently localize the correct surgical level.

Failure of Chondrification or Ossification

Early in embryonic development, mesodermal cells form somites lateral to the notochord. The paired somites divide into a medial part (the sclerotome) and a lateral part (the dermomyotome). Mesenchymal cells from each sclerotome pair migrate to the location of the presumptive vertebral body, giving rise to 3 chondrification centers per side. Once vertebral chondrification is complete, primary ossification centers appear: 1 ventrally and 2 at the level of the pedicles.3,45 Failure of formation of 1 of the chondrification centers or subsequent failure of ossification results in the absence of part of a vertebra. The more severe malformations are not incidental findings and present with a deformity at birth or during infancy. Absence of an anterior chondrification center, for example, gives rise to a hemivertebra or kyphosis (Fig. 4). More mild developmental anomalies can present as incidental findings as discussed below.

Synchondrosis Mimicking Fracture. Knowledge of normal embryological development is essential to avoid misinterpretation of normal epiphyses or synchondroses as pathological conditions. Ossification of the vertebra continues after birth, and the synchondroses visible on imaging in young children can be mistaken for fractures.3,25 The diagnosis of a fracture in the immature spine can be problematic because the synchondrosis can resemble a fracture or can itself be fractured. A common clinical scenario involves axial imaging through the immature spine following trauma. In general, synchondroses should be recognized by their characteristic locations and smooth margins with cortication. In contrast, fractures can occur at any location and have irregular, nonsclerotic margins.37 A typical location for synchondroses include the 3 primary ossification centers of the atlas: 1 for the anterior arch and 1 for each of the lamina.41 The lamina fuse posteriorly around 3 years of age and the anterior arch by 7 years of age. Another typical location of synchondroses is the axis. The second cervical vertebra has 4 ossification centers at birth: 1 for the odontoid process, 1 for the body, and 1 at each of the lamina. The odontoid process fuses with the body of the axis normally by 3–6 years of life, although the subdental synchondrosis may be visualized up to 11 years of age and should not be confused with a fracture.37,42 As is the case for other conditions in which instability is suspected, controlled dynamic imaging can be helpful, as can delayed repeat imaging.26

Absent Pedicle. Congenital absence of a pedicle or part of a facet can occur when a lateral chondrification center fails to form or subsequently ossify.19 Congenital absence of the pedicle is rare. The C-5 and C-6 levels are most commonly affected. Because the chondrification center also forms part of the facet and lamina, these structures are usually dysplastic. An absent pedicle can present as an incidental finding or in association with neck pain. Failure to recognize this entity can lead to unnecessary halo fixation or instrumented fusion for presumed traumatic instability.56 Given the associated dysplasia of the articulating pillar, it is recommended that the integrity of the articulating pillar be studied with CT scanning and that the stability of the column be assessed with dynamic radiographs. Unlike os odontoideum, this malformation is often associated with a stable spine and has been described in a football player.19

Butterfly Vertebra. Another rare failure of ossification is a butterfly vertebra.44 This congenital anomaly is typically encountered incidentally but has been associated with pain. It probably represents a ventral ossification failure. Two chondrification centers that form the vertebral body fail to fuse and ossify across the midline. This results in an area of relative bone deficit in the midline. Because it is not associated with a significant deformity, it is typically incidental and can present later in life. Clinically, the scenario is encountered when a patient presents after an injury, and a compression fracture is suspected on plain radiographs. Computed tomography scanning often confirms the diagnosis. The presence of sclerotic bone along the suspected cleft is diagnostic.50

Os Odontoideum. An os odontoideum consists of an

Fig. 4. Lateral lumbar radiographs (left) and sagittal T2-weighted MR imaging (right) of a patient who presented with symptoms of stenosis and a hemivertebra body (arrow). He was unaware of his vertebral abnormality and associated focal kyphosis.
ossicle-like odontoid process unconnected to the body of the axis. It remains uncertain whether this entity is developmental or posttraumatic. The odontoid synchondrosis closes at 5–6 years of age, and os odontoideum may represent a fracture through the synchondrosis. Although it is commonly encountered as an incidental finding, it can be associated with cervical instability and spinal cord injury. A cord signal change at the level of the odontoid suggests the sequelae of repetitive trauma. It is important to note that cord thinning or traumatic myelomalacia can occur in the absence of a history of frank traumatic spinal cord injury. Flexion-extension radiographs may disclose occult instability. The role of surgery is still being defined, and factors such as patient age, cervical instability, and lifestyle weigh into the decision. At present there are only Class III data, and the opinion of experts varies regarding whether or not to perform surgery. At a minimum, when os odontoideum is encountered, the patient should be evaluated for signs of instability and myelopathy. Figure 5 illustrates the case of an incidentally discovered os odontoideum on lateral radiography obtained in the emergency department after a fall. The surgical risks including the biomechanical consequences of a C1–2 fusion and the possibility of pseudarthrosis should be weighed against the possibility of spinal cord injury with a minor trauma. As with cervical stenosis, patients should also be warned about the risk of chronic progressive myelopathy.

Spinal Bifida Occulta. When the neural arch ossification centers fail to fuse, a posterior bone defect occurs, resulting in spina bifida. Spinal bifida occulta is common, with estimates of incidence in a general population of approximately 22%. There is some evidence that links spina bifida occulta with other spinal anomalies and clinical syndromes, such as intraspinal lipoma, tethered cord syndrome, genitourinary dysfunction, increased incidence of disc pathology, lumbar spondylolysis, foot deformities, and syringomyelia. However, some studies have shown that spina bifida occulta is not a reliable indicator of spinal cord structural abnormalities. Spinal bifida occulta can take on subtle forms, such as incomplete formation of the ventral or dorsal arch of C-1 (Fig. 6) and can involve a large section of the sacrum. Spinal bifida occulta is important to recognize, not because it is pathologic per se, but because surgical exposure of the spine requires careful attention to the exposed neural structures (Fig. 7). The authors have treated a patient referred for a cauda equina injury and associated pseudomeningocele related to exposing the L5–S1 junction in a patient with no dorsal sacral bone. When exposing the cervical spine, especially the third, fourth, and fifth vertebrae, the surgeon can encounter a fully bifid spinous process. Failure to recognize this can cause one to enter the spinal canal while dissecting along the medial surface of a bifid process.

Degenerative and Other Changes

Diffuse Vertebral Changes

Some systemic conditions can produce incidental imaging changes in the vertebral body. Because these abnormalities are related to an underlying metabolic process, they affect the spine diffusely. As such, they may be less obvious than focal lesions and are more likely to be missed.

Osteoporosis. One of the most common conditions affecting the spine is osteoporosis, affecting more than 10 million people in the US. It is responsible for more than 388,000 vertebral compression-type fractures annually. The greatest risk for developing osteoporosis occurs in postmenopausal women and is related to maximum peak bone mass and bone mineral density development in young

![Fig. 5. Lateral CT scan of a patient who presented to the emergency department after a fall. An incidental os odontoideum was discovered.](image)

![Fig. 6. Axial CT scan obtained in a trauma patient demonstrating incomplete anterior fusion of the C-1 arch (arrow). This can also occur posteriorly and is typically not associated with mechanical instability.](image)
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Fig. 7. Anteroposterior radiograph (left) and axial T1-weighted MR image (right) of a patient with spina bifida occulta. Surgical approach to the lumbosacral junction, if it were needed, could risk inadvertent entry into the spinal canal.

adult life.9,31 Weight-bearing exercise, nutrition, and cigarette smoking are key factors in this disease process.22 Diagnosis can be assessed by dual-energy x-ray absorptiometry, with the given T-score being a value that can also be used to track response to therapy.27 With MR imaging, the appearance of the normal bone marrow normally varies. For example, with the normal aging process, bone marrow undergoes a gradual replacement from red marrow to yellow marrow, reflecting a loss of hematopoiesis.46 The increased fat content is visible on imaging as an increase on T1-weighted images. These changes can be uniform, but on occasion focal areas of increased yellow marrow can be observed. In severe osteoporosis, there is greater than expected fatty replacement of bone marrow.52

Marrow Reconversion. At the opposite end of the spectrum, bone marrow reconversion refers to the return of hematopoiesis or to bone marrow hyperplasia. This can produce an incidental imaging finding opposite of the expected aging process, with decreased T1 signal intensity. When this is encountered, pathological conditions such as anemia should be suspected. Neoplastic marrow infiltration can also produce similar signal changes, but the pattern of involvement tends to be more diffuse with anemia.

Another diffuse incidental finding is related to hemosiderin deposition, which causes decreased signal intensity in the bone marrow on both T1- and T2-weighted images. Increased marrow hemosiderin can be seen in cases of hemolytic anemia, sickle cell anemia, thalassemia, and in cases of chronic transfusions. Complete blood counts and serum iron studies can be useful in further investigation.

Focal Changes

Vertebral Hemangioma. Hemangiomas are benign lesions of bone (Fig. 8). They are common and may be more prevalent in adults than in children, suggesting that they are not congenital.43 In a recent study of over 1200 lumbar MR imaging scans, incidental hemangiomas were encountered in 1.5% of patients.43 Histologically, they consist of endothelium-lined sinuses interspersed between bone trabeculae. The bony trabeculations produce a characteristic speculated appearance on axial CT scans. They contain fat and have a characteristic T1 MR imaging appearance.58 Some, however, demonstrate a low fat content and have an atypical MR imaging scan appearance. When examined with out of phase imaging, however, they demonstrate a greater degree of signal loss than malignant lesions and this can be used to distinguish them from metastatic processes.58 In rare instances, the hemangioma can be so large that it becomes painful, perhaps related to a compromise of the stability of the vertebral body16 or even fracture (Fig. 9). Symptomatic vertebral hemangiomas can be treated by a variety of modalities and strategies. A combination of transarterial embolization with or without the use of surgical decompression can be used to treat the majority of lesions and is effective at treating cord compression and pain syndromes.1 Vertebroplasty is another technique that can be useful for improving pain symptoms, especially when no neurological deficit is present.2 Other treatments include radiotherapy21 and alcohol embolization.54

Lipoma. Intraosseous lipomas of the vertebral bodies are rare lesions and are typically asymptomatic.13,57 When discovered incidentally, careful interpretation of the MR image can obviate further investigation. Since these le-
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Sions have the potential to be confused with aneurysmal bone cysts, chondroid tumors, simple bone cysts, and fibrous dysplasia, detailed understanding of the MR imaging characteristics is necessary. For lipomas, the T1- and T2-weighted sequences are uniformly hyperintense and there is an absence of cortical destruction and soft tissue abnormality.

Degenerative Endplate Changes. Modic changes affecting the endplates are well-characterized. Occasionally, however, the signal abnormality is so extensive that a more serious lesion is suspected. There are 2 main types and a less common type of endplate lesion. The first type represents endplate edema and is well-characterized on MR imaging; relative to marrow, Type I endplate changes demonstrate T1 signal hypointensity and T2 hyperintensity. Type II changes are more chronic and believed to represent fatty infiltration; relative to marrow, they are characterized by both T1 and T2 signal hyperintensity (Fig. 10). The third type represents endplate sclerosis and is characterized by signal hypointensity to normal marrow on both T1- and T2-weighted images.

There are certain characteristics of endplate changes that merit follow-up, because Modic changes do not always account for all signal changes on MR imaging. Whereas the MR imaging characteristics of fulminant osteomyelitis are easy to recognize, the signal characteristics of very early osteomyelitis are subtle and easy to overlook. An example of this difference is when an MR image is obtained that demonstrates signal abnormality bridging 2 vertebral bodies, thereby raising the suspicion of osteomyelitis. Both degenerative changes and early osteomyelitis affect the endplate region. Classically, an MR imaging signal change that spans 2 vertebra, or is associated with inflammatory changes in the disc, is a sign of potential osteomyelitis/discitis. Dunbar et al. reviewed patients with confirmed vertebral osteomyelitis and identified 4 instances in which an initial early MR image was not diagnostic. These patients had demonstrated Modic Type I changes. Even though all 4 patients were receiving antibiotics, they later developed the full changes noted with infection. In the proper clinical context, repeat MR imaging should be performed a few days later because the infection can progress over days, even in the setting of antibiotic treatment. Additional suspicion for early osteomyelitis over degenerative endplate changes should be raised when the patient has an elevated serum C-reactive protein level, an elevated serum white blood cell count, pain out of proportion to the radiographic findings, or bacteremia.

In patients with a history of cancer, one must consider the possibility of metastasis if the endplate change is extensive. One technique that may help distinguish benign disease from metastasis involves examining the ratio of signal intensity between abnormal and normal bone mar-

![Fig. 9. Sagittal T1- (A) and T2-weighted (B) MR images in a patient with a very large hemangioma that fractured, causing compression of the cauda equina as noted on axial T2-weighted imaging (arrow, C).](image)

![Fig. 10. Endplate changes related to degeneration of the spinal unit are classified as Modic changes. Type II, or fat-type changes, show both T1 (left) and T2 (right) signal hyperintensity (arrow) on MR imaging.](image)
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row for in-phase and out-of-phase images;\(^5\)\(^6\)\(^7\)\(^8\) the following formula is useful: signal intensity ratio = (out-of-phase abnormal vertebra / in-phase abnormal vertebra) / (out-of-phase normal vertebra / in-phase normal vertebra). Benign lesions, including vertebral endplate changes, benign hemangiomas, and bone edema around Schmorl nodes, demonstrate greater loss of signal intensity between out-of-phase imaging and in-phase imaging compared with malignant lesions, where little loss of signal intensity is noted.\(^5\)\(^8\)

**Bone Stress.** In areas of mechanical stress, a signal change can be observed prior to the occurrence of a fracture. This change can occur in the pedicle or in the pars interarticularis. It is uncertain whether these changes are strongly associated with a subsequent fracture or whether they even cause symptoms. Information on the management of these lesions is sparse. It is reasonable to assume that the risk of fracture is increased and that physical activity should be modified to avoid loading the spine, particularly in extension, and to strengthen the supporting core muscles.

**Paget Disease.** Paget disease, a disorder of disorganized bone formation, can be associated with pain. It can also be encountered in asymptomatic individuals. When symptomatic, it can cause considerable morbidity, causing bone pain, fractures, and hypercalcemia. In incidentally discovered lesions, systemic treatment with bisphosphonates is controversial.\(^5\)\(^8\) The prospective randomized PRISM trial suggests that intensive medical management of Paget disease aimed at maintaining normal serum alkaline phosphatase levels conferred no advantage over symptomatic treatment.\(^5\)\(^8\) Referral to a rheumatologist is appropriate. Figure 11 illustrates the case of a 58-year-old woman who presented for evaluation of sciatic pain and was found to have Paget disease of the L-1 vertebra without associated symptoms, despite loss of vertebral height. No additional treatment was undertaken.

**Enostosis.** Enostosis or “bone island” refers to an area of cortical bone within cancellous bone. It is more common in long bones, but can be found in the spine.\(^2\)\(^4\) In a study of over 1200 patients who underwent CT scanning of the cervical spine for evaluation of trauma, only a single case was encountered.\(^6\) Histologically these lesions are benign, and it remains unclear if they represent developmentally aberrant ossification or hamartomatous lesions. Enostosis has the imaging characteristics of cortical bone on MR imaging and can usually be diagnosed on imaging. However, CT scanning can help confirm the diagnosis. On plain radiographs the dense lesion has a peripheral brush-like spiculated appearance.\(^2\)\(^3\) Because these lesions are usually metabolically hypoactive relative to bone marrow, bone scanning can also help confirm the diagnosis.

**Conclusions**

Incidental findings in the vertebral column are common and typically benign. While most have a characteristic imaging appearance, others must be differentiated from potentially concerning pathology and may require complementary or repeat imaging.

**Disclosure**

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

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