A 79-year-old man presented to our clinic with a history of unsteady gait and loss of hand dexterity for many years. He had undergone decompression and fusion without instrumentation at C2–C6 >20 years ago. Three months prior to presentation, his symptoms worsened considerably and he was forced to ambulate with a walking aid. He also complained of neck stiffness and intermittent bilateral hand numbness. On examination, he could not perform tandem gait and had bilateral upper limb weakness (C5–T1) that was more pronounced in the left limb. Hoffmann’s reflex and inverted supinator jerk were positive. Deep tendon reflexes in lower and upper limbs were exaggerated.

A comprehensive radiologic evaluation that included dynamic radiographs, magnetic resonance images (MRI) and computed tomography (CT) scans was performed.

What are the 2 pathologies that cause stenosis at the atlantoaxial region?

A. Ossification of posterior longitudinal ligament
B. Ossification of ligamentum flavum
C. Ossification of transverse ligament of atlas
D. Hypoplastic posterior arch with ligamentous thickening
E. Bony hypertrophy of posterior arch

Findings and Diagnosis

Dynamic radiographs did not show instability (spondylolisthesis) at C1–C2 complex (Fig. 1) and MRI revealed the previous C2–C6 decompression site. Sagittal MRI showed anterior and posterior compression of the spinal cord at the level of C1–C2 with an anteroposterior diameter of 0.42 cm (Fig. 2A). Axial MRI showed a thickened ligament—likely the posterior atlantoaxial ligament—that caused compression from the posterior aspect (Fig. 2B).

CT images suggested C2–C6 fusion that patient underwent earlier (Fig. 3A) and atlantoaxial fusion (Fig. 3B). Axial CT image demonstrated ossification of transverse ligament of atlas (OTLA) and hypoplastic posterior arch with only flecks of bone (Fig. 3C); inner diameter of spinal canal on axial CT image was 1.63 cm.

Based on these radiologic findings, a diagnosis of cervical myelopathy attributed to atlantoaxial spinal stenosis caused by anterior OTLA and posterior ligamentous thickening was made. Patient was offered posterior decompression and instrumentation but he declined surgery. At follow-up 2 years later, his condition and symptoms remain stable and he continues to ambulate with the aid of a quadstick.

Fig 1. Extension (A) and flexion (B) lateral view radiographs showed fusion of C1–C6 with no signs of instability.

Answer: C and D
Discussion

Adjacent segment disorders—such as degeneration and instability—following spinal fusion are common. However, reports of ossification of spinal ligaments adjacent to fusion levels are rare. Although ossification can occur naturally due to increased stress on ligaments, additional mechanical load on lower and upper levels adjacent to the fusion can accelerate this process.

Below the axis (C2) level and throughout the spine, the posterior longitudinal ligament or ligamentum flavum (yellow ligament) can become ossified. However, since these ligaments are not found at the atlantoaxial level,
transverse ligament of the atlas (a strong band that crosses the ring of the atlas and maintains the odontoid process by being in contact with the atlas) or atlantoaxial ligament can become ossified.5-9

In this patient, MRI findings were suggestive of anterior and posterior stenosis of the spinal canal that could be attributed to ossification on both sides. However, CT scans showed only anterior OTLA and a hypoplastic posterior arch with mere flecks of bone, thus ruling out the possibility of posterior bony hypertrophy or ligamentous ossification. Given its anatomical location, the posterior element of the compression was considered a thickened posterior atlantoaxial ligament.

As seen in our patient, posterior ligamentous thickening and OTLA can potentially cause cord compression that can lead to myelopathy.10 Consequently, a definite diagnosis of cervical myelopathy attributed to anterior and posterior atlantoaxial spinal canal stenosis was made.

Since our patient has a history of C2–C6 fusion, it was hypothesised that the upper adjacent C1–C2 level was subjected to increased stress that led to OTLA, posterior ligamentous thickening and atlantoaxial fusion. In our patient, it is debatable whether this phenomenon occurred secondary to adjacent segment degeneration or from natural degeneration secondary to ageing irrespective of the adjacent segment stress. Even so, this presentation is extremely rare and emphasises the possibility of symptomatic OTLA and/or posterior ligamentous thickening occurring in patients with prior fusion involving C2 and below.

REFERENCES