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Case Study

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Persistent notochordal canal imitating compression fracture – plain film, CT and MR appearance

Authors' Contribution:
A Study Design
B Data Collection
C Statistical Analysis
D Data Interpretation
E Manuscript Preparation
F Literature Search
G Funds Collection

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Summary

Background: Persistent notochordal canal is a rare developmental anomaly, typically restricted to 1 or 2 vertebral levels, and only in exceptional cases extending over a longer segment. It is usually asymptomatic and discovered incidentally.

Case Report: A 30-year-old woman reported to our department for control lumbar spine plain films. About 2 years before, after minor trauma, she had been misdiagnosed on radiograms in another hospital as having a compression L5 vertebra fracture.

The assessment of current plain films and analysis of previous radiograms delivered indicated the need for taking a developmental anomaly into consideration.

Based on CT and MR of lumbar spine performed, a final diagnosis of a persistent notochordal canal was established.

Conclusions: In spite of its rare occurrence, the characteristic features of a persistent notochordal canal should be known by radiologists, particularly to avoid misinterpretation in post-traumatic patients.

key words: **notochord • spine • abnormalities • CT • MR**

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Background

Persistent notochordal canal is a rare developmental anomaly, typically restricted to 1 or 2 vertebral levels, and only in exceptional cases extending over a longer segment.

Only about 20 cases have been reported in the literature, and we have found only 3 reports presenting findings on MR images [1–3].

The anomaly is usually asymptomatic and discovered incidentally.

In some cases the characteristic appearance of a persistent notochordal canal cannot be well depicted on plain films, and must be verified by CT [4] or MR [1–3].

We present the case of a woman who had initially been misdiagnosed on plain radiograms as having a compression fracture. Final diagnosis of a persistent notochordal canal was established after CT and MR.

Case Report

A 30-year-old woman reported to our department for control lumbar spine plain films. About 2 years before, after minor trauma, she had been diagnosed on radiograms in another hospital as having a compression L5 vertebra fracture.

Current plain films obtained and previous radiograms delivered (Figure 1) revealed a decrease in height at the anterior portion of L5 vertebra with accompanying end-plate irregularity and vertically oriented central cleft. However, the cleft had sclerotic rims on both current and previous exams, and there was no significant difference between both images, as expected for fracture healing. Hence, developmental anomaly was taken into consideration, and spine CT and MR were ordered.

Spiral CT of the lumbar spine (Figure 2) showed a vertically oriented canal traversing the central portion of the L5 vertebra and abutting both end-plates. The canal had sclerotic rims, and, at its lower end, a small area of "vacuum phenomenon" was found, representing degenerative changes in nucleus pulposus.

MR of lumbar spine (Figures 3,4) also presented a vertically oriented canal in the central part of the L5 vertebra, outlined by rims of very low signal, compatible with sclerosis, and communicating with both adjacent intervertebral disks.

In T2-weighted sequences (Figure 3), the central part of the canal was hypointensive similarly to the anuli fibrosi. At the disk spaces, rounded central hyperintensive components were present, corresponding to the nuclei pulposi, with hyperintense signal extensions for a short segment superiorly and inferiorly inside the intravertebral canal.

In T1-weighted sequences (Figure 4), before and after i.v. contrast media administration, the canal had a signal similar to the signal of intervertebral disks, except for small foci of low signal with no enhancement in the topography of nuclei pulposi.

Based on CT and MR, a final diagnosis of a persistent notochordal canal was established.

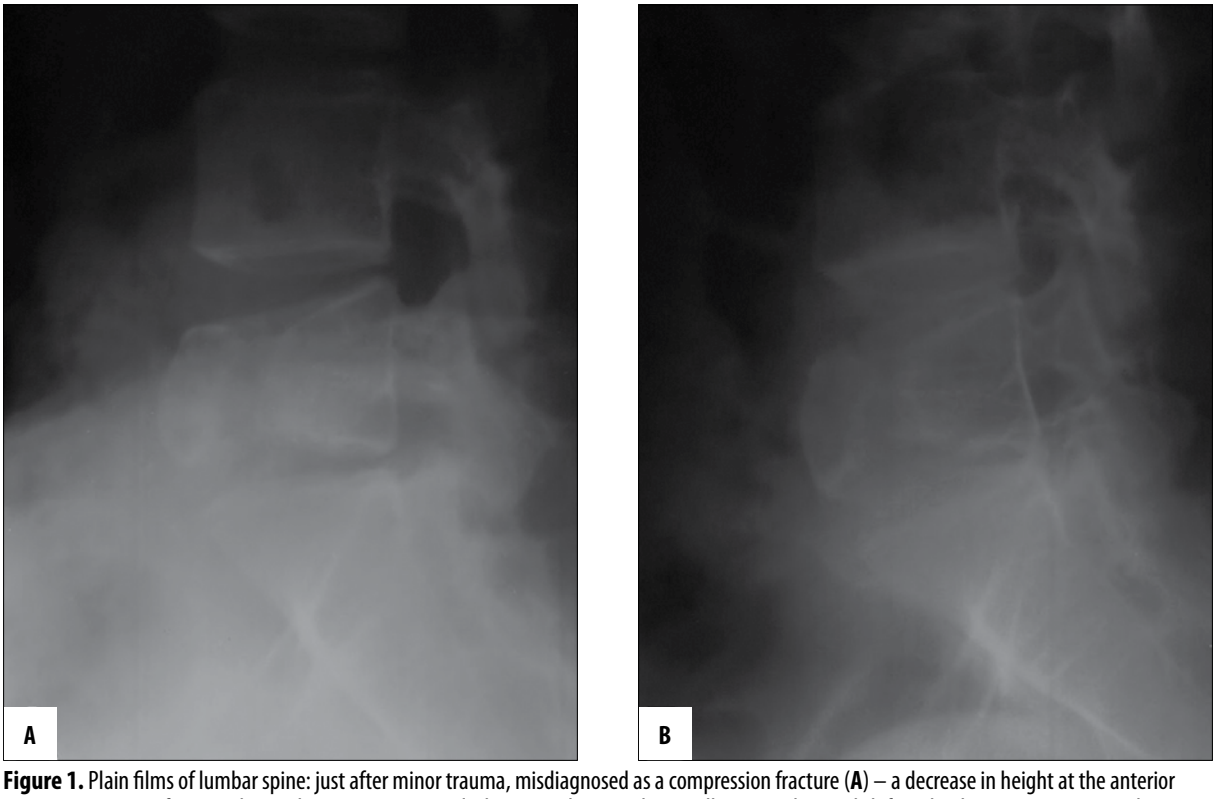

Figure 1. Plain films of lumbar spine: just after minor trauma, misdiagnosed as a compression fracture (**A**) – a decrease in height at the anterior portion of L5 vertebra with accompanying end-plate irregularity and vertically oriented central cleft with sclerotic rims; two year later with no significant difference (**B**).

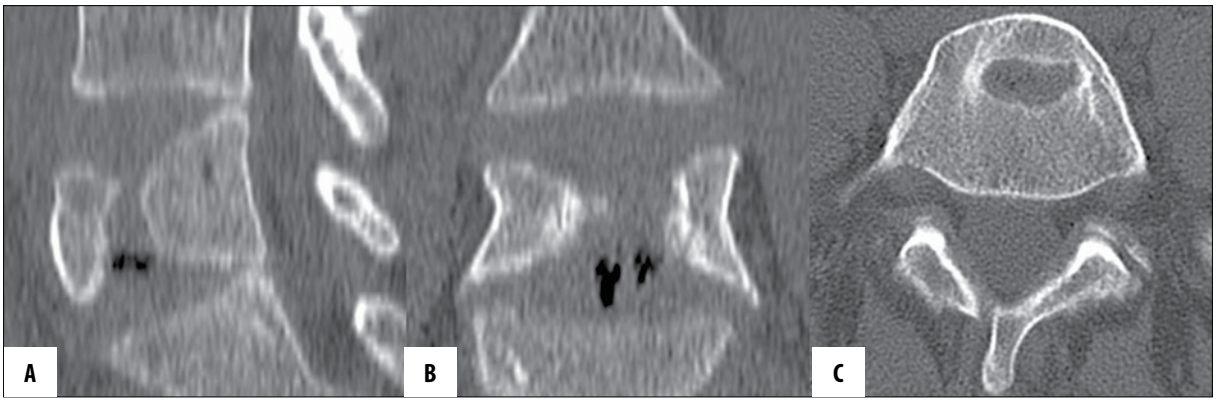

Figure 2. CT of lumbar spine: sagittal (**A**), coronal (**B**) reconstructions and axial slice (**C**) – a vertically oriented canal traversing the central portion of L5 vertebra and abutting both end-plates. The canal has sclerotic rims and at its lower end a small area of "vacuum phenomenon" is found representing degenerative changes in nucleus pulposus.

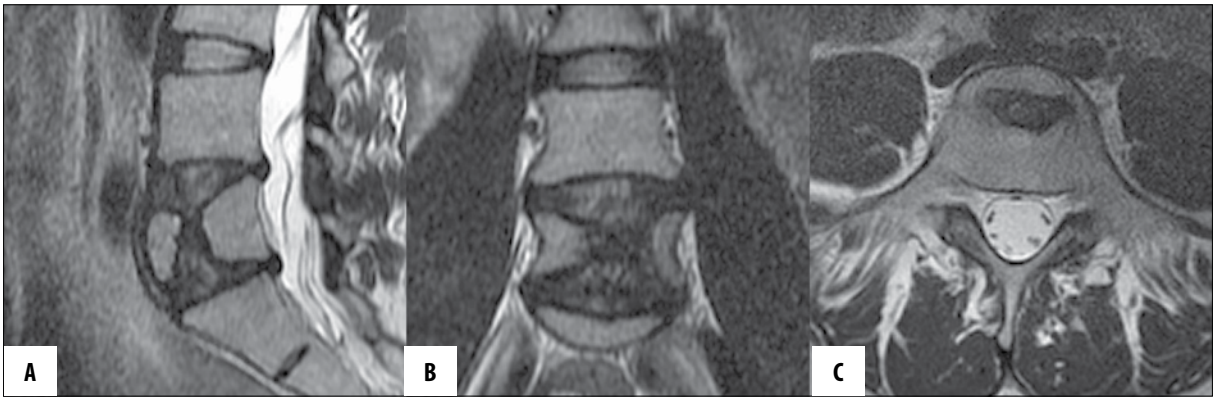

Figure 3. MR of lumbar spine, T2-weighted sequences: sagittal (**A**), coronal (**B**) and axial slice (**C**) – a vertically oriented canal in the central part of L5 vertebra outlined by rims of very low signal compatible with sclerosis and communicating with both adjacent intervertebral disks. The central part of the canal is hypointensive similarly to the anuli fibrosi, at the disk spaces rounded central hyperintensive components are present, corresponding to the nuclei pulposi, with hyperintense signal extensions for a short segment superiorly and inferiorly into the intravertebral canal.

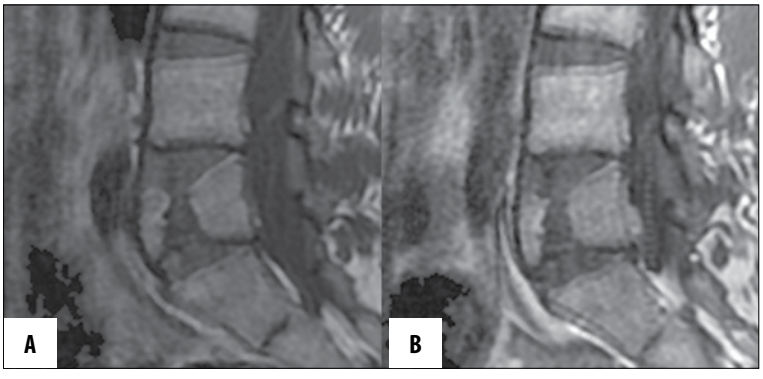

Figure 4. MR of lumbar spine, T1-weighted sequences: before (**A**) and after i.v. contrast media (**B**) – the canal has a signal similar to the signal of intervertebral disks, except for small foci of low signal with no enhancement in the topography of nuclei pulposi.

Discussion

The notochord forms after the third week of development. It is initially a well-defined column of polygonal-shaped cells in an acellular mucoid matrix [2,3]. It induces the formation of a neural plate and serves as a scaffold for axial skeleton development.

During embryonic life the notochord undergoes a series of changes, including proliferation at the disk spaces, forming nuclei pulposi of the intervertebral disks, and regression of the intravertebral segments [2]. Normally no residual notochordal tissue remains in the vertebral body.

When there is no typical regression of the notochord within a vertebral body, the central part of the latter shows a vertical canal, narrower in its centre and wider at its extremities.

A persistent notochordal canal was first described by Musgrove in 1891 as a 'core of unossified tissue, resembling the appearance of an intervertebral disc' [5].

It is typically restricted to 1 or 2 vertebral levels [1,3,4], and only in exceptional cases extending over a longer segment [2].

On plain films, a change in the shape and height of the central part of the vertebral body may be present; hence the differential diagnosis includes butterfly malformation and post-traumatic vertebra collapse [4].

CT, however, shows a well-defined vertical central canal surrounded by a rim of osteosclerosis without abnormality of the surrounding bone, allowing the correct diagnosis to be made [4].

MR also presents the low signal outlining the periphery of the canal, compatible with sclerosis. The canal exhibits identical signal characteristics to the adjacent intervertebral discs on T1- and T2-weighted images [1,3]. Additionally, on T2-weighted images, hyperintense, rounded central components at the disk spaces may be found, corresponding to the nuclei pulposi, with hyperintense signal extending for a short segment superiorly and inferiorly inside the intravertebral portions of the canal [2]. On contrast-enhanced T1-weighted images enhancement of the canal may be visible, except for the rounded central components at the disk spaces corresponding to the nuclei pulposi [2].

A persistent notochordal canal should be differentiated from a pseudo-persistent canal. In Taylor's study of fetuses, infants and children from 25 weeks of gestation to 10 years of age, traces of notochordal canal were present in 7% [6]. This finding is due to a transient delay in ossification in children, and regresses with age. The malformation observed in adults is far less common and represents true persistence of notochordal elements.

Two other, very rare, forms of nonregressing notochordal tissue in adults are a giant notochordal rest and ecchordosis physaliphora.

A giant notochordal rest is a new, benign entity with only 7 cases reported in the literature to date, which has in the past been mistaken for an intravertebral chordoma [7–9]. While vertebral chordomas require vertebrectomy, it is agreed by most authors that giant notochordal rests need only careful clinical and radiological follow-up in order to detect possible transformation into chordoma [9].

Ecchordosis physaliphora is an extraosseous mass typically present in the clivus and dorsum sella [10], but it can be found all along the vertebral column. The frequency of this finding varies between 0.5 and 2% of autopsies [11]. It is usually asymptomatic and few authors have reported symptoms due to tumor expansion and compression of the surrounding structures [12], with only exceptional fatal subarachnoid bleeding due to rupture [13].

Chordoma is a rare tumor arising from intraosseous remnants of the notochord, most commonly at the cranial and caudal junction of the spinal column. It usually progresses slowly, and at the time of diagnosis it presents bone destruction and soft tissue extension. However, in the early phase of the disease, with no evident bony tissue disturbance and soft tissue involvement, differential diagnosis with notochordal remnants may be a challenge [8]. In such cases, monitoring by imaging may assess slow lesion enlargement, confirming tumoral growth.

Conclusions

In spite of its rare occurrence, the characteristic features of a persistent notochordal canal should be known by radiologists, particularly to avoid misinterpretation in post-traumatic patients.

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