Stabilization with a String-of-Pearls Plate as Treatment for Degenerative Lumbosacral Disease in a Young German Shepherd Dog

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Abstract

Objective The aim of this study was to report the use of a String-of-Pearls (SOP) plate for lumbosacral stabilization as treatment for a 1-year-old German Shepherd suffering from sacral osteochondritis and compressive myelopathy due to lumbosacral subluxation and causing a chronic right hindlimb lameness.
Clinical, imaging, and surgical features are described.

Study Design Case report.

Results A dorsal laminectomy allowed for debridement and lumbosacral stabilization using an SOP plate which led to significant improvement of clinical signs and resolution of pain and discomfort.

Conclusion Successful surgical management was achieved with good long-term results using an SOP plate to stabilize the lumbosacral junction.

Keywords
- osteochondritis
- lumbosacral
- String-of-Pearls
- dorsal laminectomy
- dog

Introduction

Osteochondrosis (OC) is a common developmental condition of the growth of cartilage in skeletally immature animals.[1] It has been characterized as a focal or multifocal skeletal disorder[1] of endochondral ossification in which either parts of the physis (growth plate) or lower layers of the articular surface (articular epiphyseal cartilage complex) fail to mature into bone.[2] When OC progresses to the formation of a cartilaginous flap, the term osteochondritis dissecans (OCD) is used.[1] While there are many reports in the literature which relate to OC of the appendicular skeleton, few concerning OC of the axial skeleton exist.[3–5] German Shepherd dogs (GSDs) are overrepresented when it comes to sacral OC.[3,6] The majority of affected GSDs have a defect in the dorsal part of the sacral endplate and a detached bone fragment in the vertebral canal with mild or no clinical signs.[3] For the GSD, OC is an important differential to consider in patients presented with hindlimb (HL) lameness that cannot be attributed to orthopaedic disease. Conservative management is unsuccessful in the long term[6] making surgical management the gold standard for patients with clinical signs. An L7-S1 laminectomy followed by stabilization of the lumbosacral junction (LSJ) in distraction is described as the treatment of choice to improve hind gait, but also to prevent deterioration over time.[6]

Case Description

A 1-year-old male intact GSD was referred for mild right HL lameness associated with lower back pain after activity. The

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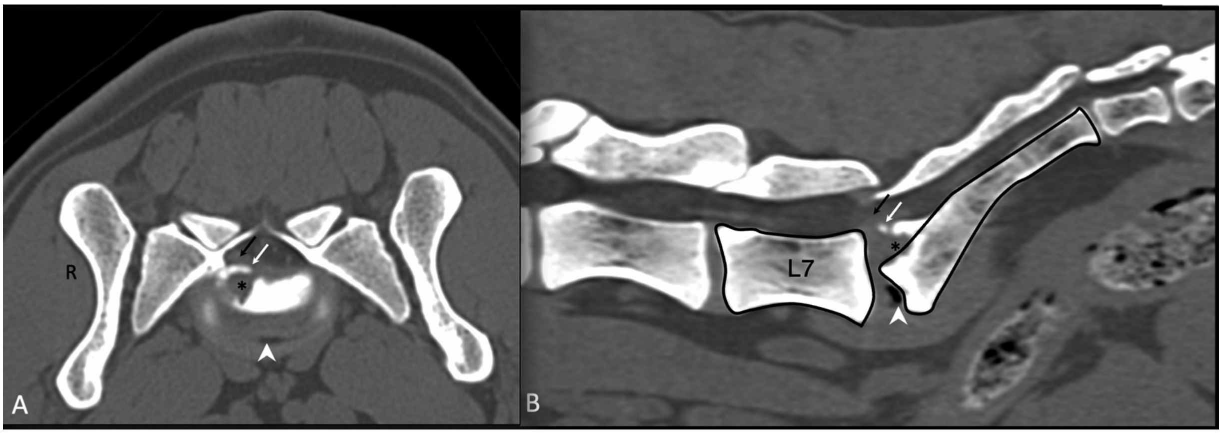

Fig. 1 Computed tomography of the lumbosacral junction with transverse (**A**) and sagittal (**B**) views. A large subchondral defect is noted along the dorsal right aspect of the sacral promontory (black asterisks). The white arrow points to the osteochondral fragment surrounded by sclerosis. The black arrow points to dense material at the interval disc space with compression of the spinal nerve and cauda equina. The white arrow head shows gas in the L7-S1 intervertebral disc space.

owners reported that since the adoption at 2 months of age, the dog had been standing and walking abnormally with the stifle internally rotated and occasional crisscrossing of HLs. Conservative management consisting of nonsteroidal anti-inflammatory drugs, 1 month of physiotherapy, and restricted activity did not provide improvement.

On presentation, general physical examination was within normal limits. The orthopaedic and neurological examination revealed an abnormal HL gait with a cow-hocked and narrow HL stance, mild HL paresis, a sloped hind end, and crouched HL stance. Mild bilateral HL muscle atrophy, worse on the right side, was noted. Nails of both HLs were scuffed, worse on the right paw. The remainder of the orthopaedic examination did not show any additional abnormalities. No neurologic deficits were noted. Spinal reflexes were normal and no back pain was elicited except for a moderate response during the "tail jack" test.

Plain lateral and ventrodorsal radiographs of the LS region did not show any radiographic abnormality. An unenhanced transverse computed tomography (CT) scan of the lumbar spine was performed (Toshiba Aquillion 64 Slice, United States). The images identified an 8-mm diameter concave subchondral defect along the dorsal right sacral promontory (proximal aspect of the endplate). A 6×2 mm, curvilinear osteochondral fragment was present dorsal to the defect surrounded by a thin rim of sclerotic bone. Sclerosis of the cranial sacral endplate was also noted. Distally, there was a second, 3 mm, mildly irregular region of endplate lucency. A mild soft tissue density was described within the spinal canal abutting the right seventh lumbar spinal nerve. At the LSJ, ventral subluxation of the cranial aspect of the sacrum and increased soft tissue to mineral density of the intervertebral disc space at L7-S1 were identified (►**Fig. 1**).

On this dog, lameness and discomfort was very likely secondary to the identified LS subluxation and presence of the OCD lesion. LS subluxation can be interpreted as a sign of dynamic instability of the LSJ explaining the reported clinical signs and examination findings.

Surgical management was elected to distract and stabilize the LSJ. Anesthesia was performed routinely as for a young healthy dog. The dog was placed in sternal recumbency, and the lumbar and sacral regions prepared aseptically. A dorsal approach was made to the dorsal surfaces of L6, L7, and to the entire sacrum.[7] A dorsal laminectomy was performed at L7-S1. The sacral defect was located, and the site was explored and debrided using a curette. The osteochondral fragment could not be visualized. The site was lavaged and the laminectomy site was protected using Gelfoam. The LS space was distracted manually and maintained in position using Gelpi forceps. Screws of diameter 2.7 mm were placed across the right and left LS facets. Two contoured 6-hole 3.5-mm String-of-Pearls (SOP) plates were placed lateral to the midline with five 3.5-mm screws in each plate. The cranial two screws engaged the body of L7 and the caudal three were placed in the body of the sacrum and ilium. The site was lavaged and the Gelfoam was removed. Routine closure was performed. Postoperative CT confirmed correct placement of the implants (►**Fig. 2**).

Postoperative Care and Follow-Up

The following day, the dog was ambulatory with no lameness and was discharged with meloxicam (0.1 mg/kg orally once daily for 30 days, Metacam, Boehringer Ingelheim, Canada), gabapentin (8.5 mg/kg orally thrice daily for 14 days, Gabapentin, Apotex, Canada), and cephalexin (14.3 mg/kg orally every 8 hours for 7 days, Cephalexin, Apotex) with 8 weeks of restricted activity. For 2 weeks following the surgery, the dog was kept confined and was walked with a HL sling. Rehabilitation therapy commenced at 4 weeks postoperatively where a mild intermittent right HL lameness and increased muscle tone in both HLs were noted.

Outcome

Two months after surgery, the dog presented with a mild lameness of the right HL with no ataxia or paresis. The muscle tone was improved as well as strength in both HLs, and no pain was elicited on LS palpation. Recheck radiographs showed correct implants positioning and static LSJ (►**Fig. 3**). Six months after surgery, muscle mass was within normal limits and a mild intermittent lameness of the right HL was present. The owner reported a marked improvement in the dog's quality of life.

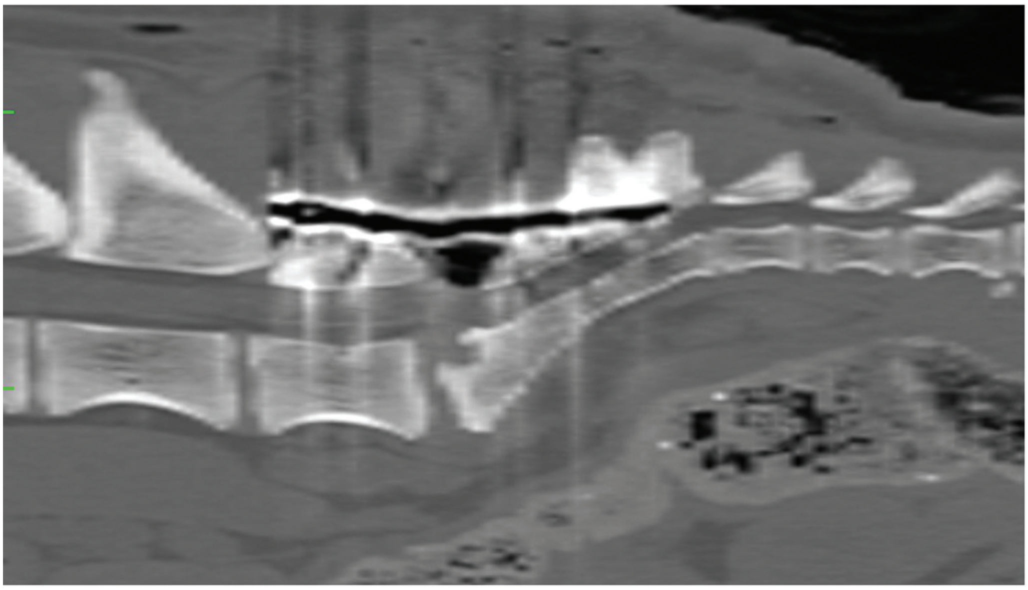

Fig. 2 Immediate postoperative computed tomography of the lumbosacral junction with reduction and stabilization of the lumbosacral subluxation.

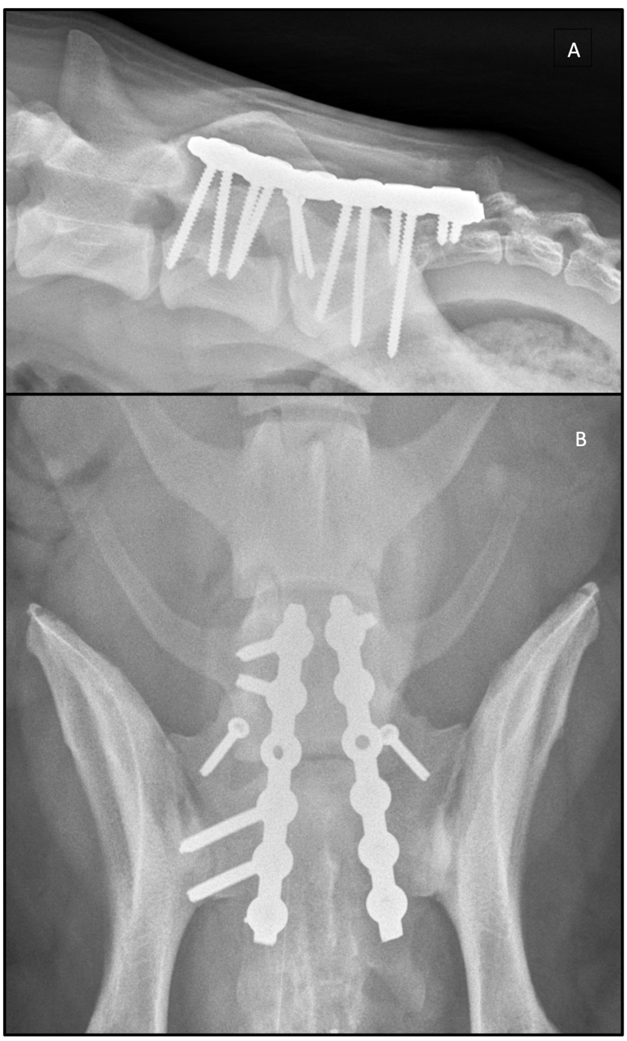

Fig. 3 Two-month recheck radiographs with a lateral (**A**) and ventrodorsal (**B**) view.

Discussion

Sacral OC is a developmental condition with a predisposition in the GSD, males being significantly overrepresented.[3,6,8] GSD represent approximately 60% of the population of dogs suffering from equina syndrome which, in this breed, is mainly due to LS OC.[8]

The described age of onset for clinical and asymptomatic cases ranges from 9 months to 13 years of age.[3,5,6,9] In the case described herein, the dog had clinical signs that may have developed as early as 2 months of age. Although it is unclear if these signs were due to sacral OC. It has been suggested that neurological signs can manifest secondary to OC.[3] Osteochondral flaps were found to be a major contributor to myelocompression.[6] Typically, compared with the patient described herein, the late diagnosis may be explained by the fact that the OC itself was not the cause of the clinical signs, but rather the LS instability associated with a mobile flap compressing the cauda equina are. Degenerative cartilage changes tend to be more advanced at the time of diagnosis and clinical signs develop when the cartilage defect protrudes into the spinal canal.[9] Sacral OC can also be present in asymptomatic dogs. In 250 asymptomatic GSD studied by Lang et al, 6.4% of them were diagnosed with sacral osteochondritis, a percentage that is probably underestimated considering it was established only with radiographs and smaller lesions could have been missed.[3] Moreover, sacral OC has been reported to disrupt the integrity of the LS intervertebral disc and can promote LS foraminal stenosis and cauda equina syndrome.[8] In this context, the OC itself may represent more of a positive risk factor rather than the direct cause of the clinical signs.

Diagnosis of LS OC is made by clinical examination and imaging. The dogs usually present with signs of mild or intermittent HL lameness associated with LS pain and mild HL spinal reflex deficits or signs of cauda equina dysfunction.[3,4,6] Plain radiographs of the LSJ in neutral position may provide a diagnosis.[3] In the present case, the lesion could not be identified on radiographs. Thus, this method of investigations may not be sensitive enough to rule out sacral OC and more advanced imaging may be necessary to identify small OCD lesions. Magnetic resonance imaging (MRI) and CT are often used to further define the size of the lesions and the amount of nerve root compression.[9,10] In this case, MRI was deemed unnecessary, the diagnosis being clear on CT only. The ventral displacement of the sacrum relative to the vertebral body of L7 occasionally present in dogs with degenerative LS stenosis (DLSS) has been proposed as evidence of instability of the LSJ[11–13] which matches our conclusions in this case. However, dynamic imaging could help confirm the instability.[11] Indeed, during dynamic imaging techniques, the patient is imaged with the spine in a flexed and extended position which can show changes that may be underestimated or missed on neutral imaging.[14] A marked narrowing of the intervertebral foramina (IVF) was described in dogs during extension of the LSJ and especially with GSDs with DLSS.[11] The changes in dimensions of the IVF with positioning may be clinically relevant because the narrowing of the IVF with certain movements that extend the spine such as jumping may result in mechanical compression of L7 nerve roots, inducing pain, lameness, and/or neurological dysfunction.[11,13,14]

Surgical treatment for DLSS is the standard choice of treatment in the presence of severe caudal lumbar pain or neurologic deficits that do not respond to conservative therapies like in our case.[6,12,13,15] The techniques described consist of a dorsal laminectomy associated with the removal of the osteochondral fragment if possible, plus distraction-fusion of the LS space[6,9] as decompressive surgery alone could further aggravate instability of the LSJ.[13,15] Fenestration of the LS disc space has also been described.[12] Distraction-fusion consists of expanding the L7-S1 IVF and spinal canal, alleviating pressure on the nerve roots, and permanently stabilizing L7-S1 vertebrae in "normal alignment."[6,12,13,16] The success of this distraction-fusion technique is related to elimination of nerve root irritation by expanding the IVF, stretching the dorsal annulus of L7-S1, and halting degenerative changes.[6,12,16] To make the construct stiffer and stronger and avoid implant failure, multiple pins or screws can be placed within L7 and S1, and then bonded into a dorsal construct with bone cement (polymethyl methacrylate [PMMA]) while maintaining the LSJ at a neutral angle.[12,13,17] Thus, screws and PMMA usually is the treatment of choice in stabilization of LS fractures and following LS decompression because it allows good stability while adapting to the anatomy of the LSJ.

Pedicle screw-rod fixation (PSRF), commonly used for posterior fusion in people, is now being trialed in dogs using either the USS Small Stature system (DePuy Synthes, Amersfoort, The Netherlands)[18] or the SOP (Orthomed, Huddersfield, United Kingdom) locking plate.[17] PSRF can be associated with an intervertebral body cage to increase stability of the LSJ and open the foraminal aperture, thereby relieving the pressure on neural tissues.[19] In the long term, the titanium cage, especially if loaded with bone graft material, promotes ingrowth of bone and fuse the vertebrae together, resulting in a more rigid spinal segment.[19] However, the use of an intervertebral cage has only been described on a very small number of cases in live dogs and never in a case of LS OCD where the degenerative changes of the bone could exacerbate the risk of complication. In the here reported case, the SOP system alone was used. It is a locking plate system that can be contoured in 6 degrees of freedom; medial to lateral bending, cranial to caudal bending, and torsion.[20–22] In this way, it can be contoured to the anatomy of the LSJ as much as the screws and PMMA cement. Its efficacy has been proven in the treatment of iliac fractures in cats.[23] Nel et al compared the stability of the screws and PMMA system to the SOP plate system for LS stabilization and did not show any difference between the two fixation systems in terms of stability.[24] The stability of the joint space against micromotion was not described. A major advantage of the SOP plate is that it does not pose a risk of thermal injury to the laminectomy site from the exothermic reaction during setting of the PMMA.[25] It is also less bulky than the pins with PMMA technique thereby facilitating wound closure.[24,25] Moreover, the porosity of the PMMA cement makes it more prone to harvest bacteria and thus leads to an increased infection rate compared with plate stabilization.[24,26] However, in both techniques, freehand placement can be problematic for placing the implants in the ideal trajectory and carries a potential risk of iatrogenic injury to vital structures including cauda equina, nerve roots, and vasculature.[15,27,28] In this case, the free-hand technique led to adequate bone anchorage with no damage to the surrounding structures (►**Fig. 2**). To avoid these risks, Fujioka et al described a three-dimensional patient-specific drill guide for LSJ fixation that would allow more accuracy and safety in the placement of implants especially in a LSJ with degenerative changes.[15]

The removal of the OCD flap can be essential for the achievement of permanent recovery in some cases while other patients show great results with only stabilization.[6] The need to remove the osteochondral flaps is therefore based on determining the contributory factors of compression.[6] In this case, the compression was thought to be caused by the LS subluxation and the stabilization alone was successful.

In conclusion, DLSS with sacral OC should be part of the differential diagnosis when investigating HL lameness especially in young, skeletally mature GSD. In this case, the use of the CT scan was necessary to confirm the diagnosis because the OCD lesion was not visible on radiographs. The goal of treatment for sacral OC is to alleviate pain, relieve pressure on neural tissues, and permit either complete recovery or stabilization of clinical signs. The application of the SOP plate following surgical decompression permitted achievement of these objectives and no complications were encountered in this case.

Authors' Contributions

A.B. performed the surgery and revised the manuscript.

A.F. performed the neurological assessments and revised the manuscript.

J.G. produced the figures from the CT images and revised the manuscript.

C.B. assisted on the surgery and submitted the manuscript.

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Conflict of Interest
None declared.

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