Reactive Sclerosis of the Pedicle

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Introduction

The vertebral pedicles of the neural arch represent the “eyes” through which normal variants, anomalies and acquired pathologic conditions can be detected on lumbar spine radiographs. Close scrutiny of the size, shape, density and margins of the pedicles may permit the radiologist to suggest a wide variety of disease.

Radiologic attention is almost always directed at determining of sclerosis or lysis of the pedicle. Numerous conditions causing sclerosis of the pedicle have been reported and among them osteoid-osteoma and osteoblastoma are well known tumors (1,2). However the real significance of reactive sclerosis of the pedicle related to the unstable neural arch such as contralateral spondylolysis have drawn little attention in the literature (3,5).

The purpose of this report is to analyze the nature of arch deficiency which is the primary lesion related to the sclerotic pedicle, and emphasizes the significance of radiologic features of reactive pedicular sclerosis for clinical practice.
Materials and Method

We reviewed retrospectively 800 lumbar spine radiographs including 650 randomly selected patients and 250 spondylolysis or spondylolisthesis. The radiographic criterias in evaluation of lumbar spine radiographs were central sclerosis, enlargement and cortical thickening of the pedicle (as compared with that of the upper and lower lumbar vertebrae or contralateral one) and we defined the sclerotic pedicle which had more than two of above described radiographic criterias. We excluded instances which had severe scoliosis causing multileveled neural arch asymmetry and minor unilateral pedicular hypoplasia.

We have found 18 cases of the sclerotic pedicle in 17 patients and two levels were involved in one patient (case 1). Other combined imaging modalities were CT (computed tomography) scan in seven cases and Tc-99m MDP scan in four cases and we referred these findings to radiographic analysis of the sclerotic pedicle.

In this study, we assessed the following points: (a) coexisting lesions in the ipsilateral neural arch of the same vertebral segment, i.e., sclerosis/hypertrophy in the pars, lamina, superior and inferior facet, defect in the pars interarticularis, and retrosomatic (pediculate) cleft (b) the nature of arch deficiencies which are the primary lesion, i.e., defective pars, facet joint hypoplasia and infralaminar defect.

There were ten males and seven females and the age on diagnosis was ranged from 13 to 53 years. Ten of the 17 patients had had back pain from one to four years. Other seven were asymptomatic and discovered by chance.

Results

There were several radiologic factors usually contributed to the sclerotic pedicle, the most frequent being cortical thickening of the pedicle (17 cases), and central sclerosis was homogeneous.

Coexisting ipsilateral neural arch lesions in the same vertebral segment were hypertrophy/sclerosis of the pars interarticularis in 16 cases, the lamina in seven cases and the superior facet in five cases, retrosomatic (pediculate) cleft in two cases, and spondyloysis in two cases. The sclerotic pedicle was generated by contralateral arch deficiencies in the same vertebral segment: 17 cases of spondyloysis (case 1-3) and one case of hypoplastic facet joint with mild hypoplastic pedicle (case 4).

On CT scan performed in seven cases, retorsomatic (pediculate) cleft, and enlargement, cortical thickening and central sclerosis of the pedicle were more conspicuously demonstrated than plain radiographs and the pedicle was homogeneously sclerotic.

On Tc-99m MDP scan taken in four cases, no abnormally increased or decreased uptake of radionuclide was observed at the sclerotic pedicles and the defective neural arches.

Case Reports

Case 1

A 24-year-old man had experienced back pain for three years. There was tenderness in the region of right side of the fourth lumbar vertebra and others were not remarkable on physical examination. The anteroposterior view of the lumbar spine showed enlargement, cortical thickening and central sclerosis of the right pedicle of L4 with densification of the corresponding pars interarticularis, and mild enlargement and sclerosis at the left pedicle of L2 (Fig. 1-a). The contralateral pars defects at the L2 and L4 were evident on oblique views. CT scan demonstrated more obvious sclerosis/hypertrophy of the pedicle, lamina, superior facet and pars interarticularis of the corresponding lumbar spines, and a thin transverse bony cleft within the sclerotic pedicle of L4, indicating retorsomatic (pediculate) cleft (Fig. 1-b and c). No abnormality was observed on Tc-99m MDP scan.

At that time preoperative diagnosis was osteoid osteoma without consideration of contralateral pars defect and another similar lesion in L2. Resection of the sclerotic pedicle of L4 and a three-level spinal fusion on contralateral spondyloysis was performed. Histologic examination of the resected pedicle revealed homogeneous dense bone and no evidence of
Fig. 1. Case 1. Radiography (a) reveals cortical thickening, enlargement and central sclerosis of the right pedicle of L4 and similar change in the left pedicle of L2, and a defect in the contralateral pars and densification of the ipsilateral pars in the corresponding vertebrae. The CT scan at L2 level (b) and L4 level (c) demonstrate homogeneously increased density of the pedicle, hypertrophy/sclerosis of the lamina and pars interarticularis. A thin cleft is also noted in the sclerotic pedicle of the L4. The radiograph obtained 2 years after operation (d) reveals good fusion state and newly developed cortical thickening of the left pedicle of L4, opposite to the resected pedicle.

osteoid osteoma. Two years later, radiographs demonstrated solid fusion of spondylolysis and newly developed cortical thickening of the left pedicle of L4, opposite to the resected pedicle (Fig. 1-d).

This patient was instrumental in our decision to investigate reactive sclerosis of the pedicle related to opposite neural arch deficiency. On reviewing this case retrospectively with knowledge gained from our study, we recognized that pedicular sclerosis had the characteristic radiologic appearance of reactive sclerosis (pseudotumor) of the pedicle, not sclerosis related to tumor.

Case 2

This 40-year-old man visited our oriental hospital due to low back pain for four years, but information for physical examination was not available. Plain lumbar spine series demonstrated enlargement, sclerosis and cortical thickening with bizarre contour of the right pedicle of L4, and left pars interarticularis defect (Fig. 2-a). Although we thought this was likely to be a sclerotic pedicle related to the defective pars, CT scan was performed to exclude the possibility of tumor. CT scan showed more evident sclerosis/hypertrophy of the right neural arch of L4 and also a thin bony cleft through the whole thickness of the sclerotic pedicle on sagittal reformatted image (Fig. 2-b and c). Operation was not performed.

Case 3

A 19-year-old male patient had experienced a sudden onset of low back pain after a fall since one year ago. There was positive SLR test. The lumbar spine radiographs demonstrated enlargement and cortical thickening of the right pedicle of L3 with moderate condensation of the corresponding pars interarticularis, which enabled us to predict a left pars defect. On oblique view of the lumbar spines, this was confirmed and also an irregular bony excrescence on the superomedial border of the defective pars (hypertrophied pseudoarthritisbuttress) was observed (Fig. 3-a and b). The CT scan and myelogram revealed
bulged disc at L3-4 and L4-5, and also pedicular sclerosis was evident on CT scan.

Laminectomy for bulged disc of L4-5 and L3-4 was performed, but lesion in the L3 was not evaluated owing to surgeon's indifference to that lesion.

Case 4

A 33-year-old male had complained of neck and back pain after trauma since three years ago. There was positive SLR test. There was subtle central sclerosis, enlargement and cortical thickening of the left pedicle of L3 with mild condensation of the corresponding pars. On oblique view of the lumbar spine and CT scan, the expected contralateral pars defect was not observed but the right facet joint at L2-3 appeared to be hypoplastic with mild hypoplastic ipsilateral pedicle, which may be the primary lesion for the sclerotic pedicle (Fig. 4-a and b).

Discussion

Unilateral sclerosis/hypertrophy of the pedicle has been described in association with two different arch deficiencies: a contralateral pars interarticulars
defect, and a contralateral pedicular anomaly associated with some developmental defect of the corresponding facet joint. Maldegue and Malghem (5) reported 86% of sclerotic pedicle related to contralateral spondylolysis and 17% of the latter anomaly in their 50 patients. Wilkinson and Hall (3) reported 12 similar cases. However Hart and Brower (6) reported a case of unilateral pedicle hypertrophy/sclerosis without accompanying contralateral arch anomalies.

In authors cases reported here, the sclerotic pedicle was generated by a contralateral neural arch deficiency, the most frequent being a pars defect (17 out of 18 cases studied). However the incidence of the sclerotic pedicle in patients with spondylolysis is relatively infrequent, which is hard to explain.

The exact mechanism of the sclerotic pedicle related to neural arch deficiency is difficult to assess, but appears to represent a physiologic reaction to stress on an unstable neural arch. The suggested contributing factors and implications for the hypertrophied neural arch are (5): (a) the particular location of deficiency which is at the retropedicular portion of the arch, i.e., pars and/or facet joint, seems to play a more crucial role in the pathogenesis than other factors which have been previously emphasized, such as hypoplasia of the pedicle itself or the patients’ age (7,8). (b) The supralaminar lesions, i.e., pars defect and/or superior facet deficiency, will induce hypertrophy at the same level, while infralaminar deficiencies induce contralateral hypertrophy at the level below. The particular distribution of reactive changes according to the primary lesion accounts for mechanical pathway via the lamina toward the other side. The superior facet, pars and pedicle seem to be more sensitive to the concentrated stresses than the inferior facet and lamina at the overloaded portion of the arch. In author’s cases, modification (densification) of the pars is more remarkable than other portion of the neural arch, which is similar to results of Maldegue and Malghem (5).

Reactive sclerosis of the pedicle related to contralateral arch anomalies is a rare disorder and should not be mistaken for a neoplastic or inflammatory lesions involving the pedicle. These include metastasis, sclerotic myeloma, chondrosarcoma, Ewing’s sarcoma, osteoid-osteoma, osteoblastoma, tuberous sclerosis, Paget’s disease and sarcoidosis (1-3). The history and physical findings may be so non-specific that they are of little help in differentiating unilateral reactive sclerosis from some sinister lesions (4). In evaluation of a sclerotic lesion in the posterior elements of the lumbar spine, careful radiographic evaluation of both the involved side and the contralateral pars interarticularis is indicated. Homogeneous density of sclerotic lesion without appearance of osteolytic lesion supports the diagnosis of secondary stress hypertrophy, while the presence of a nidus confirms the diagnosis of the osteoide-osteoma. The recognition of existence of a contralateral neural arch deficiency may suggest reactive sclerosis of the pedicle. On the other hand, one can predict existence of arch deficiency when
recognize the described sclerotic pedicle from a lumbar spine AP view.

Spine CT may be helpful in demonstrating both nidus and isthmal defect if plain radiographs are not diagnostic. CT scan is valuable in the differential diagnosis and provides definite diagnosis without the need for more expensive and aggressive examinations. In authors’ two cases (case 1 and 2), clefts in the sclerotic pedicle (retrosomatic cleft) were diagnosed by CT scan, which may be interesting finding coexisting with reactive sclerosis. To our knowledge, retrosomatic clefts have been reported in less than 30 cases and are considered by many to be congenital abnormalities associated with other vertebral anomalies. The affected pedicle may be elongated, shortened, thickened, or attenuated and coexisting contralateral spondylosis in the same vertebral segment has also reported (7,10). The retrosomatic clefts are located anterior to the transverse process, where they are easily distinguished from other clefts located laterally in the neural arch, and must be differentiated from traumatic pedicle fractures.

There has been few reports about scintigraphic findings of reactive sclerosis of the pedicle. The normal scintigraphic findings in our four cases indicates that the pedicular sclerosis is an inactive process and a pars defect is not of recent origin. However the length of time between pars defect and the appearance of reactive change is still uncertain (1,6).

The treatment of reactive sclerosis of the pedicle is different from that of a neoplastic condition since excision of a sclerotic pedicle associated with contralateral spondylosis create painful instability. In asymptomatic patients observation is sufficient, while in those with symptoms not relieved by external immobilization, three level spine fusion is indicated. Exceptionally local resection of the bone is required in rare situation with bony outgrowth sufficient to lead to nerve compression (3,5).

In summary, cautious observation of both sclerotic lesion and the contralateral neural arch is essential in radiologic evaluation of the sclerotic pedicle and the presence of a contralateral pars defect in the same vertebral segment suggests reactive sclerosis of the pedicle.

REFERENCES

6. Hart KZ, Brower AC. Unilateral hypertrophy of multiple pedicles. AJR 1977; 129:739-740