

## Navigation-assisted resection of tumoral calcinosis of the lumbosacral spine: illustrative case

Oliver Y. Tang, BS,<sup>1</sup> Patricia Zadnik Sullivan, MD,<sup>1</sup> Teddi Tubre, MD,<sup>2</sup> Joshua Feler, MD,<sup>1</sup> Belinda Shao, MD, MPH,<sup>1</sup> Jesse Hart, DO,<sup>2</sup> and Ziya L. Gokaslan, MD<sup>1</sup>

Departments of <sup>1</sup>Neurosurgery and <sup>2</sup>Pathology, Rhode Island Hospital, The Warren Alpert School of Medicine, Brown University, Providence, Rhode Island

**BACKGROUND** Tumoral calcinosis is an uncommon disease resulting from dystrophic calcium phosphate crystal deposition, with only 7% of cases involving the spine, and it may diagnostically mimic neoplasms.

**OBSERVATIONS** In this case, a 54-year-old woman with history of systemic scleroderma presented with 10 months of progressive left lumbosacral pain. Imaging revealed an expansile, 4 × 7-cm, well-circumscribed mass in the lumbosacral spine with L5–S1 neuroforaminal compression. Because intractable pain and computed tomography (CT)-guided needle biopsy did not entirely rule out malignancy, operative management was pursued. The patient underwent L4–S2 laminectomies, left L5–S1 facetectomy, L5 and S1 pediclectomies, and en bloc resection, performed under stereotactic CT-guided intraoperative navigation. Subsequently, instrumented fusion was performed with L4 and L5 pedicle screws and S2 alar-iliac screws. Pathological examination was consistent with tumoral calcinosis, with multiple nodules of amorphous basophilic granular calcified material lined by histiocytes. There was no evidence of recurrence or neurological deficits at 5-month follow-up.

**LESSONS** Because spinal tumoral calcinosis may mimic neoplasms on imaging or gross intraoperative appearance, awareness of this clinical entity is essential for any spine surgeon. A review of all case reports of lumbosacral tumoral calcinosis (n = 14 from 1952 to 2016) was additionally performed. The case featured in this report presents the first known case of navigation-assisted resection of lumbosacral tumoral calcinosis.

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**KEYWORDS** spine; calcinosis; tumoral calcinosis; spine surgery; interoperative navigation; scleroderma

Tumoral calcinosis is an uncommon disease presenting as a tumor-like calcified mass in periarticular soft tissue that forms from dystrophic calcium phosphate crystal deposition. It predominantly occurs in the hips, elbows, and shoulders, with only 7% of cases occurring in the spine.<sup>1,2</sup> Spinal tumoral calcinosis can present with pain or neurological deficits due to compressive radiculomyelopathy or may be discovered simply as a growing or painful paravertebral mass.<sup>3–5</sup> These lesions can mimic neoplasms on imaging or even gross intraoperative appearance, presenting a diagnostic challenge.<sup>6</sup> Although the lesion itself is benign, its resultant symptoms or an uncertainty of diagnosis can lead to operative management. Awareness of this clinical entity is thereby essential for any spine surgeon. Here, we present the case of a 54-year-old woman with lumbosacral tumoral calcinosis causing significant nerve root

compression, managed via navigation-assisted en bloc resection and posterior lumbosacral fusion.

### Illustrative Case

#### History

A 54-year-old woman with a history of systemic scleroderma presented with 10 months of progressive and intractable left lumbosacral pain with intermittent radiation down the left leg. The pain worsened with prolonged sitting or standing and improve with ambulation and exercise. Her motor and sensory examinations were normal. Given failure of conservative management, magnetic resonance imaging and computed tomography (CT) were obtained, demonstrating a large, expansile, 4 × 7-cm, well-circumscribed mass in the lumbosacral spine (Fig. 1). The mass was cauliflower-

**ABBREVIATION** CT = computed tomography.

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**FIG. 1.** Sagittal (A) and axial (B) T2-weighted magnetic resonance imaging demonstrating presenting lesion. Pre- (C) and postresection (D) CT images of a case of lumbosacral tumoral calcinosis. A heterogeneous, cauliflower-like 4 × 7-cm mass is seen with bony destruction of the left L5–S1 neural foramen. The lesion was resected en bloc in two portions and necessitated L4–S2 laminectomies and L5–S1 facetectomies and pediclectomies. L4–S2 alar-iliac instrumented fixation (not pictured) was performed to reconstruct the spine. (Z. L. Gokaslan and P. Z. Sullivan retain copyright.)

like in architecture, appearing heterogenous with both calcified and soft tissue areas. It appeared to originate from the L5–S1 facet joint and S1 pedicle with extension through the left L5–S1 neural foramen, compressing the exiting L5 and traversing S1 nerve roots. It extended epidurally into the spinal canal and dorsally through the paraspinous soft tissue up to the fascia in an overall eggplant-like shape.

A metastatic workup was pursued, with a CT of the chest, abdomen, and pelvis revealing no other masses. A CT from 4 years prior containing the lumbosacral region did not demonstrate this mass. Given the imaging characteristics and rapid growth, at this point a primary bone tumor, such as chondrosarcoma, or soft tissue tumor, such as synovial sarcoma, was suspected. A CT-guided needle biopsy was performed per our institution's spinal oncology protocol. The biopsy demonstrated no evidence of malignancy but revealed granulation tissue, inflammation, and calcified amorphous material, with no ultimate definitive diagnosis.

At this point, the case was presented at a multidisciplinary complex spine forum. Upon rereview of the CT-guided biopsy specimen,

discussion with a sarcoma pathologist concluded that although no definite calcium pyrophosphate crystals were seen on biopsy, the lesion was likely tumoral calcinosis, especially in light of the patient's history of scleroderma. Because of the patient's continued intractable pain and the biopsy not entirely ruling out malignancy because of calcifications potentially representing reactive changes found adjacent to tumor, operative management was pursued. The patient consented to surgery, with the goals of foraminal decompression to treat pain and maximal safe resection of the mass to rule out malignancy.

## Operation

The patient was taken to the operating room, intubated under general anesthesia, and positioned prone with Mayfield pin fixation. Circumferential excision of the mass was performed with the aid of stereotactic CT-guided intraoperative navigation (Fig. 2) and neuro-monitoring, including electromyography, motor evoked potentials, somatosensory evoked potentials, and sphincter signals.

The lesion was widely dissected up to the L3–4 facet, down to the sacroiliac joint and posterior iliac crest, laterally out to the dorsal transverse processes, and medially to the spinous processes. To access the intraspinal elements of the mass and decompress the neural foramen and cauda equina, L4–S2 laminectomies, left L5–S1 facetectomy, and L5–S1 pediclectomies were performed. A large lobular calcified mass was found to be compressing the exiting L5 nerve root at the L5–S1 foramen, consistent with the patient's symptoms and imaging. The lesion was ultimately resected in two separate pieces: one dorsal to the L5 pedicle and the other below the aspect filling the L5–S1 foramen. Both L5 and S1 nerve roots were able to be completely visualized at the completion of resection. The postresection, preinstrumentation intraoperative CT scan is shown in Fig. 1D.

Given the extensive bony resection of biomechanical spinal elements, subsequent stabilization was required and achieved with instrumented fusion, including L4 and L5 pedicle screws and S2 alar-iliac screws. Given the radical soft tissue dissection involved in the case, closure was aided by the plastic surgery team.

Pathological examination of the surgical specimen was consistent with tumoral calcinosis without evidence of malignancy. The mass was composed of multiple nodules of amorphous basophilic granular calcified material lined by histiocytes and multinucleated giant cells (Fig. 3).

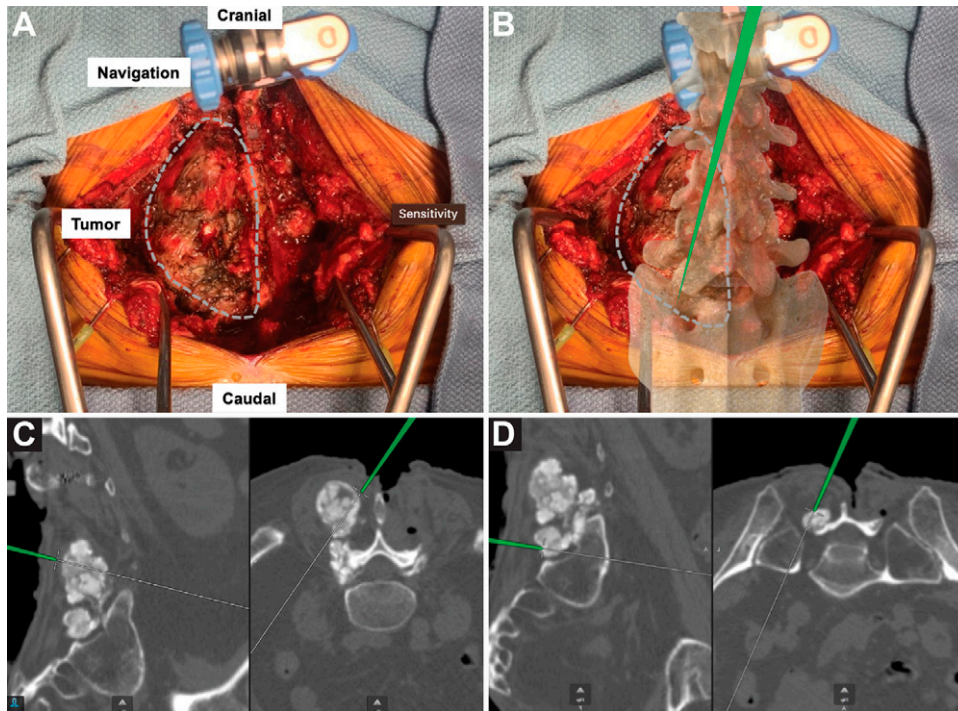
## Postoperative Follow-Up

Postoperatively, the patient recovered well, with complete resolution of radiculopathy but some persistent axial back pain relieved by multimodal analgesics. She was discharged home with no neurological deficits. She presented again 2 weeks after surgery with positional headaches and high-volume, clear-colored output from her surgical drain, but a cerebrospinal fluid leak was ruled out by imaging, and symptoms resolved promptly following observation and drain removal. There was no evidence of recurrence or neurological deficits at 5-month follow-up (Fig. 4).

## Discussion

### Observations

While "tumoral calcinosis" was first termed by Incan et al. in 1943,<sup>7</sup> descriptions of this clinical entity date back to 1899.<sup>8</sup> Only an estimated 7% of cases occur in the spine.<sup>1,2</sup> Most spinal tumoral



**FIG. 2.** Intraoperative photographs of tumor (A, gray outline) with lumbosacral spine anatomical overlay (B) with a green pointer corresponding to the intraoperative neuronavigation seen in panel D. Screenshots (C and D) demonstrate the use of intraoperative neuronavigation. (Z. L. Gokaslan and P. Z. Sullivan retain copyright.)

calcinosis involves the lumbar spine,<sup>9–15</sup> but lesions in the cervical,<sup>1,3,4,14,16,17</sup> thoracic,<sup>2,15,18</sup> and sacral regions have also been described.<sup>12</sup> In contrast to the commonly painless and asymptomatic presentation of its extraspinal counterparts, spinal tumoral calcinosis often presents with pain or neurological deficits.<sup>3–5</sup>

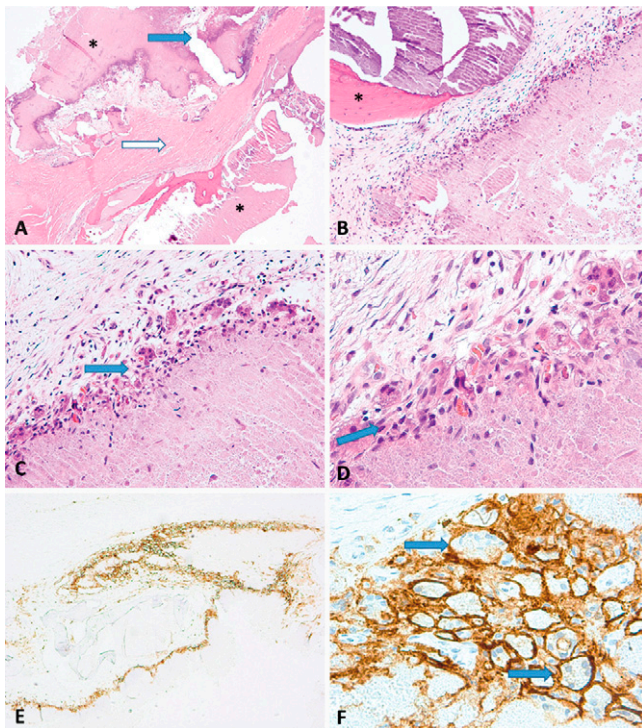
Tumoral calcinosis can arise in patients with underlying genetic or metabolic disorders that result in hyperphosphatemia,<sup>19</sup> uremia,<sup>3,4,20</sup> scleroderma,<sup>5,13,18,21</sup> rheumatoid arthritis,<sup>22</sup> and seronegative spondyloarthropathy.<sup>12</sup> Idiopathic cases have been reported<sup>9–11,16,23,24</sup> as well as those arising secondary to surgery or trauma at the site of involvement.<sup>14,25</sup> In the spine, posited mechanisms of calcium phosphate crystal deposition in the spine include origination from the articular capsule, disc or facet joint degeneration, and hemorrhage with foamy histiocytic aggregation.<sup>2,11–13,19</sup> In the present case, the lesion appeared to be centered in the bony spine, within the L5–S2 neural foramen, possibly arising from the L5–S1 facet joint. The case discussed was also associated with scleroderma. Soft tissue calcinosis is estimated in 9% to 27% of patients with sclerodermal disorders.<sup>18</sup> Tumoral calcinosis in scleroderma may occur via osteolysis or chronic inflammation in the setting of tissue hypoxia.<sup>5</sup>

As seen in Fig. 3, the characteristic pathology of tumoral calcinosis entails amorphous calcium hydroxyapatite crystals surrounded by a reactive process in response to this material, involving foreign body giant cells and a histiocytic reaction, occasionally with intermixed detritic bone.<sup>6</sup> Lesions are often cystic or multiloculated, which may manifest as a “sedimentation sign” on imaging due to fluid-fluid levels from calcium layering.<sup>14,20,23</sup> Earlier lesions may contain a large amount of eosinophilic material, but as they

continue to calcify over time, the crystals can condense and the overall appearance may become more basophilic, with some cases containing psammomatous elements.

As in the present case, tumoral calcinosis can present a diagnostic challenge due to mimicking neoplasms on preoperative imaging or even gross intraoperative appearance.<sup>6</sup> Moreover, given that similar appearing calcification can also be found adjacent to bony or soft tissue tumors, a CT-guided biopsy without evidence of malignancy may not sufficiently rule out the possibility of unsampled malignancy adjacent to these findings. The suspected tumoral calcinosis found in the patient’s initial biopsy could have represented reactive changes adjacent to an unsampled malignant tumor. Given the patient’s imaging findings and rapid lesional growth raising concern for malignancy, a more radical resection was undertaken.

Nonoperative modalities of management have included observation, due to reported cases of spontaneous resolution,<sup>23</sup> or addressing an underlying medical condition promoting soft tissue calcification.<sup>3,4</sup> Nevertheless, surgery is frequently indicated for spinal tumoral calcinosis due to progressive neurological symptoms.<sup>14</sup> Such procedures aim for resection, decompression of neurovascular structures with laminectomy or facetectomy, and promotion of stability by fusion or arthrodesis.<sup>1,6</sup> Case series imply that incomplete resection may be associated with recurrence risk, with recurrence rates for tumoral calcinosis up to 75% following subtotal resection.<sup>2,12,26–28</sup> While the risk of recurrence is less understood for spinal tumoral calcinosis specifically, in one review of 41 cases, Kalani et al. estimated successful gross total resection in 92.7% of patients and no reported episodes of recurrence.<sup>15</sup>

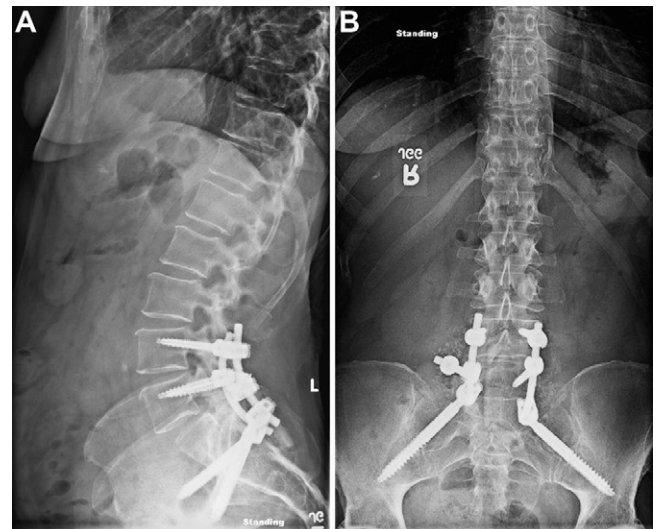


**FIG 3.** A case of paraspinal tumoral calcinosis in a patient with scleroderma. **A:** At low power, the lesion is characterized by multiple nodules of amorphous granular calcified material separated by fibrous septae (hematoxylin and eosin [H&E], original magnification  $\times 4$ ). **B:** The lesion erodes bony trabeculae (H&E, original magnification  $\times 10$ ). **C:** Amorphous basophilic material is separated by fibrous tissue (H&E, original magnification  $\times 20$ ). **D:** On higher power, the histiocytes and multinucleated giant cells line the calcified material (H&E, original magnification  $\times 40$ ). **E:** An immunohistochemical stain for CD45 highlights histiocytes and giant cells lining the calcified material (original magnification  $\times 4$ ). **F:** At higher magnification, membranous CD45 staining highlights individual multinucleated giant cell membranes (original magnification  $\times 40$ ). (Z. L. Gokaslan and P. Z. Sullivan retain copyright.)

Following a review of the literature, we report on the characteristics of 14 case reports of lumbosacral tumoral calcinosis from 1952 to 2016 (Table 1),<sup>9–14,29–36</sup> 42.9% (6/14) cases were idiopathic and the most commonly known cause was scleroderma disease (35.7%). Eleven cases (78.6%) received surgical management, with fusion reported in only 2 cases (14.3%).<sup>13,14</sup> No cases of recurrence were documented. The present case is only the third reported case to describe the use of fusion to promote biomechanical stability after lumbosacral tumoral calcinosis resection. Moreover, CT-guided navigation of tumoral calcinosis has not previously been discussed in the literature. The present case is a salient example of the use of intraoperative navigation as an aid in achieving gross total resection (Fig. 2).

### Lessons

Spinal tumoral calcinosis is a rare disease that can masquerade as a malignancy and may require surgical treatment because of neurological symptoms and the need for a definitive diagnosis. Although cases may be idiopathic, potential etiologies include hyperphosphatemia, uremia, scleroderma, rheumatoid arthritis, and seronegative spondyloarthritis. An illustrative case of lumbosacral tumoral calcinosis



**FIG 4.** Sagittal (A) and coronal (B) radiographs at the 5-month follow-up demonstrating lack of recurrence of lumbosacral tumor calcinosis. (Z. L. Gokaslan and P. Z. Sullivan retain copyright.)

associated with scleroderma and causing radiculopathy is presented, in which stereotactic-guided surgery led to safe gross total resection and symptomatic resolution. This report presents the first known case of navigation-assisted resection of lumbosacral tumoral calcinosis.

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**TABLE 1. Summary of case reports of lumbosacral spinal tumoral calcinosis**

Authors & Year	Age, Sex	Location	Etiology	Presenting Symptoms	Resection of Mass	Treatment	Recurrence
Blay et al., 2001 <sup>29</sup>	44, F	L5	Inherited metabolic disorder	Low back pain	No	Conservative treatment w/ analgesics	NA
Cho et al., 2007 <sup>30</sup>	37, F	L3–4	Idiopathic	Tender paravertebral mass	Yes	En bloc resection	None (at 12 mos)
Durant & Farge-Bancel, 2011 <sup>31</sup>	62, M	L4–S1	Sclerodermal disease	Incidentally found	No	Not reported	NA
Ebot & Nottmeier, 2019 <sup>14</sup>	51, F	L4–5	Previous op at site	Tender sacroiliac joint, pain w/ weight bearing	Yes	L4–5 HL & L4–5 transforaminal lumbar interbody fusion	None (at 3 mos)
Emon et al., 2011 <sup>12</sup>	70, F	L5–S1	Seronegative spondyloarthropathy	S1 hypoesthesia, hypoactive Achilles reflex, positive straight-leg raise	Yes	L5–S1 HL	NR
Iglesias et al., 2002 <sup>32</sup>	55, M	L5–S1	Idiopathic	Numbness & weakness of lt leg, hypoactive Achilles reflex	Yes	L5–S1 laminectomy	NR
Liberato et al., 2016 <sup>33</sup>	47, F	L5–S1	Sclerodermal disease	Low back pain, L5 radiculopathy	No	Conservative treatment w/ analgesics & steroids	NA
Riemenschneider & Ecker, 1952 <sup>34</sup>	59, F	L5	Idiopathic	Low back pain, hypoalgesia of posterior thighs	Yes	L5 HL	None (at 5 mos)
Sharma et al., 2005 <sup>9</sup>	55, M	L3	Idiopathic	Low back & rt leg pain, areflexia in bilat LE	Yes	L3 laminectomy	NR
Shibuya et al., 2006 <sup>13</sup>	49, F	L3–4	Sclerodermal disease	Low back pain, bilat LE weakness, gait disturbance, L3–4 spondylolisthesis	Yes	En bloc resection & posterolateral fusion (levels not reported)	None (at 22 mos)
Vaicys et al., 1999 <sup>10</sup>	49, M	L3	Idiopathic	Growing paravertebral mass	Yes	Not reported	NR
Ward et al., 1997 <sup>35</sup>	53, M	L3–S1	Sclerodermal disease	Lt leg pain & weakness	Yes	L4–5 laminotomy & discectomy, L5 HL, & S1 laminotomy	NR
Watanabe et al., 2000 <sup>11</sup>	55, M	L4–5	Idiopathic	Gait disturbance	Yes	L5 laminectomy & duraplasty	None (at 3 yrs)
Weerakoon et al., 2011 <sup>36</sup>	60, F	L4–5	Sclerodermal disease	Low back & lt leg pain	Yes	En bloc resection	None (at 12 mos)

HL = hemilaminectomy; lami = laminectomy; LE = lower extremity; NA = not applicable; NR = not reported.

Summary of 14 earlier case reports of lumbosacral spinal tumoral calcinosis published from 1952 to 2016. There were no operative case series found on literature review. The search strategy encompassed a search of (“spine” OR “spinal” OR “vertebral”) AND (“tumoral calcinosis” OR “tumor calcinosis”) on PubMed on December 31, 2021. All articles discussing lumbosacral tumoral calcinosis were included for summary. The references list for all included articles was also manually reviewed for additional articles that warranted inclusion.

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### Disclosures

The authors report no relevant disclosures or conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

### Author Contributions

Conception and design: Gokaslan, Zadnik Sullivan, Tubre, Shao. Acquisition of data: Gokaslan, Zadnik Sullivan, Feler. Analysis and interpretation of data: Gokaslan, Tang, Tubre, Shao, Hart. Drafting the article: Tang, Feler, Shao, Hart. Critically revising the article: Gokaslan, Tang, Zadnik Sullivan, Feler, Shao. Reviewed submitted version of manuscript: Gokaslan, Tang, Zadnik Sullivan, Feler, Shao, Hart. Approved the final version of the manuscript on behalf of all authors: Gokaslan. Administrative/technical/material support: Feler.

### Correspondence

Ziya L. Gokaslan: Rhode Island Hospital, Providence, RI. [ziya.gokaslan@lifespan.org](mailto:ziya.gokaslan@lifespan.org).