Case Report

Multiple internal resorption in permanent teeth associated with hyperparathyroidism

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ABSTRACT

Internal resorption has been described as a resorptive defect of internal aspect of tooth. It is caused by transformation of normal pulp tissue into granulomatous tissue with giant cells which resorb dentin. Though mostly idiopathic in origin, trauma, caries and restorative procedures have also been suggested to be contributing factors of internal resorption of pulp. Most of the internal resorption cases were found on isolated individual tooth. There are few case reports on multiple root resorptions. External root resorption have been frequently associated with systemic disorders like renal diseases hyperparathyroidism, rickets etc. A rare association of multiple internal resorption of permanent teeth (including lateral incisors, canines, premolars, sparing 2nd and 3rd. Permanent molars and missing 35,36, 42) with elevated levels of PTH and uric acid was evident in a 28 year old female patient who presented with fracture of crowns of permanent teeth. Though many cases related to dental manifestations of chronic renal disease and internal resorption of permanent teeth have been documented in literature, there is no evidence on association of hyperparathyroidism with multiple internal resorptions of teeth.

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Internal resorption has been described as a resorptive defect of internal aspect of tooth following necrosis of odontoblasts as a result of chronic inflammation and bacterial invasion of pulpal tissue.[1] Internal resorption can be called intracanal resorption[2] and also canal root resorption.[3] It is considered as a rare case of resorption,[4,5] appearing as a typical dystrophy of the pulp which jeopardize the hard tissue of the teeth changing its normal morphology. It is caused by transformation of normal pulp tissue into granulomatous tissue with giant cells which resorb dentin. Trauma, caries and restorative procedures have been suggested to be contributing factors, but it also occurs as an idiopathic dystrophic change.[1]

Hyperparathyroidism (HPT) is a disease in which there may be a complex of biochemical, anatomic and clinical abnormalities resulting from increased secretion of parathyroid hormones (PTH). It may occur in primary, secondary and tertiary forms. Primary HPT results from autonomous hyperplasia or tumor, usually an adenoma. Secondary HPT develops in response to chronic low levels of calcium usually associated with chronic renal failure. Occasionally, parathyroid tumors develop after long standing secondary HPT, known as tertiary hyperparathyroidism.[6]

This case presents multiple internal resorptions in permanent dentition and associated findings of hyperparathyroidism based on various investigations.

CASE REPORT

A 28 year female patient reported to the Department of Oral medicine and Radiology with the chief complaint of mobility of the right lower back tooth 1 year ago. History revealed that her left lower back tooth had fractured one year back and was subsequently removed. Intraoral examination revealed grade 1 mobility in 45 with missing 35, 36, and 42. There was mild gingival inflammation in relation to lower anteriors [Figures 1 and 2].

Orthopantomogram (OPG) [Figure 3] revealed widened pulp chambers and root canals suggestive of internal resorption in 13, 14, 15, 22, 24, 25, 33, 34, 43, 44, and 45.

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Comprehensive blood investigations along with biochemical investigations were advised, which revealed an elevated serum uric acid level of 7.9 mg/dl and calcium level of 9.0 mg/dl. Hormonal assay was performed, revealing an elevated (PTH) level of 92.9 pg/ml. A repeat biochemical test after one year revealed PTH level elevated to 146.5 pg/ml and uric acid falling within range of 6.2 mg/dl and calcium level decreased to the level of 7.7 mg/dl. There was an increase in serum PTH level, suggestive of hyperparathyroidism.

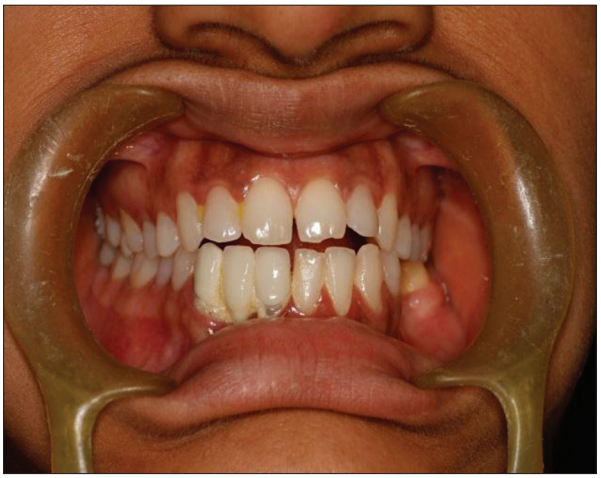

Figure 1: Intraoral image

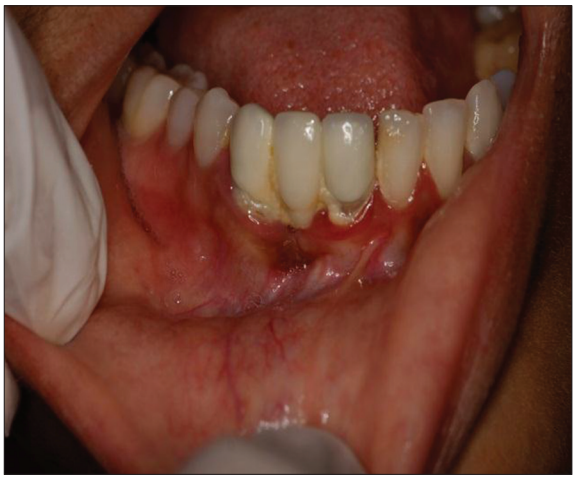

Figure 2: Mild gingival inflammation in relation to lower anteriors

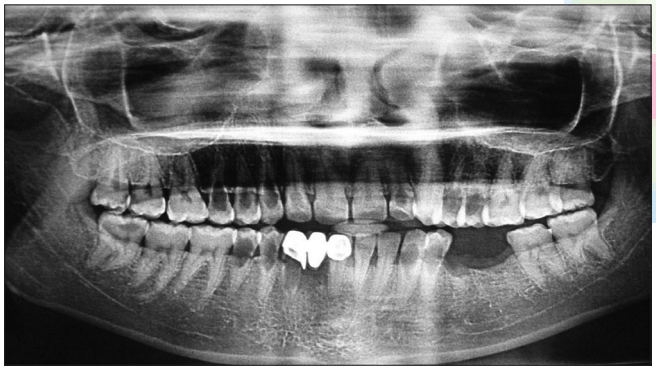

Figure 3: Orthopantomogram showing widening of pulp chamber and root canal

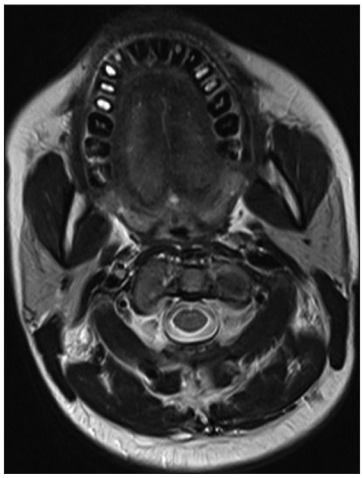

Figure 4: MRI (axial view) showing hyperintense areas within pulp chamber in T2 weighted images

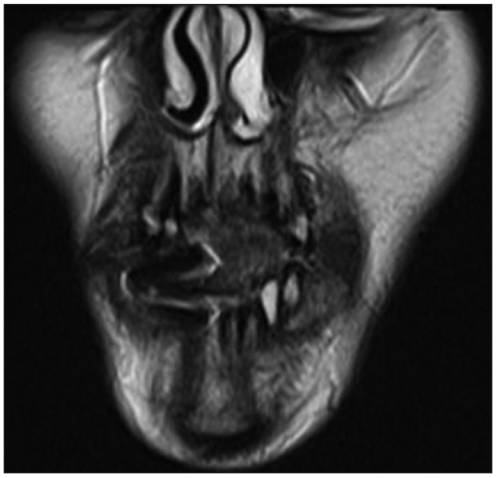

Figure 5: MRI (coronal view)

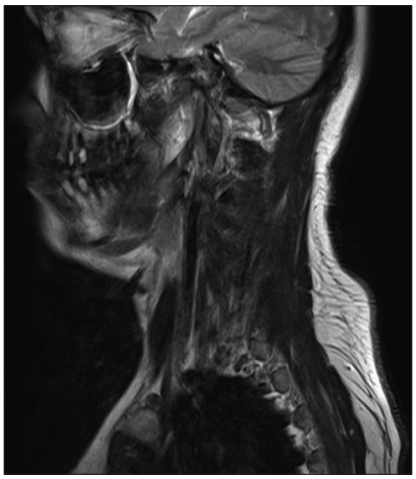

Figure 6: MRI (sagittal view) showing no evidense of adenoma in parathyroid gland and hyperintense areas within the pulp chambers

MRI [Figures 4-6] of jaw and parathyroid gland was performed which revealed no evidence of parathyroid adenoma. T_2 hyperintense areas were noted in multiple teeth with no periapical erosion/marrow edema.

The above mentioned investigations revealed hyper parathyroid state secondary in association with multiple internal resorptions of teeth.

DISCUSSION

Internal resorption is commonly termed to be idiopathic. Internal resorption is an unusual form of tooth resorption that begins centrally within the tooth, apparently initiated in most of the cases, by peculiar inflammatory hyperplasia of the pulp. The cause of pulpal inflammation and subsequent resorption of tooth substance is unknown.[7] Trauma and inflammation can be considered as possible causing factor in some cases. Resorption process can develop by shifting the pH to acidic for example in irreversible pulpitis so that enamel and dentin substances are dissolved by chelation. The untreated internal resorption can progress into external or vice versa which causes fracture of the tooth. In case of tooth trauma, intrapulpal hemorrhage can develop. Formed blood clots are then organized and replaced by granular tissue which compresses dentin wall of the pulpal chamber or root canal. With the activation of non-differentiated mesenchymalcells of the pulpal tissue they differentiate into dentinoclasts, the cells responsible for resorption of hard tooth structure.[8]

In majority of the cases the internal resorption usually is asymptomatic and detectable by routine radiographs.[9] Radiographically it is described as a radiolucent area characterized by an oval shaped enlargement of the root canal, showing many times the appearance of an ampoule[10] and which does not move with variation of radiographic angle. In more evolved cases, the fragility of the dental structure can cause areas of fracture or perforation. In this case report, the radiographic investigations revealed widened pulp chamber in OPG suggestive of internal resorption in multiple teeth. Blood chemistry of the patient showed elevated PTH level signifying hyperparathyroid state. Ultrasonographic investigation of abdomen showed a normal study and ruled out morphological alterations in kidneys inspite of marginal rise in uric acid levels. Subsequent MRI examination of jaws and parathyroid glands justifying increased vascular changes in the pulp chamber through T2 hyperintense areas in multiple teeth was performed.

Dental manifestations such as narrowing down of the pulp chamber, hypercementosis of root, bony changes including loss of laminadura, giant cell lesions of hyperparathyroidism, and bone demineralization have been reported by some studies, but multiple internal resorption associated with hyperparathyroidism has so far not been reported.[11]

Bartter FC *et al.* observed normocalceamic hyperparathyroidism without any radiologic, clinical or biochemical changes among their patients and stated that with the development of immunoassay techniques for measuring the levels of PTH, it has been shown that 50% of the detectable cases of primary hyperparathyroidism do not show radiologic, clinical or biochemical changes other than increased PTH levels.[12] Such cases are termed normocalcemic hyperparathyroidism. Radiographic changes in the bones are considered to develop and become apparent only in the more advanced stages of the disorder, changes in the jawbones are often late manifestations of radio graphically demonstrable bony disease.[13]

Rasmussen *et al.* observed among their patients that in secondary hyperparathyroidism there is an inverse relationship between the levels of serum PTH and serum calcium. The increased parathyroid activity and calcium mobilization are reflected not by an increase but by hypocalcaemia.[14]

In this case report, calcium level was decreased over the period of one year, but renal function test were normal. Investigatory picture revealed hyperparathyroid state was sustained over 1 year period, but renal function test were normal. Since there were no morphological changes in the parathyroid gland, a close periodic observation over a period of time is mandatory to see how hyperparathyroid state will progress. The present case report showed generalized resorption of permanent teeth with sustained elevation of PTH level. With no literature evidence to compare as this is the first case reported in the literature, future researches have to be performed to justify the genetic or pathological causes implicating the association of these manifestations. We could not perform radionuclide imaging as we could not obtain the patient's acceptance for the procedure. However, the radionuclide imaging is recommended to trace any possibility of occult lesions in parathyroid.

CONCLUSION

From this case report, we understand that multiple internal resorptions of permanent teeth and hyperparathyroidism can coexist together, henceforth biochemical investigation such as hormonal assays for PTH, serum alkaline phosphatase, and calcium levels along with renal profile should be performed as one of investigatory procedures in evaluating the cause of multiple internal resorptions of permanent teeth. We recommended more studies in this regard, to justify internal resorption as a definite manifestation of hyperparathyroidism.

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