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CORRECTION

Correction to: Identification of TIMPs signatures in Randall plaque from single-cell RNA sequencing (scRNA-Seq) analysis

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Correction to: Functional & Integrative Genomics (2024) 24:11
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The original article contains an error. Abstract has been removed during processing. This is now added here.

Abstract

Background Tissue inhibitors of metalloproteinases (TIMPs) are essential for controlling the dynamics of the extracellular matrix. Although their role in vascular diseases like as atherosclerosis and plaque development has been widely researched, the specific patterns of their expression and their functional significance inside Randall plaque tissues, a distinct vascular pathology linked to nephrolithiasis, have not been well investigated.

Objectives The objective of this work was to examine the expression of TIMP genes in Randall plaque tissues related to nephrolithiasis and to figure out the functional significance of these genes by analyzing their connection with gene ontology and pathways.

Methods We carefully extracted three Randall's plaque tissue samples and three normal renal papillae samples from the renal calyces utilizing BIGopsy Backloading Biopsy Forceps for our study. In Randall plaque tissues, scRNA-seq was used to study TIMP gene family expression dynamics. We isolated cells, extracted RNA, and used the 'Seurat' technology for high-throughput sequencing. 'Seurat' FindMarkers identified cluster-specific marker genes, and the 'clusterProfiler' R package examined gene ontology (GO) and Kyoto Encyclopaedia of Genes and Genomes (KEGG) pathways linked with differentially expressed genes (DEGs) to determine TIMP gene functions.

Results Clusters of renal papillae were revealed using robust principal component analysis (PCA) on 52,698 cells derived from tissue samples of individuals who had nephrectomy or calcium oxalate stones. There were five clusters with specific

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markers identified after evaluating 2,783 endothelial cells (ECs), and 21 subcategories were uncovered after analyzing 7,612 epithelial cells. With an average log fold change (avg_logFC) of 0.280932471, TIMP1 was highly expressed in 49.2% of cells in condition 1 and 32.3% of cells in condition 2. With an average log fold change (avg_logFC) of 0.257671699, TIMP2 was expressed in 74.1% of cells under condition 1 and 75.4% of cells under condition 2. Cluster 2 TIMP2 showed a clinically significant increase in log fold change, on average, of 0.283679548. An average log-fold change (avg_logFC) of 1.262202638 was observed for TIMP3-expressing cells in 90.2% of condition 1 and 70.3% of condition 2, respectively. The average log fold change (avg_logFC) for TIMP4 was 0.274234342 in 74.1% and 75.4% of cells, respectively, indicating a moderate effect.

Conclusions In conclusion, our study emphasizes the crucial roles of TIMP2 and TIMP3 in Randall plaque formation, shedding light on significant expression variations in nephrolithiasis. The observed low expression of TIMP1 and TIMP4 in Randall plaques suggests targeted interventions, presenting potential therapeutic avenues for renal stone management.

The original article has been corrected.