

## CASE REPORT



# Spinal artery syndrome following kyphoplasty in the setting of a non-compressive extradural cement extravasation: a case report

Brendan M. Striano<sup>1</sup>, Brian C. Goh<sup>2</sup>, Chason Ziino<sup>3</sup> and Saechin Kim<sup>4</sup>✉

© The Author(s), under exclusive licence to International Spinal Cord Society 2023

**INTRODUCTION:** Cement extravasation (CE) during vertebroplasty or kyphoplasty for vertebral compression fracture (VCF) is not uncommon, though neurological deficits occur rarely and when paraparesis occurs severe cord compression has been described. We report a case of progressive paraparesis in the setting of non-compressive extradural CE during kyphoplasty with evidence for spinal artery syndrome and neurological recovery after treatment.

**CASE PRESENTATION:** A 77-year-old female with T12 VCF failed conservative treatment and underwent kyphoplasty. In the recovery room, the patient was noted to have bilateral leg weakness, left worse than right, and had urgent CT scan that showed right paracentral CE without cord compression or arterial cement embolization. The patient was transferred to a tertiary hospital and had MRI of the spine that confirmed extradural CE and no cord compression. Because the patient had progression of lower extremity deficits despite medical management, she underwent surgical decompression, cement excision, and spinal fusion with instrumentation. Post op MRI showed T2 hyperintensities in the spinal cord consistent with spinal artery syndrome. One month post op, she had almost complete recovery of her neurological function.

**DISCUSSION:** Spinal artery syndrome may be considered in patients with neurological deficit s/p kyphoplasty even if the extravasated cement does not compress the spinal cord and even if the deficits are worse contralateral to the cement extravasation. If spinal artery syndrome is present and medical management does not improve the deficits, surgery may be indicated even if there is no cord compression.

*Spinal Cord Series and Cases* (2023)9:18; <https://doi.org/10.1038/s41394-023-00574-6>

## INTRODUCTION

Vertebral compression fractures (VCF) can result from minimal to no trauma in osteoporotic bone or oncologic lesions. Though cement augmentation procedures for VCF, vertebroplasty and kyphoplasty, are thought to be safe [1–4] and to improve outcome [5], complications have been described including local extravasation into paraspinal soft tissues, perivertebral veins, disc space, and spinal canal [6]. A review of neurological deficits due to cement extravasation showed that though cement extravasation into the spinal canal is common, clinical sequelae are rare, identifying 21 cases of neurologic compromise [7] because of direct compression, thermal injury, or nerve reaction to cement especially when the cement is intradural [8, 9]. Other reported causes of paraparesis following cement augmentation procedures include arterial cement embolization causing spinal artery syndrome [10, 11], epidural hematoma [12], and spinal artery syndrome [13], latter of which described a transient paraplegia following kyphoplasty without cement extravasation and without report of MR confirmation of spinal artery syndrome. The purpose of this report is to present a case of progressive paraparesis following kyphoplasty without arterial cement embolization or

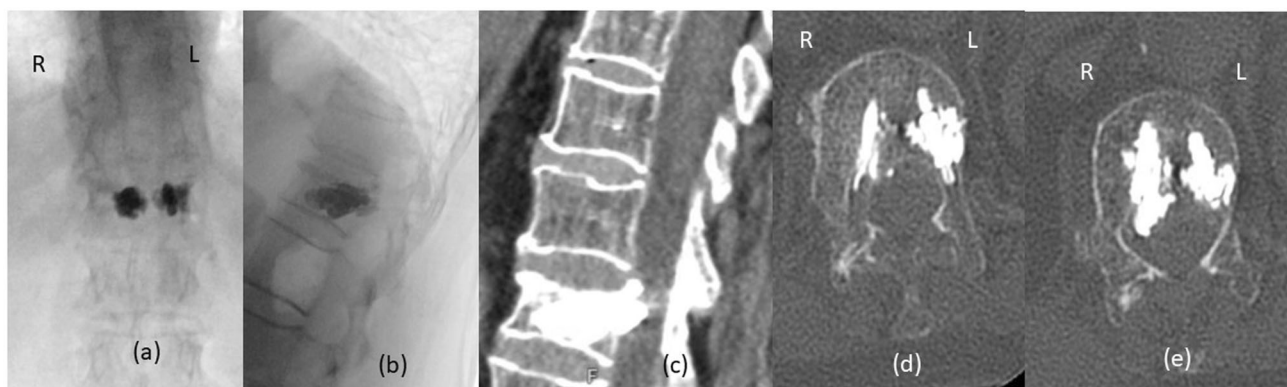
epidural hematoma but with non-compressive extravasated cement that improved after surgical treatment and with post op MRI confirmation of spinal artery syndrome.

## CASE PRESENTATION

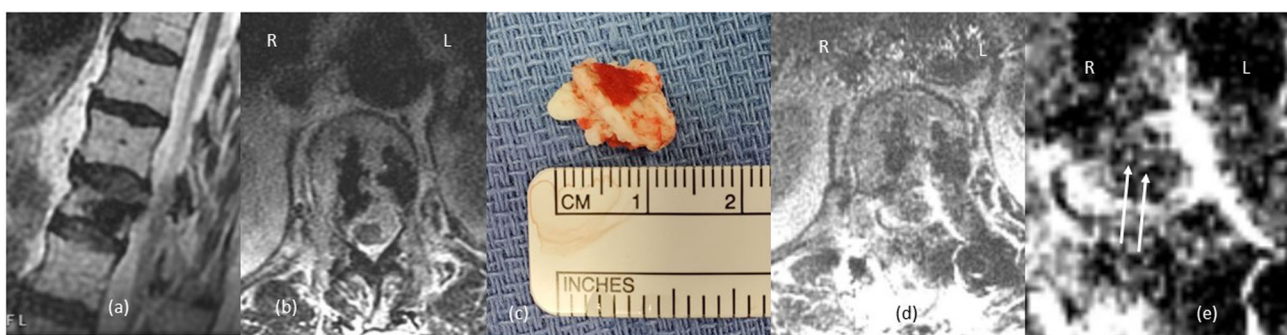
A 77F with multiple myeloma and remote history of deep venous thrombosis on apixaban presented with T12 compression fracture and persistent pain despite conservative treatment. She underwent T12 kyphoplasty with two SpineJack implants (Stryker, Kalamazoo, MI, USA) [14, 15] inserted into the T12 vertebral body under fluoroscopy using transpedicular approaches. During polymethylmethacrylate (PMMA) injection, posterior cement extravasation was noted, and the procedure was stopped (Fig. 1a, b). There were no pulmonary or hypotensive episodes during the procedure. In the recovery room, the patient was found to have left worse than right lower extremity weakness and altered sensation in bilateral lower extremities. She was started on steroids and then underwent urgent computed tomography (CT) of the thoracic spine that showed that on the right side, the approach was likely medial to the right pedicle (Fig. 1d) with

<sup>1</sup>Harvard Combined Orthopaedic Residency Program, Harvard Medical School, Boston, MA, USA. <sup>2</sup>Department of Orthopedic Surgery, Mayo Clinic, Rochester, MN, USA. <sup>3</sup>Department of Orthopedics, University of Vermont, South Burlington, VT, USA. <sup>4</sup>Department of Orthopaedics, Massachusetts General Hospital, Harvard Medical School, Boston, MA, USA. ✉email: [skim1@partners.org](mailto:skim1@partners.org)

Received: 13 November 2022 Revised: 12 April 2023 Accepted: 14 April 2023  
Published online: 25 April 2023



**Fig. 1 Cement leakage in the spinal canal.** Kyphoplasty (a) was done with SpineJacks (Stryker, Kalamazoo, MI, USA) using the transpedicular approach and cement extravasation into the canal was noted (b) during cement injection and the injection was stopped. CT scan showed right-sided intracanal cement extravasation (c, d, e) and on the right side, the approach appears to be medial to the pedicle (d).



**Fig. 2 Pre- and post-op imaging.** MRI of T spine was done that confirmed that the extravasated cement was just next to the spinal cord (a, b). Because the patient had progression of right leg weakness, she underwent decompressive surgery and intracanal cement was excised (c) that corresponded to the intracanal cement seen in the axial images (b). Post op MRI showed excision of the intracanal cement (d) and a close-up of the same image (e) showed T2 hyperintensities consistent with spinal artery syndrome (white arrows).

cement leakage into the canal and without evidence of arterial embolization (Fig. 1e). The SpineJack implants were positioned well within the vertebral body, surrounded by cement. The patient was emergently transferred to a tertiary care center where her initial physical exam was notable for 0–1/5 strength in all left lower extremity muscle groups and 4/5 strength on the right side, ASIA D according to ASIA/ISCoS International Standards for Neurological Classification of Spinal Cord Injury (ISNCSCI). Patient reported that the sensation in the left leg was 10% and the right leg was 50% normal. She had baseline lower extremity neuropathy which made sensory exams including pain, temperature, and vibratory sense difficult. Left lower extremity (LLE) also showed long tract signs of hyperreflexia and sustained clonus, but not in the right LE. The rectal tone was diminished, but she had not had episodes of urinary or fecal incontinence. Urgent MRI of the thoracic spine showed that the extravasated cement was extradural and just adjacent to the spinal cord and no obvious signs of epidural hematoma [12] (Fig. 2a, b). After multidisciplinary discussions among intensivists, neurologists, spine surgeons, and radiologists, we considered iatrogenic injury during kyphoplasty, intradural cement, spinal cord infarct, and spinal artery syndrome, though diffusion-weighted imaging (DWI) that could have identified the syndrome was not obtained. Medical management included steroids and elevated mean arterial pressure (MAP) goals  $\geq 85$ . We had decided against anticoagulation initially because epidural hematoma was a possibility and she had an increased risk for bleeding because of medial positioning of the cannula during the kyphoplasty and because she was on anticoagulation pre-op though she did report she did not take the medication for 3 days. We also discussed additional imaging such as MRI with DWI or

angiography. Serial examinations showed progression of her deficit to 0–1/5 strength in the right tibialis anterior and extensor hallucis longus (ASIA C). Given the progression of her symptoms, we recommended and went ahead with surgical treatment 9 h after the completion of kyphoplasty.

### Operation

After T11 and T12 laminectomies, the dorsal surface of the dura was carefully inspected. We found no evidence of clots that would be consistent with epidural hematoma and no evidence of dural injury on the dorsal surface that would be seen if the dura were violated during kyphoplasty. Using a right T12 transpedicular approach, we confirmed that the dura did not appear to be violated and visualized the extravasated cement with gentle retraction of the dura. The dura easily fell away from the cement. The stalk of the cement was cut from the vertebral body using a curved osteotome and the cement in the canal was removed (Fig. 2c). The size of removed cement corresponded to that seen in the CT/MRI and we concluded that the cement was extradural and decided not to proceed with a durotomy. The remainder of the procedure, T11 to L2 instrumented fusion, was uncomplicated.

### Post-operative course and recovery

Post-operatively, she was admitted to the intensive care unit to maintain MAP over 85 mm Hg for spinal cord perfusion. We obtained an MRI of the thoracolumbar spine on post op day 1 that showed no evidence of residual protruding or intradural cement (Fig. 2d) but did show T2 hyperintensities of the spinal cord at the level of cement extravasation consistent with sequelae of spinal artery syndrome (Fig. 2d, e) [16]. Immediately post op, in the

recovery room, she started to have improvement of R LE motor function and by the next morning, R LE motor function improved to 4/5 as well as improved R LE sensation to her baseline. Her LLE strength remained 1/5 with diminished sensation. On post-operative day 30, she had regained global 4/5 LLE and 5/5 RLE strength with improved bilateral LE sensation and had returned to ambulating. The final follow-up was at 6 months by telephone at which time she stated that her back pain was better and that her walking was back to baseline. Her health failed from her multiple myeloma, and she passed 8 months after the surgery.

## DISCUSSION

We presented a case of progressive paraparesis due to spinal artery syndrome following kyphoplasty in the setting of a non-compressive extradural cement extravasation with near-complete neurological recovery following surgical decompression. We searched the Pubmed and Pubmed Central for {vertebroplasty or kyphoplasty} and {spinal artery syndrome} and found three cases, two of which were due to cement embolization during vertebroplasty [10, 11] and one with transient paraplegia after kyphoplasty without cement extravasation with clinical diagnosis of spinal artery syndrome without imaging confirmation who was given thrombolytic treatment with resolution of symptoms within 6 h [13].

In our present case, the multidisciplinary team initially treated the patient with steroids and elevated MAP goals and decided against anticoagulation given the concern for epidural hematoma and our patient's increased risk for bleeding. There is general agreement that maintaining MAP greater than 85 mmHg for 7 days has been associated with improved outcomes in spinal cord injuries [17]. Though IV heparin would be indicated in cases with thromboembolism, other treatments such as steroids remain controversial [18].

As her right leg weakness continued to worsen despite medical management, we felt justified in our decision to proceed with surgery and her almost immediate improvement of symptoms post op suggested that thrombosis was unlikely to be the cause of her symptoms and supported our approach. Post op MRI showed hyperintensities in the spinal cord at the level of the extravasated cement consistent with spinal artery syndrome. To the authors' best knowledge, this is the first case of MRI-confirmed spinal artery syndrome following cement augmentation procedure without cement embolization.

What would be the mechanism of spinal artery syndrome in our present case? The acute motor deficit and decreased lower extremity sensation would be consistent with anterior spinal artery syndrome. The preop MRI showed that the cement was just adjacent to the spinal cord on the right side, where the hyperintensities were seen in the post op MRI. Though there has not been a reported case of spinal artery syndrome in patients with cement extravasation, in three cases with spinal artery syndrome where the thoracic disc herniation just touched the spinal cord, two had anterior [19, 20] and one had posterior [21] spinal artery syndrome and the authors' proposed mechanism was compression of the radicular arteries that arise from the segmental arteries. We posit a similar mechanism in our case.

Limitations of this report include the lack of angiographic evidence of the disruption of the blood supply associated with the extravasated cement. Upon presentation to the tertiary care center, the multidisciplinary team discussed repeat MRI but the progression of her symptoms resulted in surgical treatment before MRI could be obtained. Even if pre-op MRI with DWI was obtained that showed spinal artery syndrome [16], because the localization of the blood supply disruption would be limited with DWI, one could not completely rule out the possibility that the spinal cord injury occurred at the time of the kyphoplasty and that cement extravasation and MRI findings were incidental findings or that the

hyperintensities seen were due to the surgical procedure. However, if that were the case, one would not expect to see a quick recovery of the neurological deficits after the surgery that was observed. Authors believe that the simplest explanation is that the cement extravasation caused spinal artery syndrome and that its removal resulted in prompt improvement of the neurological deficits.

We also do not know for certain whether the patient's neurological deficit would not have improved given more time. For example, in the three cases spinal artery syndrome with thoracic disc herniations that just touched the spinal cord, patients were successfully treated non-operatively [19–21]. If our patient's symptoms were stable, we might have continued to observe the patient initially. However, in the case of spinal artery syndrome following kyphoplasty without cement extravasation, the patient recovered after 6 h [13]. In the review of neurological deficits after cement extravasation [7], two patients with motor deficits who did not get surgery had no improvement of their deficits and a thoracic disc herniation causing spinal artery syndrome was treated successfully with surgery [22]. Therefore, given the progression of her deficits, we felt justified proceeding with the surgery 9 hours after kyphoplasty.

Another limitation is that we did not provide an explanation for why the patient's symptoms were worse on the lower extremity contralateral to the extravasated cement. Though this had not been previously reported with cement extravasation, there is a body of literature on patients with disc herniations who present with contralateral pain and symptoms. Recent reviews noted that lumbar disc herniation with contralateral symptoms was rare and suggested that the pathophysiology of contralateral symptoms was multifactorial [23, 24]. In cervical disc herniation, the role of lateral spinothalamic tract was discussed as a possible source of pain [25] and the tract could have played a role in this patient's symptoms. In the present case, left-sided symptoms improved after the removal of the cement on the right side.

Discussions on how to avoid complications during cement augmentation procedures are beyond the scope of this report but are found in the literature including indications and contraindications [26] and techniques [27].

Neurological deficits following vertebral cement augmentation procedures are rare but when they occur especially without severe cord compression, spinal artery syndrome should be considered, and the patient should be treated with elevated MAP goals and IV heparin if thromboembolism is likely. If the symptoms do not improve, then surgical management should be considered even if the extravasated cement is non-compressive and is contralateral to the more symptomatic extremity.

## DATA AVAILABILITY

All data from the above case report that was generated can be found within the article. Please contact the corresponding author with questions regarding any of the data included and/or details of the reported case.

## REFERENCES

- Buchbinder R, Osborne RH, Ebeling PR, Wark JD, Mitchell P, Wriedt C, et al. A randomized trial of vertebroplasty for painful osteoporotic vertebral fractures. *N Engl J Med*. 2009;361:557–68. <https://doi.org/10.1056/NEJMoa0900429>
- Kallmes DF, Comstock BA, Heagerty PJ, Turner JA, Wilson DJ, Diamond TH, et al. A randomized trial of vertebroplasty for osteoporotic spinal fractures. *N Engl J Med*. 2009;361:569–79. <https://doi.org/10.1056/NEJMoa0900563>
- Laredo JD, Hamze B. Complications of percutaneous vertebroplasty and their prevention. *Skelet Radio*. 2004;33:493–505. <https://doi.org/10.1007/s00256-004-0776-8>
- Wardlaw D, Cummings SR, Van Meirhaeghe J, Bastian L, Tillman JB, Ranstam J, et al. Efficacy and safety of balloon kyphoplasty compared with non-surgical care for vertebral compression fracture (FREE): a randomised controlled trial. *Lancet*. 2009;373:1016–24. [https://doi.org/10.1016/S0140-6736\(09\)60010-6](https://doi.org/10.1016/S0140-6736(09)60010-6)

5. Anderson PA, Froysheter AB, Tontz WL Jr. Meta-analysis of vertebral augmentation compared with conservative treatment for osteoporotic spinal fractures. *J Bone Min Res*. 2013;28:372–82. <https://doi.org/10.1002/jbmr.1762>
6. Yeom JS, Kim WJ, Choy WS, Lee CK, Chang BS, Kang JW. Leakage of cement in percutaneous transpedicular vertebroplasty for painful osteoporotic compression fractures. *J Bone Jt Surg Br*. 2003;85:83–9. <https://doi.org/10.1302/0301-620x.85b1.13026>
7. Sidhu GS, Kepler CK, Savage KE, Eachus B, Albert TJ, Vaccaro AR. Neurological deficit due to cement extravasation following a vertebral augmentation procedure. *J Neurosurg Spine*. 2013;19:61–70. <https://doi.org/10.3171/2013.4.SPINE12978>
8. Wilkes RA, Mackinnon JG, Thomas WG. Neurological deterioration after cement injection into a vertebral body. *J Bone Jt Surg Br*. 1994;76:155.
9. Lai PL, Tai CL, Chen LH, Nien NY. Cement leakage causes potential thermal injury in vertebroplasty. *BMC Musculoskelet Disord*. 2011;12:116. <https://doi.org/10.1186/1471-2474-12-116>
10. Tsai YD, Liliang PC, Chen HJ, Lu K, Liang CL, Wang KW. Anterior spinal artery syndrome following vertebroplasty: a case report. *Spine*. 2010;35:E134–6. <https://doi.org/10.1097/BRS.0b013e3181b52221>
11. Yazbeck PG, Al Rouhban RB, Slaba SG, Kreichati GE, Kharrat KE. Anterior spinal artery syndrome after percutaneous vertebroplasty. *Spine J*. 2011;11:e5–8. <https://doi.org/10.1016/j.spinee.2011.06.020>
12. Zou P, Gong HL, Wei JM, Wei DM, Qian LX, Liu P, et al. Spinal epidural hematoma after percutaneous kyphoplasty: case report and literature review. *J Pain Res*. 2020;13:2799–804. <https://doi.org/10.2147/JPR.S280650>
13. Bredow J, Oppermann J, Keller K, Beyer F, Boese CK, Zarghooni K, et al. Anterior spinal artery syndrome: reversible paraplegia after minimally invasive spine surgery. *Case Rep Orthop*. 2014;2014:205732. <https://doi.org/10.1155/2014/205732>
14. Premat K, Vande Perre S, Cormier É, Shotar E, Degos V, Morardet L, et al. Vertebral augmentation with the SpineJack® in chronic vertebral compression fractures with major kyphosis. *Eur Radio*. 2018;28:4985–91.
15. Noriega DC, Rodriguez-Monsalve F, Ramajo R, Sánchez-Lite I, Toribio B, Ardura F. Long-term safety and clinical performance of kyphoplasty and SpineJack® procedures in the treatment of osteoporotic vertebral compression fractures: a pilot, monocentric, investigator-initiated study. *Osteoporos Int*. 2019;30:637–45. <https://doi.org/10.1007/s00198-018-4773-5>
16. Lohrer TJ, Bassetti CL, Löfblad KO, Stepper FP, Sturzenegger M, Kiefer C, et al. Diffusion-weighted MRI in acute spinal cord ischaemia. *Neuroradiology*. 2003;45:557–61. <https://doi.org/10.1007/s00234-003-1023-z>
17. Catapano JS, John Hawryluk GW, Whetstone W, Saigal R, Ferguson A, Talbott J, et al. Higher mean arterial pressure values correlate with neurologic improvement in patients with initially complete spinal cord injuries. *World Neurosurg*. 2016;96:72–9. <https://doi.org/10.1016/j.wneu.2016.08.053>
18. Rahman M, Rahman S, Siddik AB, Hossain MD, Musa J, Hamjah R, et al. A review on the pathophysiology and management of anterior spinal artery syndrome. *J Spine Res Surg*. 2020;2:085–96.
19. Guest JD, Griesdale DE, Marotta T. Thoracic disc herniation presenting with transient anterior spinal artery syndrome. A case report. *Inter Neuroradiol*. 2000;6:327–31.
20. Reynolds JM, Belvadi YS, Kane AG, Pouloupoulos M. Thoracic disc herniation leads to anterior spinal artery syndrome demonstrated by diffusion-weighted magnetic resonance imaging (DWI): a case report and literature review. *Spine J*. 2014;14:e17–22. <https://doi.org/10.1016/j.spinee.2013.10.050>
21. Aalbers MW, Groen RJM, Appelman APA, Heersema TDJ, Wokke BHA, Oterdoom DLM. Spinal cord ischemia related to disc herniation: case report and a review of the literature. *Int Med Case Rep J*. 2021;14:429–33. <https://doi.org/10.2147/IMCRJ.S316797>
22. Santillan A, Goldberg JL, Carnevale JA, Kirnaz S, Hartl R, Knopman J. Anterior spinal artery syndrome caused by thoracic disc herniation. *J Clin Neurosci*. 2020;77:211–2. <https://doi.org/10.1016/j.jocn.2020.05.040>
23. Kesornsak W, Wasingpongwanich K, Kuansongtham V. Posterior epidural sequestered disc presenting with contralateral radiculopathy: a very rare case. *Spinal Cord Ser Cases*. 2021;7:98. <https://doi.org/10.1038/s41394-021-00460-z>
24. Ruschel LG, Agnoletto GJ, Aragão A, Duarte JS, de Oliveira MF, Teles AR. Lumbar disc herniation with contralateral radiculopathy: a systematic review on pathophysiology and surgical strategies. *Neurosurg Rev*. 2021;44:1071–81. <https://doi.org/10.1007/s10143-020-01294-3>
25. Yeung JT, Johnson JI, Karim AS. Cervical disc herniation presenting with neck pain and contralateral symptoms: a case report. *J Med Case Rep*. 2012;6:166. <https://doi.org/10.1186/1752-1947-6-166>
26. Momomura R, Shimamura Y, Kaneko K. Postoperative clinical outcomes of balloon kyphoplasty treatment: would adherence to indications and contraindications prevent complications? *Asian Spine J*. 2020;14:198–203. <https://doi.org/10.31616/asj.2019.0010>
27. Wong W, Mathis JM. Vertebroplasty and kyphoplasty: techniques for avoiding complications and pitfalls. *Neurosurg Focus*. 2005;18:e2. <https://doi.org/10.3171/foc.2005.18.33>

## ACKNOWLEDGEMENTS

The authors would like to thank the patient and her family for generously allowing us to present this case.

## AUTHOR CONTRIBUTIONS

Primary manuscript drafting was carried out by BMS and BCG. Manuscript editing as well as concept review/generation was a group task split equally among all authors. Direct care of the patient involved was provided by CZ and senior author, SK.

## COMPETING INTERESTS

The authors declare no competing interests.

## ETHICAL APPROVAL

Consent for publication of this patient case was obtained from the index patient's husband. Unfortunately, the patient had deceased prior to the drafting of the manuscript from an unrelated cause.

## ADDITIONAL INFORMATION

**Supplementary information** The online version contains supplementary material available at <https://doi.org/10.1038/s41394-023-00574-6>.

**Correspondence** and requests for materials should be addressed to Saechin Kim.

**Reprints and permission information** is available at <http://www.nature.com/reprints>

**Publisher's note** Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.

Springer Nature or its licensor (e.g. a society or other partner) holds exclusive rights to this article under a publishing agreement with the author(s) or other rightsholder(s); author self-archiving of the accepted manuscript version of this article is solely governed by the terms of such publishing agreement and applicable law.