Papillary Thyroid Carcinoma Metastasis to the Lumbar Spine Masquerading as a Schmorl's Node

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Abstract A Schmorl's node is a common incidental finding encountered during radiologic imaging. Despite the vertebral body being a common site of metastatic disease, a lytic lesion adjacent to an endplate with typical imaging features can often confidently be called a Schmorl's node. This is a case report of a patient with a single well-defined FDG-avid papillary thyroid carcinoma metastasis to the spine that had imaging findings characteristic of a Schmorl's node on CT and MRI. This case is important to consider as it demonstrates that the imaging characteristics of metastatic disease and Schmorl's nodes can overlap.

Keywords Thyroid cancer · Schmorl's node · Thyroglobulin · FDG · PET · MRI

Introduction

A Schmorl's node is a common incidental finding encountered during radiologic imaging. While a Schmorl's node may be associated with back pain, it is typically an incidental finding discovered in a patient without a reported history of back pain

[1]. Although the precise cause of a Schmorl's node is unknown, it is thought to be related to weakening of the vertebral endplate and herniation of the adjacent disc into the vertebral body [2, 3]. The vertebral bodies most frequently involved are near the thoracolumbar junction, with the superior endplate affected more often than the inferior endplate [4]. Despite the vertebral body being a common site of metastatic disease, a lytic lesion adjacent to an endplate with typical imaging features can often confidently be called a Schmorl's node. This is a case report of a patient with a single well-defined FDG-avid papillary thyroid carcinoma metastasis to the spine that had imaging findings characteristic of a Schmorl's node on CT and MRI. This case is important to consider as it demonstrates that the imaging characteristics of metastatic disease and Schmorl's nodes can overlap.

Case Report

A 56-year-old male underwent a total thyroidectomy and lymph node dissection on 16 March 2009 for a 2.2-cm papillary thyroid carcinoma (PTC) with follicular, trabecular and Hurthle cell features. The tumor was intrathyroidal. None of five removed lymph nodes were positive for cancer. There was no prior history of head or neck irradiation. Apart from prior hypertension, his past medical history was unremarkable. When first seen at this institution on 27 April 2009, his thyroid-stimulating hormone (TSH) level was minimally elevated at 7.0 uU/ml with a serum thyroglobulin (Tg) concentration of 1.2 ng/ml. Serum thyroglobulin antibodies were negative at that time and throughout his follow-up.

Levothyroxine was stopped; liothyronine was prescribed and then discontinued after 4 weeks. A low iodine diet was employed for 2 weeks prior to radioactive iodine administration. In early June 2009, thyroid blood tests immediately prior to the administration of 2 mCi of I-131 demonstrated an

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elevated TSH value of 70.5 uU/ml, a low free T4 level of <0.4 ng/dl and a dramatically elevated serum thyroglobulin value of 3,531 ng/ml. A radioiodine whole-body scan (WBS) revealed a percent uptake of 0.85 % in the thyroid bed. No other sites of abnormal uptake were seen. The patient received 150 mCi of I-131, and a post-treatment scan again revealed only thyroid bed uptake without evidence of metastatic disease. To be certain that the serum Tg concentration was not a laboratory error, serum Tg was re-drawn. The repeat concentration was 2,528 ng/ml. Two weeks post radioiodine therapy, an FDG-PET/CT examination was performed. The only abnormality noted was an intensely FDG-avid lesion in the L5 vertebral body with a SUV_{max} of 14 (Fig. 1a and c). The L5 lesion was described as a 1.4-cm lytic lesion on CT with a sclerotic margin in continuity with the superior endplate (Fig. 1b). This was considered most compatible with a Schmorl's node, although a contrast-enhanced MRI was recommended for further evaluation.

Approximately 3 weeks after the PET/CT scan, a gadolinium-enhanced lumbar spine MRI was performed. The L5 lesion demonstrated peripheral T2 hyperintensity and central T2 hypointensity (Fig. 2a and c). On T1 imaging, there was peripheral hypointensity and central hyperintensity (Fig. 2b). There was also a rim of surrounding bone marrow edema (Fig. 2a). The lesion demonstrated peripheral enhancement with additional enhancement of the adjacent bone marrow (Fig. 2d). There was a defect in the superior endplate of L5 that communicated with the lesion, best seen on the sagittal T1 images (Fig. 2b). These findings were considered most compatible with a superior endplate Schmorl's node with a probable associated superior endplate compression fracture.

In light of the concern regarding the serum Tg concentration, careful observation was elected. Two months after the MRI, the serum TSH level was normal at 1.1 uU/ml, and the serum Tg was undetectable at 0.2 ng/ml. In March 2010, a 4-mCi I-131 WBS performed after thyrotropin alpha stimulation revealed no abnormal uptake. The serum Tg concentration

drawn at the time of the radioiodine scan was 20 ng/ml. This rise in serum Tg was attributed to the TSH stimulation. Accordingly, a follow-up serum Tg concentration drawn in January 2011 was 0.3 ng/ml.

Over the next 3 ½ years, the patient continued to feel well. The serum Tg concentration while on thyroid hormone suppressive therapy gradually increased, rising to 8.3 ng/ml in June 2013. Anatomic imaging of the neck with ultrasound and contrast-enhanced CT revealed no evidence of malignancy. A Tc-99 m MDP bone scan also did not find evidence of malignancy.

In December of 2013, the serum Tg concentration had increased further to 34.0 ng/ml, raising suspicion for recurrent malignancy. An FDG PET/CT examination was obtained and revealed an increase in size and FDG avidity of the L5 vertebral body lesion, measuring 2.4 cm with a SUV_{max} of 48 (Fig. 3a, b and c). There were no other sites of abnormal FDG uptake. In light of the increasing serum Tg and the changes on PET/CT, the decision was made to biopsy the L5 lesion as this could represent a site of metastatic disease. The lesion was biopsied under CT guidance and demonstrated metastatic PTC. After discussing various therapeutic options including radiofrequency ablation and cryoablation, the L5 lesion was treated with intensity modulated radiation therapy of 1,800 cGy in a single fraction.

Discussion

This case demonstrates a number of unusual features. Serum Tg is an important tumor marker for well-differentiated thyroid carcinoma [5]. A serum Tg value in the thousands is generally indicative of disseminated metastatic disease and is often seen in the setting of multiple bony metastases. It was therefore surprising to find no evidence of metastatic disease on initial imaging. Perhaps even more surprising was the decline in the serum Tg from the 2,000 to 30,00 range to

Fig. 1 FDG-PET/CT in July 2009. **a** FDG-PET shows intense FDG uptake in the L5 vertebra body. There are no other sites of abnormal FDG uptake. **b** CT shows an L5 lytic lesion in continuity with the L4-5 disc space. **c** Fusion imaging coregisters the two findings to the superior endplate of L5

Fig. 2 MRI of the lumbar spine in July 2009. Intravenous contrast was Magnevist (gadopentetate dimeglumine) 0.1 mmol/kg, weight-based. **a** T2-weighted imaging demonstrates a lesion in the L5 vertebral body with a ‘concentric ring’ of marrow edema. **b** T1-weighted and **c** T2-weighted fat-saturated images demonstrate signal intensity in the lesion similar to that of the adjacent intervertebral disc and continuity with the disc space. **d** Post-contrast T1-weighted fat saturation image shows enhancement of the lesion periphery and adjacent marrow

undetectable concentrations after treatment. Although Tg is known to be TSH responsive (i.e., the higher the TSH, the higher the serum Tg), TSH stimulation generally causes only a 5–20-fold increase in Tg (compared to values during TSH suppression), not a 1,000-fold stimulation. Antibodies against

Tg may cause falsely low measurements of serum Tg [5]. The serum Tg antibodies were consistently low in this case.

While serum Tg is a useful tool in the detection of residual/recurrent differentiated thyroid cancer, it does not imply that the detected malignancy will have radioiodine uptake on

Fig. 3 FDG-PET/CT in January 2014. **a** FDG-PET demonstrates an interval increase in the tracer avidity associated with the L5 vertebral lesion. As before, there are no other sites of abnormal FDG uptake. **b**

CT shows interval growth of the L5 lytic lesion. **c** Fusion imaging again coregisters the two findings to the superior endplate of L5

WBS. Likewise, sites of uptake on radioiodine WBS do not indicate that there will be elevation of the serum Tg. The complimentary nature of the tests make them both useful in the management of PTC. There are multiple reasons for possible discordance between these two tests, such as an elevated serum Tg without abnormal uptake on radioiodine WBS (as presented here). Briefly, these include improper patient preparation, defective iodine trapping and dispersed microscopic metastases too small to be detected [6]. With regard to the latter, ‘too small to be detected lesions’ are sometimes not seen with a diagnostic dose of radioiodine, but are then apparent with a therapeutic dose of radioiodine. Consequently, the presumed presence of ‘too small to be detected lesions’ is usually considered justification for empiric therapy in the setting of elevated Tg without abnormal uptake on radioiodine WBS. Still, the treatment of differentiated thyroid cancer in this setting remains controversial. A Cochrane meta-analysis performed by Ma et al. in 2009 indicated that the evidence available at that time was insufficient to reliably assess the potential of radioiodine treatment for these patients [7]. While this review was unable to make a firm recommendation, other reports have demonstrated reduction of serum Tg levels after radioiodine therapy in patients with elevated serum Tg levels without abnormal uptake on a radioiodine WBS. These have stated that radioiodine therapy may be justified in patients with serum Tg levels of >10 ng/ml and no abnormal uptake on radioiodine WBS who are at high risk of recurrence [8]. There is no definitive explanation as to why the patient had such a dramatic elevation of the serum Tg concentration followed by a decrease to undetectable levels post-therapy. Given the eventual diagnosis of metastatic PTC, a malignant explanation is possible, although this is confounded by the lack of extensive disease on initial imaging.

As stated above, not all metastatic lesions of PTC take up radioactive iodine. The BRAF V600E mutant PTC is more likely to demonstrate aggressive behavior, including cervical and distant metastases and increased mortality [9]. This mutation, which has been reported to occur in approximately 50 % of patients with PTC, also prevents radioactive iodine uptake in the tumor by downregulating the sodium-iodide symporter [10, 11]. The BRAF mutation status in this case is not known. Histologic variants of PTC also have differing levels of expected radioiodine uptake. For example, Hurthle cell carcinoma is known to have the ability to synthesize Tg, but has poor radioiodine uptake [12]. Radioactive iodine and FDG uptakes are complementary in metastatic well-differentiated thyroid carcinoma. That is, tumors that are radioactive iodine avid are less likely to be FDG avid, and tumors that are FDG avid are less likely to take up radioactive iodine [13].

After lymph node metastases, lung and bone are the most common sites of metastatic disease in PTC. For example, in a study of 5,700 patients with PTC not known to have distant metastases at diagnosis, 7 % developed nodal recurrences, 2 % developed lung recurrences and 0.6 % developed bone recurrences [14]. With PTC, the majority of patients with bony metastases also have pulmonary metastases, and the majority of patients with bony metastases have more than one focus of metastasis. Clearly this patient’s solitary L5 vertebral body lesion was an atypical presentation for metastatic PTC.

The CT appearance was unusual for a thyroid metastasis. While both Schmorl’s nodes and thyroid metastases can be described as lytic, the faint sclerotic margin seen in this case is not typical for both. Untreated thyroid metastases would not have a sclerotic margin as they do not incite an osteoblastic reaction. In contrast, Schmorl’s nodes typically have a sclerotic margin that is in continuity with the vertebral endplate, best appreciated in this case at the anterior and posterior margins (Fig. 1b). Thus, the CT morphology on initial imaging favored Schmorl’s node over metastatic PTC.

With regard to the PET evaluation, FDG avidity is a recognized feature of Schmorl’s nodes. FDG uptake was first reported in a Schmorl’s node in a 2011 case report [15]. In a subsequent study, 12 patients with Schmorl’s nodes were evaluated with MRI and FDG-PET, and all had FDG uptake [16]. It should be noted that the authors did not state the indication for FDG PET/CT in these patients, which could clearly affect the pretest probability of any lesion found on imaging. On average, the nodes with associated gadolinium enhancement had slightly more intense FDG uptake than those without enhancement, with a SUV_{max} of 1.75 in the enhancing nodes versus a SUV_{max} of 1.09 in the nonenhancing nodes. The patient reported here had similar findings of FDG uptake and MR enhancement in the lytic L5 lesion; however, the intensity of the FDG uptake in this patient was greater than the average SUV_{max} described in this article. The upper limit of FDG uptake that can be associated with a benign Schmorl’s node has not been established; thus, the intense FDG uptake in the L5 lesion was considered to be of uncertain etiology, which prompted further characterization with MRI, as described in the case report.

Wu et al. evaluated 47 patients with edematous Schmorl’s nodes on MRI [17]. A central finding of this study was the identification of concentric rings of marrow edema adjacent to the Schmorl’s node analogous to endplate degenerative changes, which had a high negative predictive value for ‘idiopathic’ Schmorl’s nodes without underlying fracture, infection or malignancy. The patient being presented also had a concentric pattern of edema on the axial T2 fast spin echo images. (Fig. 2a) The Wu et al. study also reported that T1/T2 signal isointense to the adjacent intervertebral disc was only seen in one-third of the cases, although this is a feature that has been used to describe Schmorl’s node [3]. Finally,

they identified bulging of herniated discs in half of their Schmorl's nodes, a finding present in this patient. Thus, the findings on the initial MRI were more consistent with a Schmorl's node than a metastatic lesion.

While the L5 lesion in this patient had many imaging characteristics of a Schmorl's node, the location of the lesion was somewhat uncharacteristic. The studies performed by Dar et al. and Pfirrmann et al. provide a thorough description of Schmorl's nodes throughout the spine [4, 18]. Both studies described L5 as an uncommon site for Schmorl's node formation. Additionally, the Schmorl's nodes described were typically seen at the inferior endplate and at the middle portion of the plate. The lesion in this patient was located at the anterior aspect of the superior endplate. Two subsets of Schmorl's nodes have been reported to be more common at the superior endplate: the giant cystic Schmorl's node and giant fatty Schmorl's node [19, 20]. These giant Schmorl's nodes are also more commonly found in the lower lumbar spine rather than the thoracolumbar spine. The imaging findings in the patient being reported would not support a fatty or cystic lesion; however, it is important to consider these variants when evaluating an atypical endplate lesion.

The morphology of the endplate and the intervertebral disc had conflicting features for a Schmorl's node. Flat vertebral endplates are associated with Schmorl's node formation, whereas those with a 'Cupid's bow' deformity are not [4]. It is hypothesized that a 'Cupid's bow' formation, which is more common in the lumbar spine and was seen in this patient, provides some protection against the formation of Schmorl's nodes by increasing the surface area of the endplate and placing less pressure on the nucleus pulposus. Specific non-advanced degenerative changes in the disc space are associated with Schmorl's nodes, namely disc space loss (which this patient had) and claw osteophytes (which were not present) [4].

In this patient the diagnosis of metastatic PTC was initially strongly considered based on the extremely high baseline Tg concentration. This was felt to be less definite based on the characterization by MRI, which was most consistent with a Schmorl's node. It was the slow but progressive increase in the serum Tg concentration that ultimately led to the repeat FDG PET/CT evaluation and to the diagnosis of metastatic PTC on biopsy. Giovanella et al. demonstrated the utility of serum thyroglobulin measurement in the decision to obtain an FDG-PET/CT in patients with negative sonographic and I-131 scintigraphic examinations [21]. In that study a serum Tg concentration greater than 5.5 ng/ml and/or a Tg doubling time of less than 1 year (without regard to the absolute serum value) was useful in predicting a true-positive FDG-PET/CT study.

Overall, the initial features of this L5 lesion favored the diagnosis of a Schmorl's node, despite the eventual diagnosis of metastatic disease. This case report highlights the

complexity associated with the diagnosis of metastatic disease and the need for careful assessment of multimodality imaging and laboratory findings when evaluating a complex case. This patient also represents a counterpoint to previous reports that describe false-positive FDG imaging in which a suspicious lesion was later determined to be a Schmorl's node [22, 23]. This case emphasizes the need for close follow-up when there is a high clinical suspicion of metastatic disease, even when the findings on imaging favor a benign diagnosis.

Conclusion

The assessment of suspected osseous metastatic thyroid cancer may be challenging. The imaging studies performed for this patient demonstrate that the appearance of Schmorl's nodes and metastatic disease may overlap. That is, a site of osseous PTC metastatic disease can have imaging characteristics that are indistinguishable from a Schmorl's node using current techniques. Additionally, this case reinforces the utility of thyroglobulin levels in predicting which patients are likely to have metastatic thyroid cancer at imaging.

Conflict of Interest Cory P. Daignault, Edwin L. Palmer, James A. Scott, John S. Swan and Gilbert H. Daniels declare that they have no conflict of interest.

Informed Consent All procedures followed were in accordance with the ethical standards of the responsible committee on human experimentation and with the Helsinki Declaration of 1975, as revised in 2000. This case report is a retrospective evaluation and did not require informed consent from the patient. All patient data and images have been de-identified. The investigation was approved by the hospital IRB.

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