



## Case Report

# Paraparesis As a Rare First Presentation Of Primary Hyperparathyroidism-Related Brown Tumor in The Thoracic Spine: Case report and literature review

Sultan M. Jarrar<sup>\*</sup>, Suleiman S. Daoud, Omar F. Jbarah, Iyad S. Albustami

Neurosurgery, Department of Clinical Neuroscience, Faculty of Medicine, Jordan University of Science & Technology, PO Box 3030, 22110, Irbid, Jordan



## ARTICLE INFO

## Keywords:

Brown tumor  
Primary hyperparathyroidism  
Thoracic spine tumor  
Spinal compression  
Case report

## ABSTRACT

**Introduction and importance:** Brown tumor (BT) is defined as osteolytic lesion of an underlying state of hyperparathyroidism. Hyperparathyroidism will activate osteoclasts which initiate active bone resorption foci of lytic-cysts with hemosiderin depositions that pigment it with its characteristic brown pathologic gross appearance. Devastating fractures and injuries can occur to affected bones and surrounding tissue that require emergent intervention and correction.

**Case presentation:** We present a case of a medically free 31-year-old female patient, who presented complaining of unsteadiness and progressive lower limbs weakness over 40 days of duration. Subsequent lab tests showed elevated PTH levels, along with 3.5 × 1.8 cm heterogeneous soft tissue mass involving the right pedicle on T7 level compressing the corresponding level of the spinal cord. Surgical management aimed to decompress the spinal cord and to obtain a biopsy for histopathologic examination which revealed a brown tumor. Neck ultrasound and Sestamibi scan indicated the presence of hyperactive and hyperplastic parathyroid tissue most suggestive of parathyroid adenoma.

**Clinical discussion:** Various presentations of Brown Tumor depend on the bone affected, despite the rarity of spinal involvement, yet expanding tumors can manifest either with back pain, radicular pain, paresthesia, weakness, paralysis, or incontinence. The highest incidence rates of spinal brown tumors affect adults over the age of 40. Management goals are to decompress the neuronal tissue emergently and to prevent further bony lytic deterioration.

**Conclusion:** The objective of this study is to provide an overview of primary hyperparathyroidism-related spinal brown tumors, presentation, and summary of previously reported similar cases in the literature.

## 1. Introduction

Brown tumor is an osteopathic reactive lesion that results from persistent elevation of parathyroid hormone, which aids calcium and phosphate homeostasis in the body. Hyperparathyroidism is either a primary parathyroid pathology or could be a compensatory mechanism in chronic calcium wasting conditions like chronic kidney diseases; both primary and secondary hyperparathyroidism were found responsible for the abnormal progressive bone remodeling activities seen in brown tumors [1,2].

The patient had a brown tumor involving the seventh thoracic vertebra T7 with bilateral lower limbs weakness and numbness as a rare

first presentation for primary normo-calcemic hyperparathyroidism. We have reported the case presentation, relative physical examination, management, and a literature review covering the 30 reported cases in the literature. This case report was reported in line with SCARE 2020 criteria [3].

### 1.1. Case presentation

We reported a case of a 31-year-old female who is medically free, presented with a history of gait unsteadiness and bilateral lower limbs weakness and numbness of 40 days duration. She addressed difficulties in standing and sitting when she is lying down. Her symptoms were

<sup>\*</sup> Corresponding author.

E-mail addresses: [smjarrar@just.edu.jo](mailto:smjarrar@just.edu.jo) (S.M. Jarrar), [ssdaoud@just.edu.jo](mailto:ssdaoud@just.edu.jo) (S.S. Daoud), [o.jbarah@gmail.com](mailto:o.jbarah@gmail.com) (O.F. Jbarah), [iyad.albustami@gmail.com](mailto:iyad.albustami@gmail.com) (I.S. Albustami).

<https://doi.org/10.1016/j.amsu.2021.102197>

Received 28 January 2021; Received in revised form 23 February 2021; Accepted 23 February 2021

Available online 27 February 2021

2049-0801/© 2021 The Authors. Published by Elsevier Ltd on behalf of IJS Publishing Group Ltd. This is an open access article under the CC BY license

(<http://creativecommons.org/licenses/by/4.0/>).

progressive in a manner that affected her productivity and daily activities.

The patient denied having difficulties in passing urine or stool. No reported history of fever, weight change, or trauma, and is not on any regular medications or supplements. The patient had no history of personal nor familial history of endocrine tumors. She used to take over-counter supplemental vitamins.

Clinically she was slim presented in a wheelchair with difficulty rising up to a standing position. Upon physical exam, the patient's vital signs were within normal ranges. She was awake and oriented. Cranial nerves were intact. Examination of her limbs showed full strength in upper limbs and -4/5 strength on her lower limbs. Deep tendon reflexes were 3+ on knee jerk and ankle jerk. The lower limbs were hypertonic. Babinski test showed upward going toes. Fine and pinprick sensations were intact. Digital rectal examination had normal sensation, normal tone, but weak power. No skin lesions were noted.

Her blood tests showed sky-high parathyroid hormone levels of 862 pg/ml (normal range is 15–65 pg/ml). Despite the elevated parathyroid hormone levels, Calcium, phosphorus, vitamin D, thyroid hormone levels and kidney function results were within the normal ranges. Also, a complete paraneoplastic markers assessment turned negative. Subsequently, she underwent parathyroid ultrasound which revealed evidence of well-defined hyperechoic mass lesion in the left anterior neck region, measuring about 2\*0.5\*0.6 cm. This lesion was highly suspicious for parathyroid adenoma.

Whole spine MRI was performed and revealed heterogenous soft tissue mass lesion involving the right pedicle, lamina, transverse process, and inferior articular process of T7, as well as the right posterolateral aspect of T7 vertebral body. This lesion appeared predominantly isointense on T1/T2WI with vivid enhancement on postcontrast images measuring about 3.5 × 1.8 cm. This lesion was shown to compress and displace the spinal cord from the right which causing compressive myelopathy on the corresponding level of the spinal cord (Fig. 1).

Contrasted multiple axis Computed Tomography of neck, chest, abdomen, and pelvis showed well defined enhancing mass lesion in the left anterior neck region. In addition to an expansile bony mass involving the body, right pedicles and posterior arch of T7 was noted. The lesion was associated with epidural soft tissue component extending from T6-T7 compressing the spinal cord and scalloping the T7 vertebral body (Fig. 2). Of note, there were generalized osteogenic changes involving the ribs, clavicle, scapula, and the examined spine.

Bone scan showed several foci of increased and decreased uptake in the axial bone skeleton mainly in the ribs and spine consistent with mixed osteolytic/osteoblastic bony lesions suggestive of brown tumors.

The patient expressed her distress of the emergent weakness that significantly impaired her ability to maintain daily activities, therefore surgery aimed to decompress and to stabilize the thoracic spine to allow

for motor function improvement, as well as to obtain histopathological examination.

The patient was prepared for surgery having her consent. We performed A posterior midline approach that involved T7 decompressive laminectomy, and removal of the tumor. For stabilization, we performed T5-T9 transpedicular screws fusion (Fig. 3). The surgery was conducted by team of consultant neurosurgeon and senior neurosurgery resident at tertiary university hospital. Post operatively, the patient had no complications and her lower limbs regained full strength and hypertonia was significantly improved.

Histopathological exam showed portions of bone containing a cellular spindle cell proliferation with abundant brown, granular and globular material and scattered groups of multinucleate giant cells of the osteoclast type, no malignant degeneration was noted. In the context of high parathyroid hormone levels, a diagnosis of brown tumor of hyperparathyroidism was made.

Work up for parathyroid adenoma continued with a Sestamibi scan that showed no scintigraphic evidence of parathyroid adenoma, however, findings were compatible with false negative test. The patient was referred for general surgery team for further management of her parathyroid lesion.

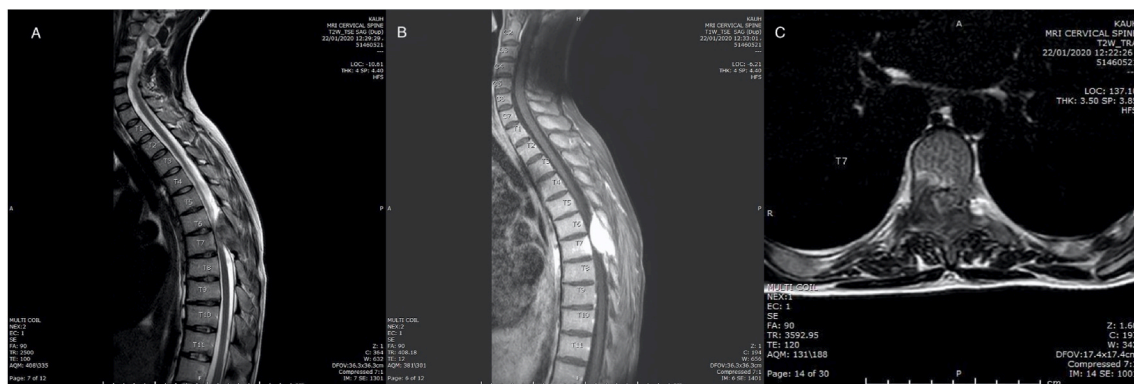
## 2. Discussion

Extracellular levels of calcium depend on regulatory mechanisms and signaling hormones. Parathyroid hormone plays an indispensable adjusting role in calcium homeostasis, tightly controlling calcium fluxing between bones, kidneys, intestine, and plasma [1].

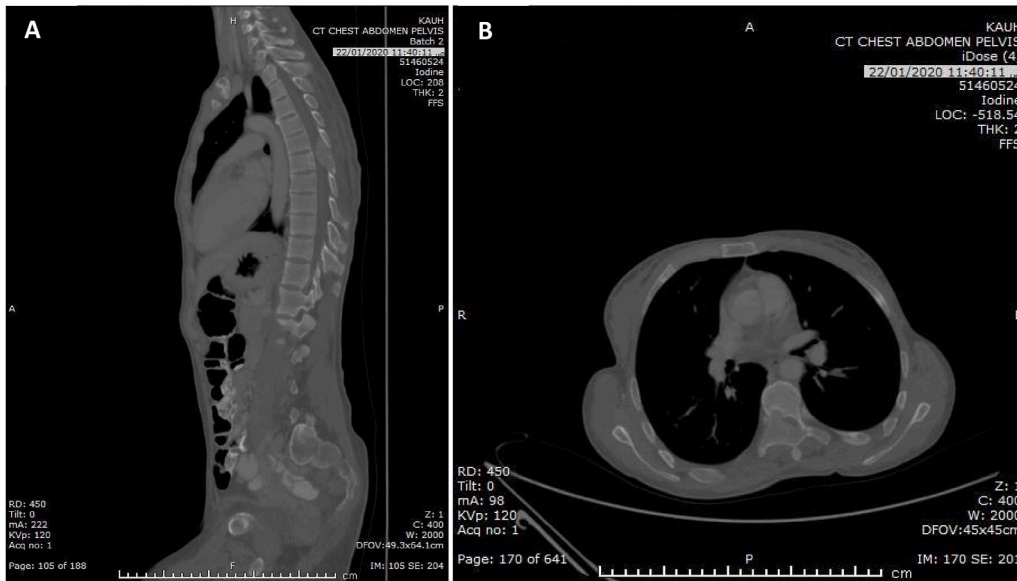
Abnormally sustained elevation of parathyroid hormone levels is considered a state of hyperparathyroidism, classified into primary, secondary, and tertiary; the latter two subtypes are sequelae of long-standing medical conditions especially chronic kidney diseases. Primary hyperparathyroidism manifest as either the classical variant of hypercalcemia or as normo-calcemic variant, the former variant is mostly attributed to parathyroid benign adenomas, malignant adenomas or parathyroid hyperplasia, while the normo-calcemic variant is of idiopathic origin, usually remains subclinical but has a potential to progress into hypercalcemic state and to cause bone targeted damage [2].

Biologically, parathyroid hormone stimulates bone resorbing activity of osteoclasts, thus hyperparathyroidism clinically manifest as metabolic bone disease; leads to abnormal structural changes and bone remodeling; includes osteopenia, salt & pepper skull, subchondral resorption, subperiosteal bone resorption-acroosteolysis, and focal lytic lesions [4].

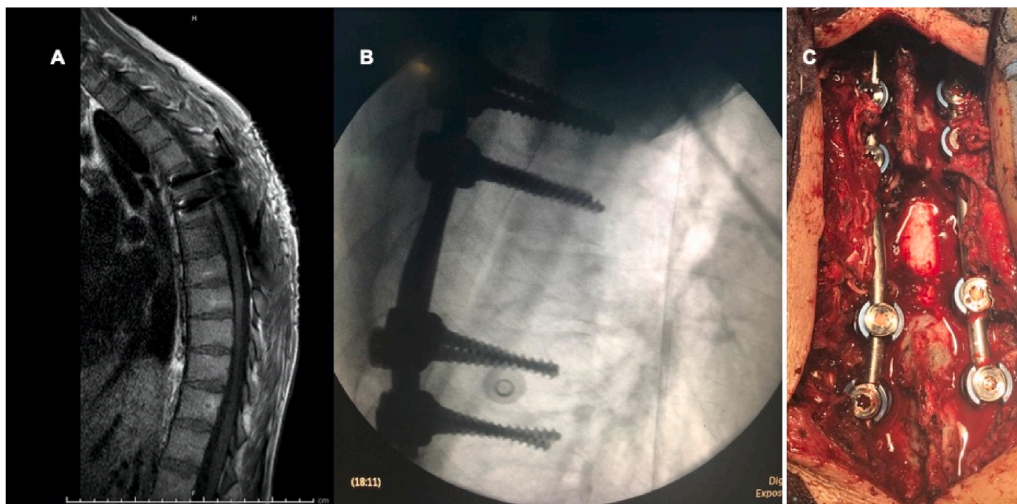
Brown tumors (BT), also known as osteitis fibrosa cystica are well demarcated, benign, rapidly growing lesions of lytic-cysts due to



**Fig. 1.** (A)(B) sagittal cervicothoracic MRI T2/T1 sequences showing large osteolytic bone lesion 3.5 × 1.8 cm, at level of T7. Significantly distorted the posterior elements of the corresponding vertebral spine. (C) axial MRI cut scan at level of T7 showing expansile osteolytic mass, involving the right pedicle and posterior arch, compressing the spinal canal and dislocating its content to the left.



**Fig. 2.** (A) sagittal chest abdomen pelvis CT showing expansile bony mass involving the body, right pedicles and posterior arch of T7. (B) axial T7 CT showing epidural soft tissue component extending from T6-T7 compressing the spinal canal, dislocating its content to the left and scalloping the T7 vertebral body.



**Fig. 3.** (A) sagittal MRI demonstrates segmental spinal instrumentation with T5-T9 transpedicular screws fusion. (B) lateral plain XRAY demonstrate T5-T9 transpedicular screws fusion. (C) intra-operative view of T5-T9 transpedicular screws fusion.

hyperparathyroidism, yet BTs are rare manifestation of today’s PTH clinical presentation and context when compared with secondary hyperparathyroidism. Researchers estimate that up to 3% of patients with PHPT develops BTs, the incidence is furtherly declining due to screening and health assessment programs [5].

Osteoclast-like multi-nucleated giant cells and hemosiderin laden macrophages cells are responsible for the radiological and gross features of brown tumor. The structural texture of brown tumor combine a hemorrhagic, cystic and solid components that constitute the distinctive diagnostic features of BTs on imaging modalities [6,7]. Brown tumor’s x-rays demonstrate a multifocal lucent lesion with cortical thinning and soap-bubbly appearance [5]. These bone destructive lesions could be single or multiple at any site involving long bones, hands, mandible, pelvis and ribs, which are way more common sites than the spine [8]. Table 1 shows a list of 30 case reports including this one, that reported a spinal involvement with a PHPT-related BT.

31 spinal lesions identified as PHPT-related brown tumors were reported in 30 case-reports, among these, 15 lesions (48.38%) were found

to affect the thoracic region which is the most common site for PHPT-BTs. The lumbar region is the second most affected site with 8 (25.80%) reported lesions. While the cervical and sacral regions were the least to be affected, 6 (19.35%), 2 (6.45%) respectively.

Brown tumor complications exceed the sole risk of pathological fractures following cortical thinning [7,9,10], since spinal BTs involving the vertebral body and posterior vertebral elements carry a potential risk of extension into the spinal canal or compressing its contents; that render patients with neurological impairments and deficits. Emergent presentations of spinal BTs require emergent decompressive interventions to relieve the nerve tissue; among these emergent presentations, asymptomatic PHPT patients show to have advanced bone damage that correlates with their sky-high levels of PTH [11–13].

### 3. Conclusions

Although of the benign nature of brown tumor, it can cause devastating fractures due to cortical thinning. In cases of spinal vertebrae

**Table 1**  
Previously reported cases of primary hyperparathyroidism-related brown tumors.

#	Author	year	Sex	Age	Level	Clinical	Management
1	Shaw and Davies [14]	1968	F	58	T10	Paraparesis, urinary retention	surgical decompression, parathyroidectomy
2	Shuangshoti et al. [15]	1972	M	32	L4	Paraparesis, radicular pain	surgical decompression, parathyroidectomy
3	Siu et al. [16]	1977	F	64	T10	Paraparesis, urinary retention	surgical decompression, parathyroidectomy
4	Sundaram and Scholz [17]	1977	F	63	T10	paraplegia and urinary retention	surgical decompression, parathyroidectomy
5	Ganesh et al. [18]	1981	M	40	T2	Paraparesis, radicular pain	Parathyroid adenoma excision only
6	Yokota et al. [19]	1989	F	58	T5	Paraparesis, numbness	surgical decompression, parathyroidectomy
7	Daras et al. [20]	1990	F	54	T9	Paraparesis	surgical decompression
8	Kashkari et al. [21]	1990	F	51	T6-T7	Paraparesis	surgical decompression Parathyroidectomy
9	Sarda et al. [22]	1993	F	23	T3-T4	Paraplegia, radicular pain	surgical decompression, parathyroidectomy
10	Motateanu et al. [23]	1994	M	57	L4-5	Radiculopathy	surgical decompression, parathyroidectomy
11	Ashebu et al. [24]	2002	F	27	C6	Bilateral renal calculi, lethargy, weakness, multiple brown tumors	Parathyroidectomy + Orthosis
12	Mustonen et al. [25]	2004	M	28	L2	Radiculopathy, numbness	Resection of parathyroid adenoma only
13	Haddad et al. [26]	2007	F	62	T2-T3	Paraparesis, numbness	surgical decompression, parathyroidectomy
14	Khalil et al. [27]	2007	M	69	L2	Radiculopathy	surgical decompression
15	Altan et al. [28]	2007	F	44	S2	Radiculopathy, low back pain	surgical decompression parathyroidectomy
16	Hoshi et al. [29]	2008	F	23	Sacrum	ureterolithiasis, Radiculopathy	Resection of parathyroid adenoma only
17	Kerstens et al. [30]	2013	M	55	C7/L3	Weight loss, bone pain	Parathyroidectomy
18	Lee et al. [12]	2013	M	65	L1-L2	Low back pain, radicular pain	surgical decompression parathyroidectomy
19	Khalatbari and Moharamzad [6]	2014	F	52	C6	Radiculopathy, neck pain	Parathyroidectomy, surgical decompression
20	Khalatbari and Moharamzad [6]	2014	M	38	T7	Paraparesis, sphincter dysfunction	Parathyroidectomy, surgical decompression
21	Khalatbari and Moharamzad [6]	2014	M	16	L2	Paraparesis, sphincter dysfunction	Parathyroidectomy, surgical decompression
22	Khalatbari and Moharamzad [6]	2014	F	46	L3	Paraparesis, low back pain	Parathyroidectomy, surgical decompression
23	Sonmez et al. [8]	2015	M	50	T9	Paraparesis, sphincter dysfunction	surgical decompression, Parathyroidectomy
24	Alfawareh et al. [13]	2015	F	26	C2	axial neck pain	Parathyroidectomy + Orthosis
25	Heidarpour et al. [11]	2017	M	33	T4	paraplegia, GI bleeding	parathyroidectomy, surgical decompression
26	Carta et al. [9]	2019	M	48	C2	neck pain and quadriparesis	non-operative orthosis with parathyroidectomy
27	Hu et al. [10]	2019	F	50	T9	Paraparesis, thoracic back pain	parathyroidectomy
28	Hammou et al. [31]	2020	F	65	C5	Quadraparesis	surgical decompression, Parathyroidectomy
29	Shaaban et al. [32]	2020	M	37	T4-T5	Paraparesis	Emergent decompression and parathyroidectomy
30	this one	2021	F	30	T7	Paraparesis	Surgical decompression and parathyroidectomy

involvement, brown tumors can cause neurologic symptoms of paresis, paresthesia, or paralysis because of spinal compression. In symptomatic cases it is recommended to ensure tumor resection, spinal decompression and stabilization, along with medical or surgical parathyroid intervention to control the underlying cause.

### Methodology

This case-report has been reported in line with the SCARE 2020 criteria.

### Source of funding

This study was not funded

### Consent

Written informed consent was obtained from patient for publication of the case report and any related images. A copy of the written consent is available for review by the editor-in-chief of this journal on request.

### Author contribution

This work was carried out in collaboration between all authors. Authors SMJ and SSD designed the study. Author ISD managed the literature searches, data collection and wrote the first draft of the manuscript. Author OFJ managed the literature searches and completed the final draft. All authors read and approved the final manuscript.

### Ethical approval

It is not applicable.

### Sources of funding

The authors declare that this case report and literature review are not funded.

### Author contribution

This work was carried out in collaboration between all authors. Authors SMJ and SSD designed the study. Author ISD managed the literature searches, data collection and wrote the first draft of the manuscript. Author OFJ managed the literature searches and completed the final draft. All authors read and approved the final manuscript.

### Registration of research studies

Name of the registry: N/a.

Unique Identifying number or registration ID: N/a.

Hyperlink to your specific registration (must be publicly accessible and will be checked): N/a.

### Guarantor

Sultan M. Jarrar, MD, Assistant professor of Neurosurgery, Department of clinical neuroscience.

Faculty of medicine, Jordan University of Science & Technology PO

Box 3030 zip code 22110. IrbidJordan, Telephone: 00962 790033567, Email: smjarrar@just.edu.jo.

### Provenance and peer review

Not commissioned, externally peer-reviewed.

### Declaration of competing interest

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

### Appendix A. Supplementary data

Supplementary data related to this article can be found at <https://doi.org/10.1016/j.amsu.2021.102197>.

### References

- [1] M. Peacock, Calcium metabolism in health and disease, *Clin. J. Am. Soc. Nephrol.* 5 (2010) S23–S30, <https://doi.org/10.2215/CJN.05910809>.
- [2] J.P. Bilezikian, N.E. Cusano, A.A. Khan, J.M. Liu, C. Marcocci, F. Bandeira, Primary hyperparathyroidism, *Nat. Rev. Dis. Prim.* 2 (2016) 1–16, <https://doi.org/10.1038/nrdp.2016.33>.
- [3] R.A. Agha, T. Franchi, C. Sohrabi, G. Mathew, A. Kerwan, A. Thoma, A.J. Beamish, A. Noureldin, A. Rao, B. Vasudevan, B. Challacombe, B. Perakath, B. Kirshtein, B. Ekser, C.S. Pramesh, D.M. Laskin, D. Machado-Aranda, D. Miguel, D. Pagano, F. H. Millham, G. Roy, H. Kadioglu, L.J. Nixon, I. Mukhejee, J.A. McCaul, J. Chi-Yong Ngu, J. Albrecht, J.G. Rivas, K. Raveendran, L. Derbyshire, M.H. Ather, M. A. Thorat, M. Valmasoni, M. Bashashati, M. Chalkoo, N.Z. Teo, N. Raison, O. J. Muensterer, P.J. Bradley, P. Goel, P.S. Pai, R.Y. Affifi, R.D. Rosin, R. Coppola, R. Klappenbach, R. Wynn, R.L. De Wilde, S. Surani, S. Giordano, S. Massarut, S. G. Raja, S. Basu, S.A. Enam, T.G. Manning, T. Cross, V.K. Karanth, V. Kasivisvanathan, Z. Mei, The SCARE 2020 guideline: updating consensus surgical CAse REport (SCARE) guidelines, *Int. J. Surg.* 84 (2020) 226–230, <https://doi.org/10.1016/j.ijsu.2020.10.034>.
- [4] J. Bennett, J.W. Suliburk, F.E. Morón, Osseous manifestations of primary hyperparathyroidism: imaging findings, *Internet J. Endocrinol.* 2020 (2020), <https://doi.org/10.1155/2020/3146535>.
- [5] C. Grégoire, M. Soussan, M.L. Dumuis, N. Naggara, A. Martin, R. Dhote, V. Audard, A. Neuman, P. Weinmann, Contribution of multimodality imaging for positive and aetiological diagnosis of multiple brown tumours, *Ann. Endocrinol.* 73 (2012) 43–50, <https://doi.org/10.1016/j.ando.2011.10.002>.
- [6] M.R. Khalatbari, Y. Moharamzad, Brown tumor of the spine in patients with primary hyperparathyroidism, *Spine (Phila. Pa. 39 (2014) (1976) E1073–E1079*, <https://doi.org/10.1097/BRS.0000000000000455>.
- [7] C.Y. Chang, D.I. Rosenthal, D.M. Mitchell, A. Handa, S.V. Kattapuram, A.J. Huang, Imaging findings of metabolic bone disease, *Radiographics* 36 (2016) 1871–1887, <https://doi.org/10.1148/rg.2016160004>.
- [8] E. Sonmez, T. Tezcaner, I. Coven, A. Terzi, Brown tumor of the thoracic spine : first manifestation of primary hyperparathyroidism, *J. Korean Neurosurg. Soc.* 58 (2015) 389–392, <https://doi.org/10.3340/jkns.2015.58.4.389>.
- [9] S. Carta, A. Chung, S.R. Gowda, E. Synodinou, P. Sauve, J. Harvey, Brown tumour in the cervical spine : case report and review of literature, *Case Reports Clin. Pathol.* 6 (2019) 27, <https://doi.org/10.5430/crep.v6n1p27>.
- [10] J. Hu, S. He, J. Yang, C. Ye, X. Yang, J. Xiao, Management of brown tumor of spine with primary hyperparathyroidism: a case report and literature review, *Medicine (Baltim.)* 98 (2019), e15007, <https://doi.org/10.1097/MD.00000000000015007>.
- [11] M. Heidarpour, M. Karami, P. Hedayat, A. Aminorroaya, Two rare manifestations of primary hyperparathyroidism: paralysis and peptic ulcer bleeding, *Endocrinol. Diabetes Metab. Case Reports* (2017) (2017), <https://doi.org/10.1530/edm-17-0059>.
- [12] J.H. Lee, S.M. Chung, H.S. Kim, Osteitis fibrosa cystica mistaken for malignant disease, *Clin. Exp. Otorhinolaryngol.* 6 (2013) 110–113, <https://doi.org/10.3342/ceo.2013.6.2.110>.
- [13] M.D. Alfawareh, M.M. Halawani, W.I. Attia, K.N. Almusrea, Brown tumor of the cervical spines: a case report with literature review, *Asian Spine J* 9 (2015) 110–120, <https://doi.org/10.4184/asj.2015.9.1.110>.
- [14] M.T. Shaw, M. Davies, Primary hyperparathyroidism presenting as spinal cord compression, *Br. Med. J.* 4 (1968) 230–231, <https://doi.org/10.1136/bmj.4.5625.230>.
- [15] S. Shuangshoti, C. Hongsaprabhas, S. Chandraprasert, B. Rajatapiti, Parathyroid adenoma, brown tumor and cauda equina compression, *J. Med. Assoc. Thai.* 55 (1972) 251–258, accessed, <http://www.ncbi.nlm.nih.gov/pubmed/5022500>. (Accessed 25 April 2020).
- [16] K. Siu, M. Sundaram, C. Schultz, L. Kirwan, Primary hyperparathyroidism presenting as spinal cord compression: report OF a case, *Aust. N. Z. J. Surg.* 47 (1977) 668–672, <https://doi.org/10.1111/j.1445-2197.1977.tb06601.x>.
- [17] M. Sundaram, C. Scholz, Primary hyperparathyroidism presenting with acute paraplegia, *Am. J. Roentgenol.* 128 (1977) 674–676, <https://doi.org/10.2214/ajr.128.4.674>.
- [18] A. Ganesh, S. Kuriant, L. John, Complete recovery of spinal cord compression following parathyroidectomy, *Postgrad. Med.* 57 (1981) 652–653, <https://doi.org/10.1136/pgmj.57.672.652>.
- [19] N. Yokota, T. Kuribayashi, M. Nagamine, M. Tanaka, S. Matsukura, S. Wakisaka, Paraplegia caused by brown tumor in primary hyperparathyroidism. Case report, *J. Neurosurg.* 71 (1989) 446–448, <https://doi.org/10.3171/jns.1989.71.3.0446>.
- [20] M. Daras, T. Georgakopoulos, D. Avdelidis, A. Gravani, A.J. Tuchman, Spinal cord compression in primary hyperparathyroidism: report of a case and review of the literature, *Spine (Phila. Pa. 15 (1990) (1976) 238–240*, <https://doi.org/10.1097/00007632-199003000-00019>.
- [21] S. Kashkari, T.R. Kelly, D. Bethem, R.G. Pepe, Osteitis fibrosa cystica (brown tumor) of the spine with cord compression: report of a case with needle aspiration biopsy findings, *Diagn. Cytopathol.* 6 (1990) 349–353, <https://doi.org/10.1002/dc.2840060512>.
- [22] A.K. Sarda, Arunabh, M. Vijayaraghavan, M. Kapur, Paraplegia due to osteitis fibrosa secondary to primary hyperparathyroidism: report of a case, *Surg. Today.* 23 (1993) 1003–1005, <https://doi.org/10.1007/bf00308978>.
- [23] M. Motateanu, J.P. Déruaz, H. Fankhauser, Spinal tumour due to primary hyperparathyroidism causing sciatica: case report, *Neuroradiology* 36 (1994) 134–136, <https://doi.org/10.1007/bf00588079>.
- [24] S.D. Ashebu, M.H. Dahniya, S.A. Muhtaseb, P. Aduh, Unusual florid skeletal manifestations of primary hyperparathyroidism, *Skeletal Radiol.* 31 (2002) 720–723, <https://doi.org/10.1007/s00256-002-0578-9>.
- [25] A.O.T. Mustonen, M.J. Kiuru, A. Stahls, T. Bohling, A. Kivioja, S.K. Koskinen, Radicular lower extremity pain as the first symptom of primary hyperparathyroidism, *Skeletal Radiol.* 33 (2004) 467–472, <https://doi.org/10.1007/s00256-004-0803-9>.
- [26] F.H. Haddad, O.M. Malkawi, A.A. Sharbaji, I.F. Jbara, H.R. Rihani, Primary hyperparathyroidism. A rare cause of spinal cord compression, *Saudi Med. J.* 28 (2007) 783–786.
- [27] P.N. Khalil, S.M. Heining, R. Huss, S. Ihrler, M. Siebeck, K. Hallfeldt, E. Euler, W. Mutschler, Natural history and surgical treatment of brown tumor lesions at various sites in refractory primary hyperparathyroidism, *Eur. J. Med. Res.* 12 (2007) 222–230.
- [28] L. Altan, Z. Kurtoğlu, U. Yalçınkaya, U. Aydınlı, E. Ertürk, Brown tumor of the sacral spine in a patient with low-back pain, *Rheumatol. Int.* 28 (2007) 77–81, <https://doi.org/10.1007/s00296-007-0380-z>.
- [29] M. Hoshi, M. Takami, M. Kajikawa, K. Teramura, T. Okamoto, I. Yanagida, A. Matsumura, A case of multiple skeletal lesions of brown tumors, mimicking carcinoma metastases, *Arch. Orthop. Trauma Surg.* 128 (2008) 149–154, <https://doi.org/10.1007/s00402-007-0312-0>.
- [30] M.N. Kerstens, R. De Vries, J.T.M. Plukker, R.H.J.A. Slart, R.P.F. Dullaart, Multiple skeletal lesions on FDG PET in severe primary hyperparathyroidism, *Eur. J. Nucl. Med. Mol. Imag.* 41 (2014) 182–183, <https://doi.org/10.1007/s00259-013-2509-5>.
- [31] O. Hammou, H. Azzouzi, L. Ichchou, Spinal cord compression secondary to brown tumor as first manifestation of primary hyperparathyroidism, *World Neurosurg* 137 (2020) 384–388, <https://doi.org/10.1016/j.wneu.2020.02.076>.
- [32] A.T.E. Shaaban, M. Ibrahim, A. Saleh, A. Haider, A. Alyafai, Brown tumor due to primary hyperparathyroidism resulting in acute paraparesis: case report and literature review, *Surg. Neurol. Int.* 11 (2020) 355, [https://doi.org/10.25259/sni.653\\_2020](https://doi.org/10.25259/sni.653_2020).