The missing pedicle:
a radiodiagnostic challenge

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The absence of a vertebral pedicle, regardless of location, always merits careful consideration. There are a variety of disease states in addition to intrinsic mechanisms which may influence the development of the spine and hence potentiate pedicular absence. Each of these diseases or processes must be considered when confronted by a missing pedicle. This paper will identify and briefly discuss the most common conditions resulting in pedicular absence. (JCCA 1989; 33(4): 187–190)

KEY WORDS: pedicle, metastasis, chiropractic, osteolysis, manipulation.

L'absence de pédicule vertébral, peu importe l'endroit, mérite toujours un considération soignée. Il existe une variété d'états maladifs en plus des mécanismes intrinsèques qui peuvent influencer le développement de la colonne vertébrale et, en conséquence, potentialiser l'absence pédiculaire. Il faut étudier chacune de ces maladies ou processus lorsque l'on fait face à un pédicule manquant. Ce document identifiera et parlera brièvement des conditions les plus communes potentialisant l'absence pédiculaire. (JCCA 1989; 33(4): 187–190)

MOTS CLÉS: pédicule, métastase, chiropratique, ostéolyse, manipulation.

Each condition potentiating pedicular absence invariably displays its own unique radiographic characteristics. Although conventional radiology has obvious limitations, its analysis is often sufficient for the rendering of a diagnostic opinion. Frequently, however, more sophisticated imaging procedures are required for the establishment of a definitive diagnosis. The purpose of this article is to briefly review common etiologies of pedicular absence and the specific radiographic features which characterize each.

Common etiologies

Malignant processes
When an absent pedicle is radiographically identified, the likelihood of metastatic disease must always be considered. Lytic metastatic foci, whose primary source may be situated within the bronchus, breast, kidney, thyroid or gastrointestinal tract, commonly possess a disseminating predilection for the cancellous bone of vertebral bodies and pedicles.[1] This process often results in the total destruction of a vertebral pedicle. When observed in an anteroposterior projection, this feature has been termed the "winking owl sign". Invariably, a diagnosis of

metastatic neoplastic bone disease can be rendered if this sign is present. Although metastasis is the most common precipitant of pedicular loss, further radiographic survey may disclose the presence of additional multiple, ill-defined, osteolytic defects of varying size disseminated throughout other regions of the axial skeleton. In the spine, the lesions may favour the vertebral bodies, one or both pedicles or occasionally the neural arch. A metastatic lesion is frequently characterized by its possesssion of an ill-defined margin which may imperceptibly extend from the pedicle into the posterior portion of the adjacent vertebral body. This appearance may be appreciated in both the lateral and oblique projections. Other areas, most notably the dorsal and lumbar regions, may be affected resulting in a pathological vertebra plana, pedicular loss, Schmorl's node formation or a spinal stenotic appearance.[1] If the contralateral pedicle and adjacent vertebral body are spared, then the remaining pedicle usually appears normal in size, density and contour. Myelography may demonstrate spinal canal blocks. Computed tomography (CT) permits appreciation of the extent of the lesion; while bone scintigraphy will verify the altered metabolic activity of the affected sites.

Another disease process which may exhibit a similar radiographic appearance, is multiple myeloma. Although multiple myeloma may result in pedicular destruction, it remains a significantly lesser cause than metastatic disease. The primary neoplastic plasma cells of myeloma possess a distinct tendency to vertebral body involvement as opposed to favouring pedicular structures, which are usually spared until late in the disease

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process.[1] Like metastatic disease, multiple myeloma generally favours most regions of the axial skeleton and can simultaneously involve multiple areas. Myelomatous lesions are usually small, well-defined, lucent defects distributed diffusely throughout the axial skeleton. Typically, these lesions are equal in size and may possess a sclerotic zone of transition. With spinal involvement, pathological fracture and "punched-out", lytic defects are usually encountered; however, generalized osteopenia may, on occasion, be the only detectable radiographic feature. Bone scintigraphy is unproductive in the presence of multiple myeloma.[1]

Intraspinal processes

Extradural or intradural intraspinal masses, due to their expansile nature, may produce mechanical extrinsic osseous erosion of adjacent pedicular structures. Extradural masses, such as arachnoid cysts, usually arise in the thoracic spine and produce scalloping of the posterior aspects of one or more vertebral bodies, flattening or loss of pedicles and subsequent widening of the interpedicular distance.[2] The cysts are filled with cerebrospinal fluid and posses a pulsatile quality which results in extrinsic resorption of adjacent osseous structures. Myelography and CT scanning normally provide the required information for the diagnosis of this condition. Fortunately, arachnoid cysts are uncommon.

On the other hand, intradural and extramedullary masses are more difficult to assess and sophisticated imaging modalities are required in order to establish the diagnosis. Included in this group of lesions, neurofibromas are the most obvious. Neurofibromatosis may affect any or all areas of the spine resulting in scalloping of the posterior vertebral bodies, expansion of the intervertebral foraminae, thinning of the pedicles and osseous expansion with resultant deformity.[2] Reversal of spinal curves and scoliosis are also frequently associated. A paraspinal mass produced by lateral meningocoeles or neurofibromata may be radiographically observed, but are more evident on myelographic or CT study. Associated erosive alteration and deformities may be seen within costal elements and various long bones. Other masses such as meningioma, lipoma, dermoid and ependymoma may become sufficiently large as to produce extrinsic pressure resorption of pedicles and adjacent vertebral bodies. In these instances, diagnosis becomes difficult without the utilization of CT or magnetic resonance imaging.

Infectious process

Tuberculosis of the spine is uncommon, but this chronic infectious disease state can elicit pedicular destruction.[2] Destruction of juxtarticular vertebral end plates, intervening discs and the anterior portions of vertebral bodies are the hallmark of this spinal disease. Vertebral collapse, loss of disc height, gibbus deformity and paraspinal (thoracic) or psoas (lumbar) abcesses may follow. Pedicular destruction secondary to tuberculous infection is infrequent and occurs only late in the disease process.

Benign processes

Some benign spinal neoplasms including aneurysmal bone cysts and giant cell tumors, may result in marked pedicular expansion to the extent of appearing to obliterate the cortical margins. These tumors are uncommon in the spine but when they do occur, the vertebral body is likely to be the favoured site. Tomography or CT imaging provide the best appreciation of these expansile lesions. Osteoblastomas are primary, benign tumors of bone which are typically lytic and expansile in nature. Thus, this tumor represents an obvious diagnostic consideration, since it also possesses a distinct predilection for the neural arches of the spinal column. This disorder most usually favours the neural arches of the thoraco-lumbar junction and the mid to lowermost portions of the cervical spine.[1] Osteoblastoma generally affects the laminae, traverse processes and pedicles. The vertebral body may ultimately become secondarily involved.[1] Much like other benign bone tumors, osteoblastomas feature a well-defined, expansile corticated mass which may contain a calcific matrix. They may become quite large (4–6 cm in diameter) but are usually not quite as expansile as an aneurysmal bone cyst. Tumors such as hemangioma and reticuloendothelial disorders (i.e. eosinophilic granuloma) tend to involve only the vertebral body and represent rare causes of pedicular destruction.

Congenital anomalies

Pedicular absence may also be attributable to a congenital origin. This is usually manifested by a failure of development of one of the two cartilagenous nuclei which ultimately form the neural arch. In the cervical spine the most commonly affected sites in decreasing order of incidence are C6, C5, C4 and C7.[1] Congenital absence of a pedicle may be associated with dysplasias of the adjacent articular and transverse processes. Enlargement of the intervertebral foramina and spondylolisthesis may also be apparent. Spina bifida occulta may occur at the level of the spondylolisthesis.[1] Hypertrophy and increased density of the contralateral pedicle and posterior arch may occur in the cervical spine, but is more commonly observed in the lumbar region.[1] Thus, enlargement and increased pedicular density with absence of the contralateral pedicle strongly favours a congenital dysplasia. As with metastatic disease, scintigraphy may demonstrate an increased radionucleide uptake in the remaining pedicle. Other congenital anomalies may co-exist.

Failure of development of a lateral ossification centre of the vertebral body and posterior arch may result in a lateral hemivertebra which may once again appear as pedicular loss. The wedge configuration, adjacent end plate deformity, scoliosis and other associated congenital spinal defects usually provide sufficient clues for the rendering of a diagnosis. Lateral hemivertebrae are the most common form of hemivertebra and usually arise in the thoracolumbar region.[1]
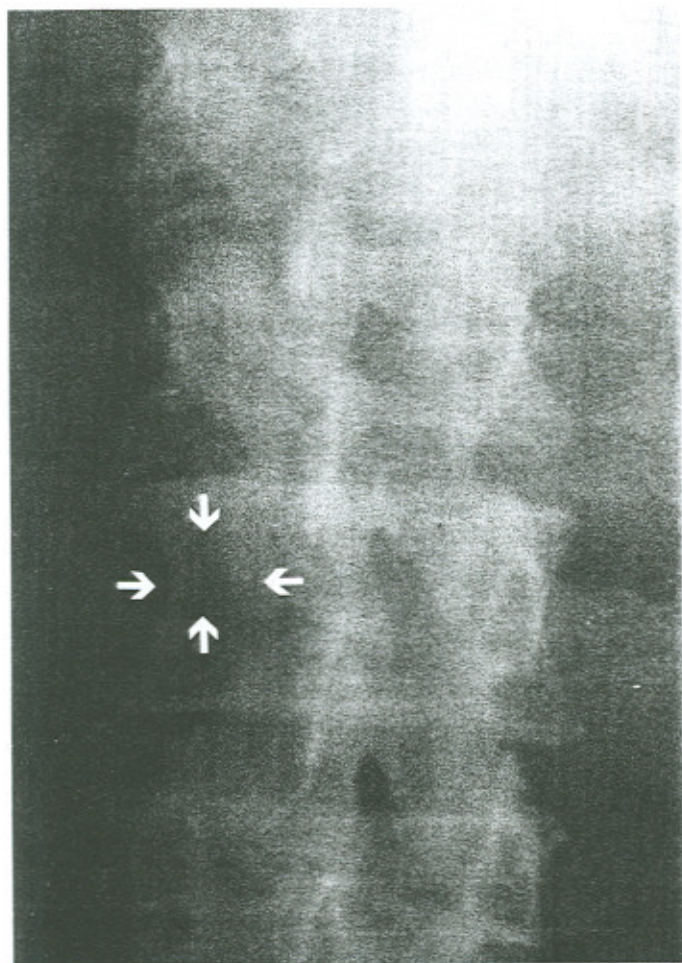
Figure 1. Anteroposterior view of the L3 vertebral segment demonstrating a loss of the left L3 pedicle (arrow).
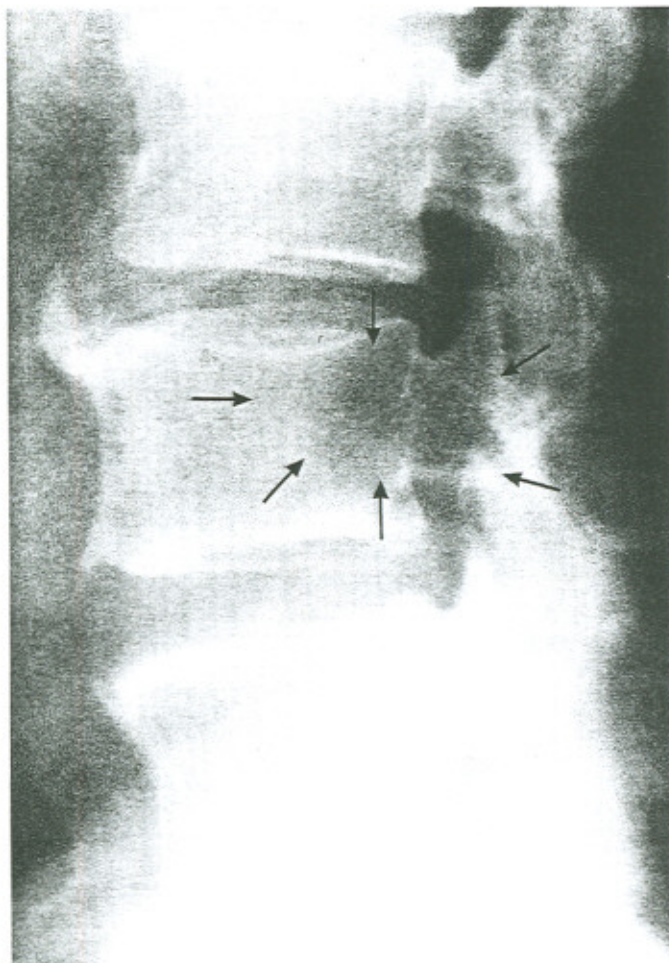

Figure 2. Spot lateral view of L3, revealing radiolucency of the pedicular structure which extends into the posterior one-third of the adjacent vertebral body. Note the poor zone of transition between the lytic region and the accompanying normal bone (arrow).

Miscellaneous conditions

On occasion, a pedicle may be surgically excised for treatment of neoplasm, infection, trauma or relief of dural compression. Surgery, however, remains an uncommon cause of pedicular loss and other associated posterior arch surgical alterations can assist in the identification of this process. Loss of lamina, spinous or transverse processes in conjunction with pedicular excision usually supply the obvious etiology. Comparison with previous radiographic studies may prove helpful in the assessment of this state.

Rare conditions including histiocytosis X, syringomyelia and cervical spinal vertebral artery aneurysm or tortuosity are less likely procedures of pedicular loss.[3]

Case example

The accompanying example is that of a 69-year-old male revealing the loss of the left L3 pedicle (figure 1). The contralateral pedicle retains its expected size, contour and density. There is evidence suggesting that the lesion extends into the posterior portion of the L3 vertebral body (figure 2). Additional radiographs of the left shoulder demonstrate an ill-defined, permeative region of osteolysis within the metaphyseal and metadiaphyseal regions of the humerus. There is also evidence of lateral cortical destruction with extension into the soft tissue (figure 3). The lesion was subsequently curretted, grafted and a stabilizing intramedullary Rush pin inserted to prevent the prospect of imminent pathological fracture (figure 4). The described lesions
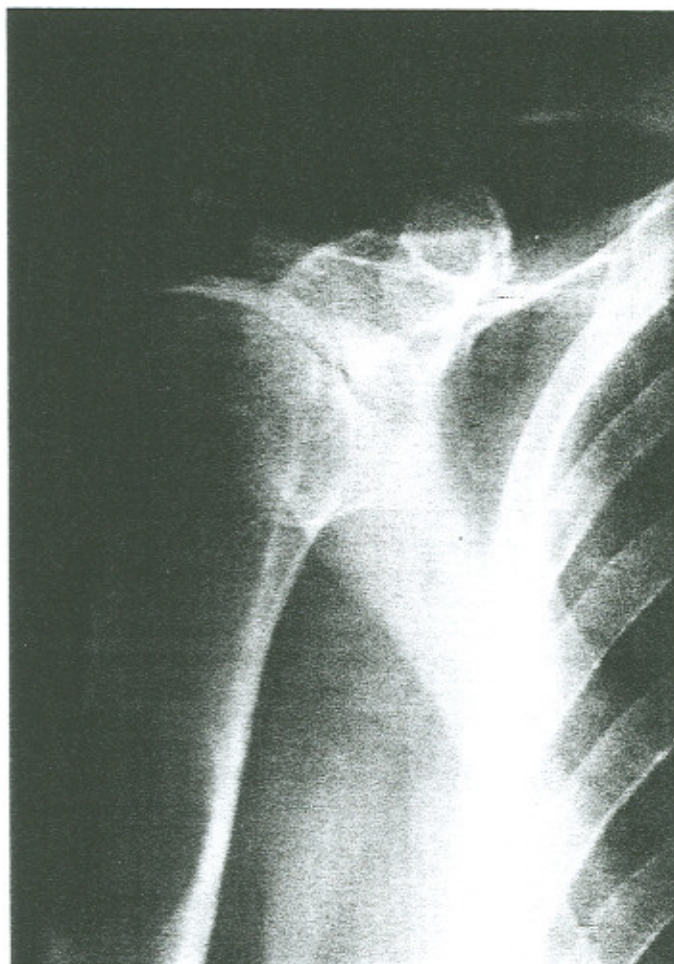
Figure 3. A large, permeative lesion is observed within the meta-diaphyseal region of the humerus. Some suggestion of cortical destruction is apparent.

Figure 4. Osseous grafting and stabilizing intramedullary Rush pin is seen in situ.

are typical of metastatic disease, whose primary site was likely bronchogenic or gastrointestinal. Tumors originating from the kidney or thyroid are usually more expansile in nature.

Conclusion

In conclusion, destruction of a pedicle may arise from any number of reasons ranging from neoplastic involvement, as illustrated, to surgical excision. Although various etiologies can be excluded by incidence and radiographic presentation, it is necessary to consider all the distinct possibilities so that prudent management can ensue.
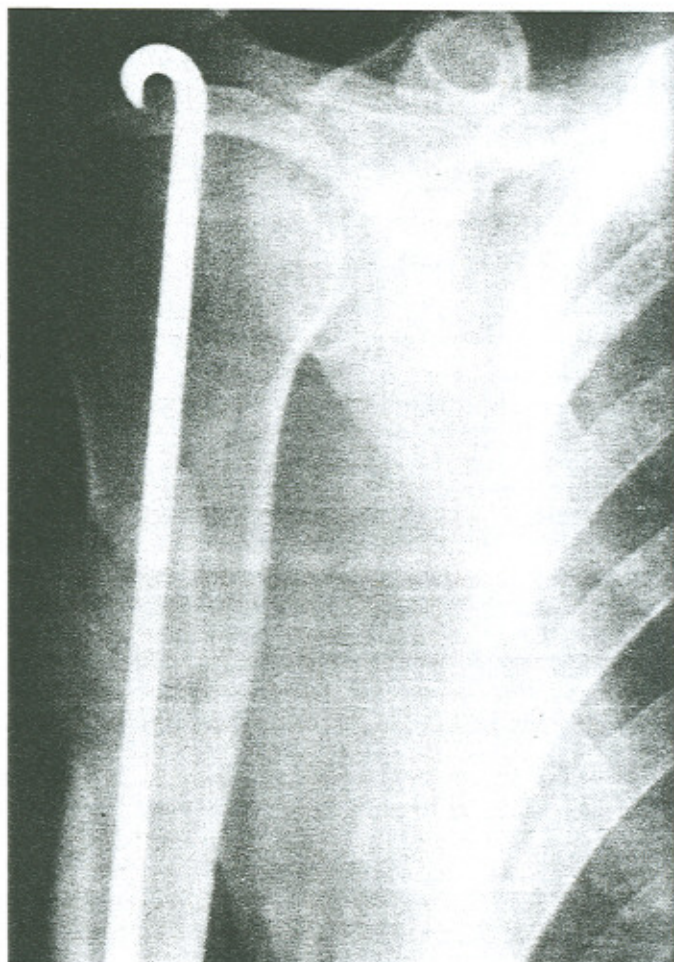
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