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Epiglottic Calcification: The Unexplored Relationship with Increasing Rates of Renal Disease

To the Editor: In the September 2022 issue the Southwest Journal of Pulmonary Critical Care and Sleep published an interesting article authored by Punatar *et al.* detailing a case of severe dysphagia that was secondary to isolated epiglottic calcifications seen on computed tomography and flexible fiberoptic laryngoscopy (1). The epiglottis plays a critical role in the prevention of aspiration and coordination of swallowing.

Calcifications can severely alter its flexibility and limit oral intake. Isolated calcifications of the epiglottis and sequential dysphagia is not a common entity. With the increasing rates of chronic and end-stage renal disease, this letter is focused on increasing the awareness of extra-osseous manifestations of secondary hyperparathyroidism.

Secondary hyperparathyroidism is characterized by adaptive parathyroid gland hyperplasia to increase the production of parathyroid hormone. Etiologies include vitamin D deficiency and chronic kidney disease. Vitamin D deficiency can be seen in about 50% of the world population and chronic kidney disease affects more than 37 million people in the United States (2-3). Manifestations of sustained increases in parathyroid hormone can cause high-turnover rates of osseous structures and extra-osseous manifestations. Among the extra-osseous manifestations, chondrocalcinosis is a known complication. Although there remains a higher risk of calcification within the larynx and trachea as opposed to the epiglottis, epiglottic calcifications should still be suspected in patients with dysphagia and chronic kidney disease (4-5). Early diagnosis and management may result in the prevention of worsening extra-osseous disease.

Given the lack of literature regarding the prevalence and characteristics of secondary hyperparathyroidism and epiglottic calcifications, all cases that identify epiglottic calcifications should note whether a secondary hyperparathyroidism work up or other causes of abnormal parathyroid hormone elevations was initiated, to aid the scientific community in identifying a potentially underdiagnosed phenomenon. My question to the authors of this study is if any further work up was completed, including biochemical serum testing for parathyroid hormone, vitamin D levels, calcium, or phosphate levels. This would be valuable for the audience of this journal and would allow providers to be increasingly aware of this clinical entity.

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The Author Replies: Dr. Ibrahim reflects on our contribution of a case of severe dysphagia secondary to epiglottic calcification. It is purported that cases of epiglottic calcification are not common, and that a workup for secondary hyperparathyroidism should be considered. This is suggested as secondary hyperparathyroidism from chronic kidney disease may cause extra-osseous manifestations such as calcifications of the larynx and trachea, and even epiglottis.
To confirm and collectively create a comprehensive understanding of our case of epiglottic calcification, we agree with the notion by Dr. Ibrahim that secondary hyperparathyroidism should be considered in causes of epiglottic calcification.
Upon further review with lab values included below, our patient had progressed from chronic kidney disease to end stage renal disease and was consistent with home peritoneal dialysis. It had been noted that the patient was undergoing care and monitoring for secondary hyperparathyroidism in the outpatient setting from nephrology services. While he was on calcitriol .5mcg daily in the outpatient, setting, we have included his pertinent lab values as follows:

- Calcium 9.3 mg/Dl(8.6-10.3 mg/Dl)
- Corrected Calcium 10.1 mg/dL
- Albumin 3.0 g/dL (3.5-5.7 g/Dl)
- Phosphorus 3.6 mg/dL (2.5-4.5mg/dL)
- Vitamin D 25 0H 37.5 ng/ML (30-100 ng/ml)

While controlled, it is possible that secondary hyperparathyroidism a contributory factor in leading to this patient's epiglottic calcification and subsequent dysphagia.

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