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Case report

Bony affair–a rare case of osseous metaplasia in a native nonfunctioning kidney

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Abstract

Renal osseous metaplasia is a rare condition in a native kidney with end-stage renal disease, which presents mature bone with marrow elements in the background of chronic pyelonephritis. Osseous metaplasia has been reported in allograft specimens; however, the incidence thereof is extremely rare in a native kidney. Although the pathogenesis thereof is unknown, chronic ischemia and inflammation may be triggering factors. Osseous metaplasia has been previously reported in renal allograft specimens, but in this case report, we present a rare histological finding of osseous metaplasia in a native kidney.

Keywords: Marrow elements, native kidney, osseous metaplasia.

Heterotopic ossification is a pathological condition where there is bone formation in bone and soft tissue.[1] Osseous metaplasia is the pathological term used to describe heterotopic ossification. Renal osseous metaplasia is a rare pathology that is found in a kidney with a longstanding injury, which may be inflammatory or neoplastic.[1] In this case report, we discuss a case of osseous metaplasia along with myeloid metaplasia in a kidney with underlying chronic renal failure. The current instance is an extremely rare occurrence, and after a thorough literature review, this is the second reported case of osseous metaplasia in a native kidney[2], whereas the other reports are in the background of a neoplastic kidney or graft rejections.

Case report

A 48-year-old male patient with previously diagnosed diabetes presented with complaints of abdominal pain. He was evaluated and scheduled for a right nephrectomy of a nonfunctioning kidney. Computed tomography program of the right kidney revealed gross hydronephrosis with calyceal calcification, parenchymal thinning, and an indistinct calyceal system. No contrast was observed being excreted into the collecting system, and the left kidney was normal with subcentimetric cortical cysts. The serum creatinine level was mildly elevated, with normal blood urea nitrogen, and the complete blood count revealed neutrophilia. The patient underwent a right nonfunctioning kidney nephrectomy, which was sent for histopathological evaluation.

The kidney was shrunken with a loss of contour, measuring 7.5 × 5.5 × 3.5 cm with an attached ureter measuring 6 cm in length and 0.5 cm in diameter. The kidney cut sections revealed cystic spaces with a loss of the pelvicalyceal system, and the corticomedullary junction could not be discerned. One focus revealed a grey-white to grey-yellow gritty area, which was difficult to cut and subsequently underwent decalcification.

Microscopic examination revealed cystic changes with extensive sclerosis, hyalinization, and dense lymphoplasmacytic infiltrate. In addition, there was a paucity of glomeruli with tubules exhibiting atrophy and thyroidization **(Figure 1).** The decalcified sections showed areas of osseous metaplasia with lamellar bone formation combined with hematopoietic marrow elements that consisted of erythroid and myeloid precursors, megakaryocytes, and adipocytes **(Figure 2).** A graphical representation thereof has been included (**Figure 3**).

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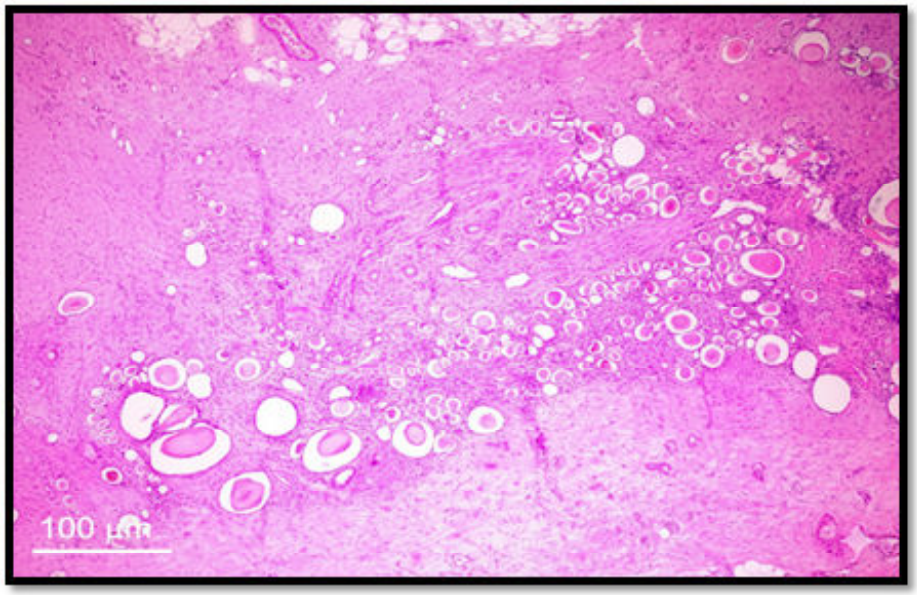

Figure 1. Haematoxylin and eosin staining: 200x showing thyroidisation of tubules and interstitial inflammatory infiltrate

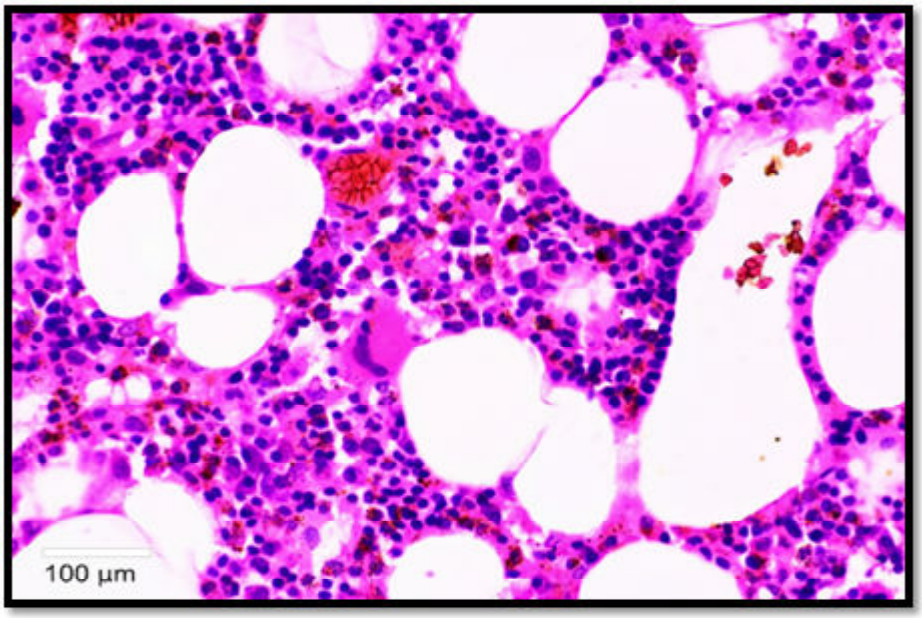

Figure 2. Haematoxylin and eosin staining: 400x showing fatty marrow elements along with megakaryocytes.

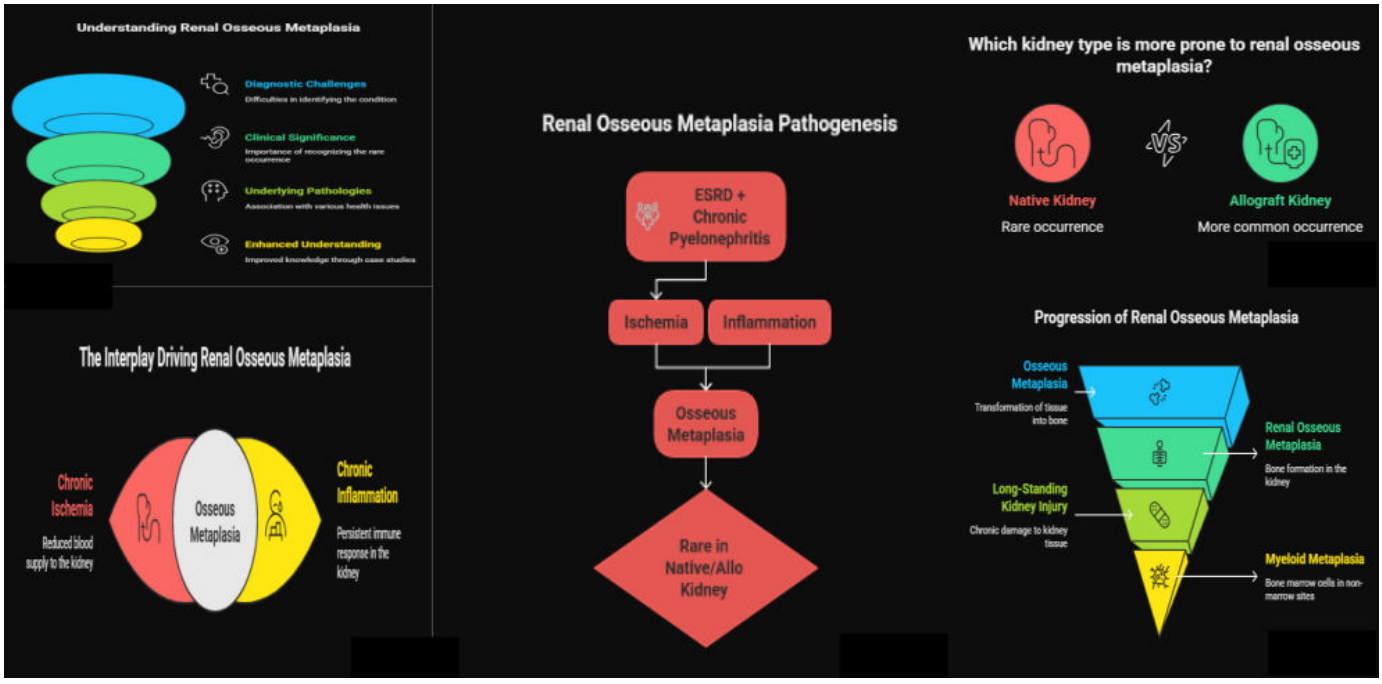

Figure 3. Overview of the clinical presentation and pathogenesis of osseous metaplasia in chronic kidney disease.

Discussion

Metaplasia is a term used wherein one tissue type is replaced by another.[3] Osseous metaplasia (OM) refers to the bony transformation in other solid organs or tissues. OM has been described in other sites, such as the uterus, parotid, and lung, in a non-transplant background.[3] In the kidney, OM has been observed in allograft specimens; however, the incidence thereof is extremely rare in the native kidney.

OM is often a secondary feature associated with chronic ischemia, inflammation, repeated injury, tumors, and hypercalcinosis.[4] The pathophysiology of OM in other sites is not clear; however, studies suggest that it arises because of the heterogeneous activation of pluripotent stem cells, followed by mineralization of the ossified matrix, and adipocytes and marrow elements occur as a later event.[4] Other studies indicate that fibroblasts play a key role in the pathogenesis, which are recruited into the renal tissue, accumulate, and thereby give rise to OM.[5] Ischemia and inflammation appear to be the favorable conditions for fibroblastic proliferation, such as in this case of end-stage renal disease, which may have triggered the formation of OM.[5] OM can sometimes be diagnosed as tumor calcinosis in radiology, thus posing a diagnostic dilemma. Calcifications may be a common finding in end-stage renal disease; however, OM with marrow elements is a rare finding.

The histology of OM in the kidney is similar to that of other organs, with mature bony trabeculae, adipocytes, and marrow elements.

Conclusion

OM is a rare event in solid organs and particularly rare in the kidneys. Although OM has been reported in allograft specimens, where the onset thereof is triggered by repeated injury, the incidence of OM with myeloid metaplasia in a native kidney with end-stage renal disease is an extremely rare occurrence. Radiology can mimic tumor calcinosis; therefore, histological evaluation is of particular importance. In this case, it was an incidental finding of bony trabeculae with marrow elements.

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Conflict of interest statement

Each of the authors has completed and ICMJE disclosure form. The authors declare that they do not have any potential or actual relationship, activity, or conflict of interest related to the content of this article.

Data sharing statement

Data generated or analyzed for the present report is included in this published article. Further details are available from the corresponding author on reasonable request after the deidentification of the patient whose data is included in the report.

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