MEDS Basic Medicine (2026)
Clausius Scientific Press, Canada

DOI: 10.23977/medbm.2026.040116
ISSN 2616-2091 Vol. 4 Num. 1

Targeting the CTSS/CAPN6 axis: a multi-omics guided strategy for diagnosis and immunomodulatory therapy in recurrent implantation failure

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Keywords: Recurrent implantation failure; CTSS; CAPN6; Biomarkers; Machine learning; Immune infiltration

Abstract: Recurrent implantation failure (RIF) remains a major clinical challenge in assisted reproduction, largely owing to the lack of validated biomarkers and mechanism-based therapies. The present study aimed to identify key molecular drivers of RIF and to discover diagnostic biomarkers and therapeutic targets by integrating multi-omics bioinformatics with experimental validation. Three endometrial transcriptomic datasets (GSE103465, GSE111974, GSE188409) from RIF patients and fertile controls were analyzed using a streamlined workflow that comprised batch correction and differential expression analysis (n = 651), WGCNA identifying the RIF-associated turquoise module ($r = 0.43$, $P = 4 \times 10^{-4}$), Random Forest prioritization of CTSS and CAPN6, ROC-based diagnostic evaluation, and functional interpretation via GO/KEGG, ssGSEA, GSVA, and drug enrichment; in addition, CTSS and CAPN6 protein expression was validated by immunohistochemistry (IHC) in independent clinical endometrial samples. CTSS and CAPN6 were robustly identified as upregulated and functionally linked RIF drivers, and a two-gene model achieved an AUC of 0.835 (95% CI: 0.731–0.925), outperforming single markers. Both genes were implicated in lysosomal proteolysis, antigen processing, and immune modulation. Notably, the RIF endometrium exhibited elevated M0 macrophages and reduced monocytes/resting mast cells, with CAPN6 strongly correlated with M0 abundance ($P < 0.001$). Drug enrichment highlighted protease inhibitors, including boceprevir, as top-ranked candidates, and IHC confirmed significantly higher CTSS and CAPN6 protein levels in RIF versus controls (both $P < 0.001$). In conclusion, CTSS and CAPN6 serve as validated diagnostic biomarkers for RIF; their involvement in proteolytic–immune crosstalk, together with the association of CAPN6 with macrophage infiltration, supports targeted immunomodulatory strategies, while the potential repurposing of boceprevir highlights the translational value of the analytical framework.

1. Introduction

Recurrent implantation failure (RIF) is defined as repeated failure to achieve clinical pregnancy after ⩾3 IVF-ET cycles with high-quality embryos [1]. It affects ~17% of infertile individuals and causes significant psychological distress, repeated procedures, and clinical uncertainty [1–3]. Three key contributors are established: uterine cavity abnormalities (e.g., polyps), immune dysregulation, and impaired endometrial receptivity—the central determinant of embryo attachment and invasion [4–7]. Yet the molecular basis of receptivity failure remains unclear, and validated biomarkers for early diagnosis or personalized therapy are lacking [8,9].

Transcriptomic profiling of the human endometrium during the implantation window is still limited [10], and prior studies—largely focused on single genes or pathways—fail to capture the integrated gene-regulatory and immune microenvironmental networks governing receptivity [11,12]. Integrating transcriptomic data with WGCNA, machine learning, and immune deconvolution is therefore essential to identify robust hub biomarkers and advance precision interventions for RIF [12,13].

To address this, we integrated and normalized multiple endometrial transcriptomic datasets from RIF patients and controls. Differential expression analysis and WGCNA identified RIF-associated gene modules; Random Forest prioritized top candidate biomarkers. We assessed their diagnostic performance and functional roles via immune infiltration, pathway activity, and drug-sensitivity analyses. Finally, protein-level expression was validated by immunohistochemistry in independent clinical samples. This workflow bridges computational discovery and experimental validation to deliver actionable diagnostic signatures and therapeutic targets for RIF.

2. Materials and methods

2.1. Data acquisition and preprocessing

We retrieved three GEO datasets (GSE103465, GSE111974, GSE188409) using the keywords “recurrent implantation failure” and “RIF”. Each included endometrial samples from RIF patients and normal controls and was generated on a distinct microarray platform (Table 1). Raw data were preprocessed in R (v4.4.1) and Perl (v5.4.0): probe-to-gene annotation, expression matrix standardization, averaging of duplicate gene symbols, and $\log_2$ transformation of non-log-transformed data.

Table 1: Summary of the datasets used in this study.

Accession	Platform	Control (n)	RIF (n)	Total (n)	Type
GSE103465	GPL16043	3	3	6	Endometrium
GSE111974	GPL17077	24	24	48	Endometrium
GSE188409	GPL26963	5	5	10	Endometrium

Note: RIF, recurrent implantation failure. Control, normal healthy group.

2.2. Identification of differentially expressed genes (DEGs) and co-expression network analysis

The three preprocessed datasets were merged and batch-corrected using limma’s removeBatchEffect. Correction efficacy was confirmed by box plots and PCA. Differentially expressed genes (DEGs) between RIF and normal controls were identified with limma ($|\log_2 FC| > 0.585$, adj. $P < 0.05$) and visualized via volcano plots and hierarchical clustering heatmaps.

We applied WGCNA to identify RIF-associated modules. Low-expression (mean < -1) and low-variance (variance < 0.5) genes were excluded. A soft-threshold power (β) ensuring scale-free

topology ($R^2 > 0.9$) was selected using pickSoftThreshold. Modules were detected from the topological overlap matrix (TOM) using dynamic tree cutting (min. module size = 60, cut height = 0.25). The module most significantly correlated with RIF status (Pearson, $P < 0.05$) was designated the key module. Within it, genes with high gene significance (GS) and module membership (MM) were retained. RIF-associated hub genes were defined as the intersection of these key-module genes and the DEGs.

2.3. Functional enrichment and characteristic gene screening

Functional enrichment analysis (GO biological process, molecular function, cellular component; KEGG pathways) was performed on RIF-associated genes using clusterProfiler ($P < 0.05$). A random forest model was trained on these genes to identify characteristic markers; the optimal number of trees was determined by 10-fold cross-validation, and genes with importance scores > 4 were selected. Their chromosomal positions were visualized with circlize.

2.4. Diagnostic model construction and in silico validation

Diagnostic performance of the characteristic genes was assessed by ROC analysis (pROC, glmnet); AUC > 0.7 was considered significant. A predictive nomogram was built using rms and rmda.

2.5. Comprehensive in silico analyses of characteristic genes

Downstream analyses of the characteristic genes included: (1) PPI network construction using GENEMANIA (integrating co-expression, genetic interactions, pathways, and physical interactions); functional annotations with FDR < 0.05 retained; (2) Pathway activity via GSVA (GSVA, GSEABase), comparing high- vs. low-expression groups; top 5 enriched pathways ($P < 0.05$, 1000 permutations) reported; (3) Immune infiltration estimated by ssGSEA for 28 immune cell types in PBMCs; Spearman correlations computed between core genes and immune cells, and among immune cells; (4) Drug enrichment analysis using clusterProfiler ($P < 0.05$) to predict candidate therapeutics.

2.6. IHC validation

Endometrial tissues from RIF patients and controls were collected at the Affiliated Hospital of Guilin Medical University (ethics approval: 2025KJJLL-15). Protein expression of two characteristic genes—CTSS and CAPN6—was validated by IHC. After deparaffinization, rehydration, and antigen retrieval, sections were incubated with primary antibodies (CTSS: Proteintech #27538-1-AP, 1:300; CAPN6: Proteintech #10120-1-AP, 1:500), HRP-conjugated secondary antibody (ABclonal #AS014, 1:500), and DAB substrate (ZSGB-BIO #ZLI-9018). Hematoxylin counterstaining was applied. Images were acquired using a Leica light microscope and quantified in Fiji (ImageJ).

2.7. Statistical analysis

All bioinformatics analyses were performed in R (v4.4.1). Intergroup differences for continuous variables were compared using Student's t-test. A P-value (or adjusted P-value/FDR where specified) of less than 0.05 was considered statistically significant.

3. Results

3.1. Identification of RIF-associated genes and functional characterization

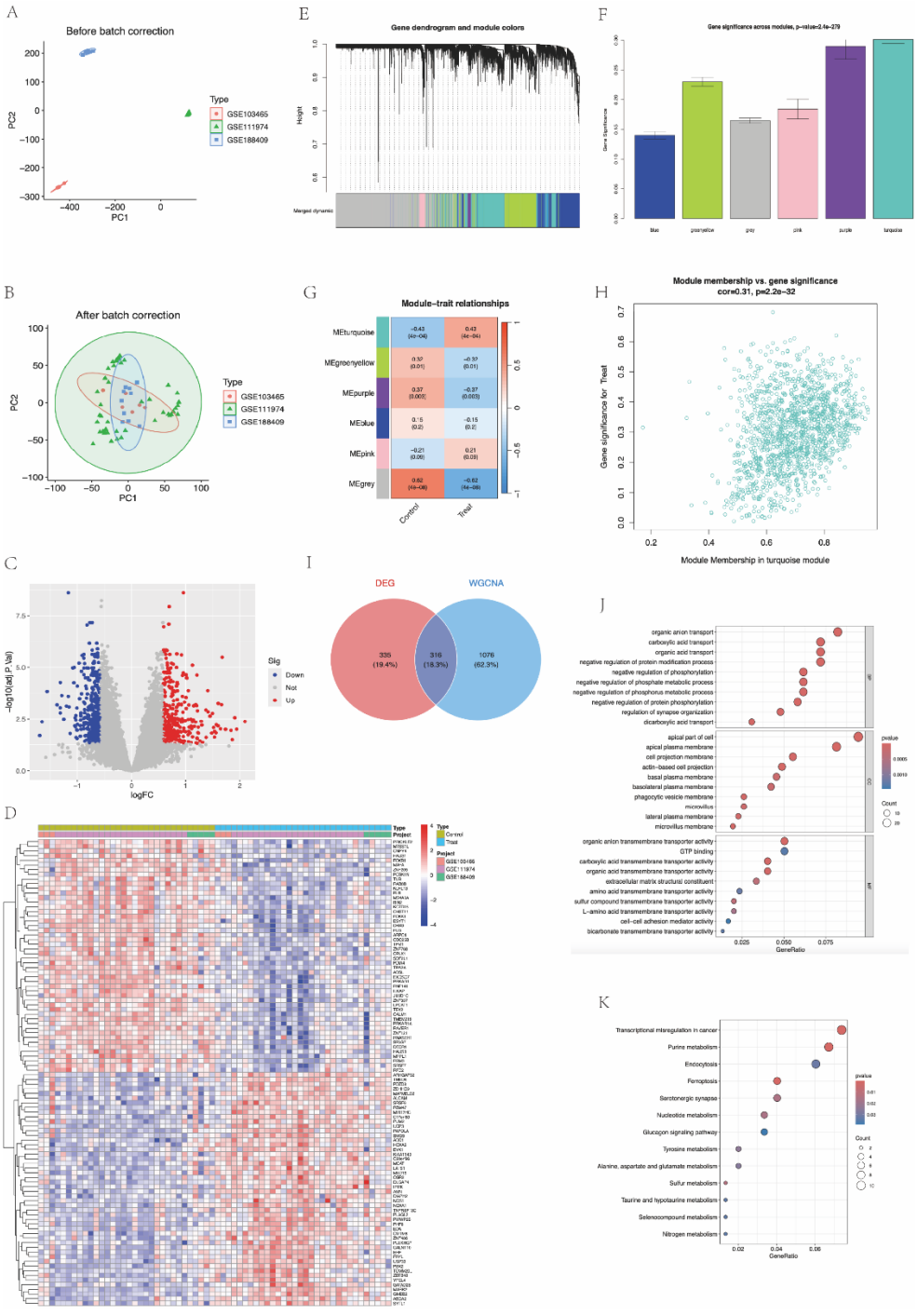

Figure 1: Identification and functional characterization of RIF-associated genes. (A, B) PCA before and after batch correction. (C) Volcano plot of DEGs in RIF. (D) Heatmap of top 50 DEGs. (E–G) WGCNA-derived co-expression modules. (H) Module–trait correlation: turquoise module most strongly associated with RIF. (I) Venn diagram showing intersection yielding 316 high-confidence RIF-associated genes. (J, K) GO and KEGG enrichment of these 316 genes.

PCA confirmed effective batch correction: post-correction samples clustered by biological group (RIF vs. control) (Fig. 1A, B). Differential expression analysis identified 651 DEGs—299 downregulated and 352 upregulated in RIF (Fig. 1C). Unsupervised hierarchical clustering of the top 50 DEGs cleanly separated RIF from control samples (Fig. 1D). WGCNA detected six gene modules (Fig. 1E–G). The turquoise module (1,076 genes) showed the strongest positive correlation with RIF status ($r = 0.43$, $P = 4 \times 10^{-4}$) (Fig. 1G, H). Its intersection with the 651 DEGs yielded 316 high-confidence RIF-associated genes (Fig. 1I). Functional enrichment of these 316 genes ($P < 0.05$) highlighted roles in organic anion transport regulation; apical plasma membrane and actin-based projections; organic acid transmembrane transporter activity; and KEGG pathways—including purine metabolism, ferroptosis, and transcriptional misregulation in cancer (Fig. 1J, K).

3.2. Screening of characteristic genes and evaluation of their diagnostic value

A random forest model prioritized candidate biomarkers among the 316 RIF-associated genes, identifying CTSS and CAPN6 as top-ranked characteristic genes by importance score (Fig. 2A, B).

Both genes showed significantly higher mRNA expression in RIF versus controls ($P < 0.001$) (Fig. 2C). ROC analysis revealed strong discriminatory power for each: CTSS (AUC = 0.802; 95% CI: 0.686 – 0.896; $P < 0.001$) and CAPN6 (AUC = 0.744; 95% CI: 0.622 – 0.862; $P < 0.001$) (Fig. 2D). Their combination in a logistic regression model improved performance (AUC = 0.835; 95% CI: 0.731 – 0.925; $P < 0.001$) (Fig. 2E). Decision curve analysis showed CTSS conferred the highest net clinical benefit across most threshold probabilities (Fig. 2F).

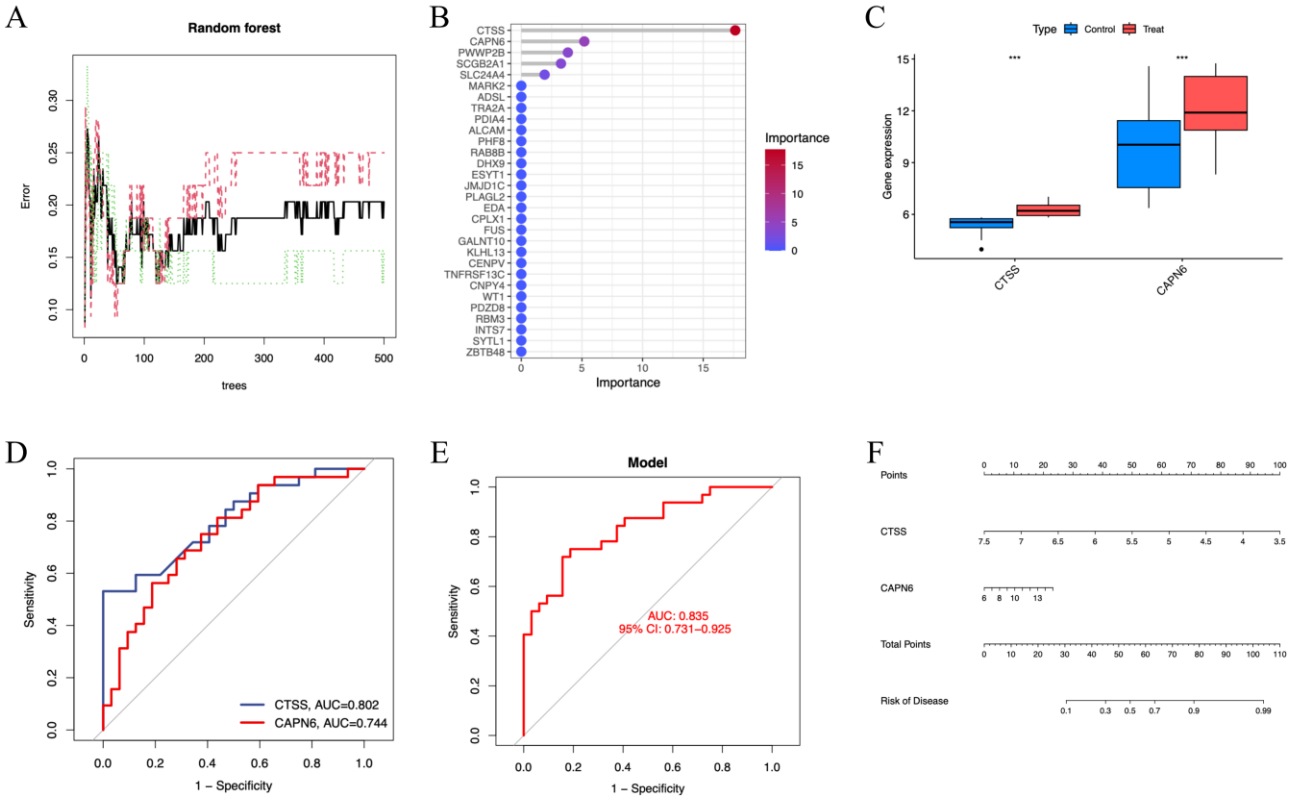

Figure 2: Screening and diagnostic evaluation of characteristic genes. (A-B) Random Forest model identifies CTSS and CAPN6 as core characteristic genes. (C) mRNA expression of CTSS and CAPN6 is significantly upregulated in RIF. (D-E) ROC curves of CTSS, CAPN6, and the combined model. (F) Decision curve analysis of the net clinical benefit.

3.3. Comprehensive bioinformatic analysis of CTSS and CAPN6

The PPI network of characteristic genes and their interactors comprised 20 nodes: CTSS linked to antigen processing/presentation and endolysosomal activity; CAPN6 to calcium-dependent cysteine peptidase activity (Fig. 3A).

GSVA revealed gene-specific pathway associations: high CAPN6 expression correlated with PPAR signaling and glycolysis/gluconeogenesis; low CTSS expression correlated with steroid hormone biosynthesis and ABC transporters (Fig. 3B, C). ssGSEA showed altered immune infiltration in RIF endometrium—increased M0 macrophages and decreased monocytes and resting mast cells versus controls (Fig. 3D). CAPN6 expression positively correlated with M0 macrophages but negatively with monocytes and resting mast cells (Fig. 3E), suggesting immunomodulatory potential. Drug enrichment identified boceprevir, telaprevir, and valsartan as top candidates targeting CTSS and CAPN6; CTSS was the primary target across all significant terms (Fig. 3F, G).

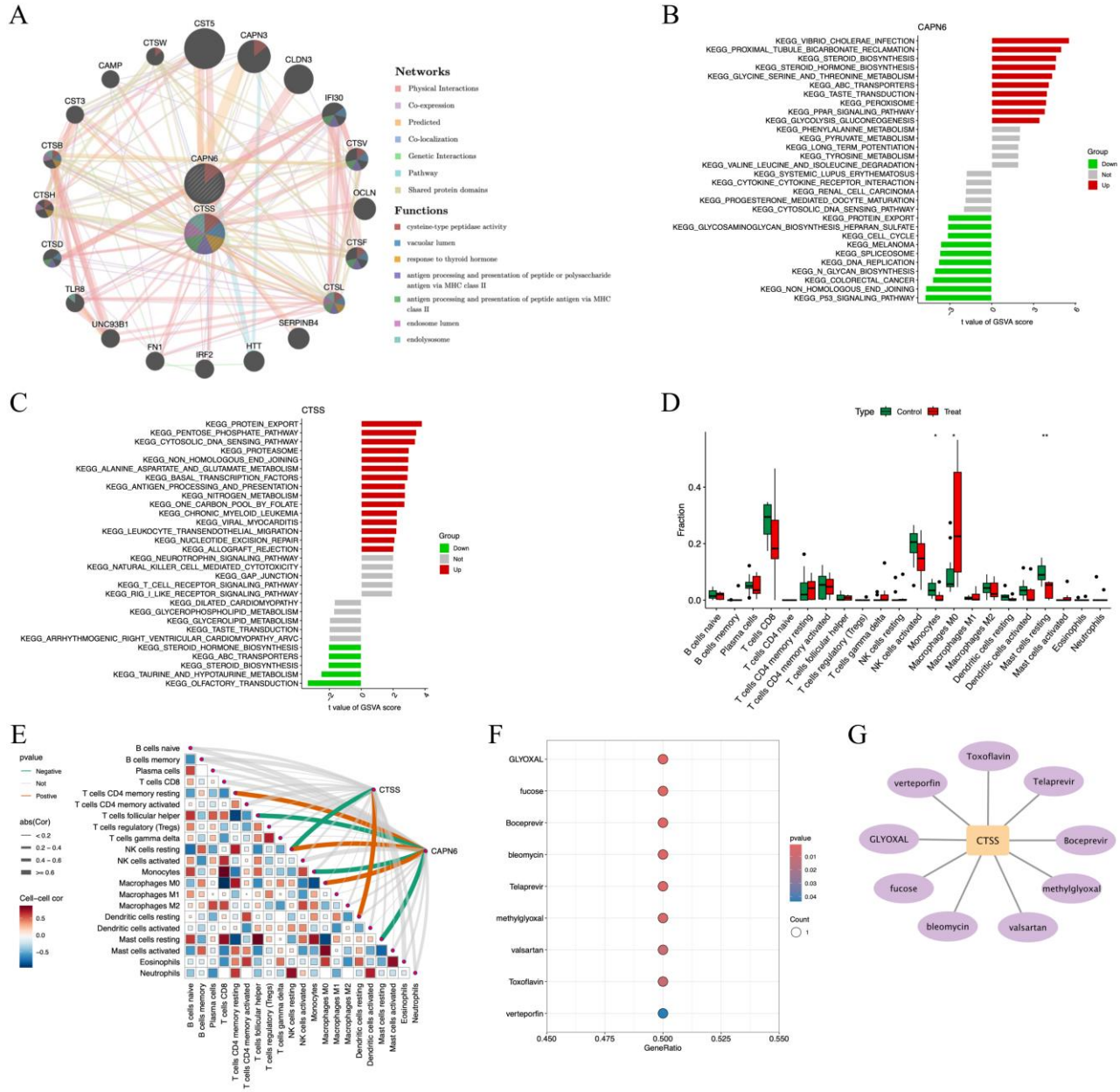

Figure 3: Comprehensive bioinformatic analysis of CTSS and CAPN6. (A) Protein-protein

interaction network. (B-C) GSVA of pathway activities. (D) Altered immune microenvironment in RIF endometrium. (E) Correlation between characteristic genes and immune cells. (F-G) Prediction of potential targeting drugs.

3.4. Experimental validation of protein expression

IHC validated CTSS and CAPN6 protein expression in independent clinical endometrial samples. Both showed significantly higher expression in RIF versus controls ($P < 0.001$) (Fig. 4A–D). Positive staining area was 58.2% ± 4.1% for CTSS and 31.8% ± 3.5% for CAPN6 in RIF tissues. All assays were performed in triplicate with high reproducibility (CV < 5%).

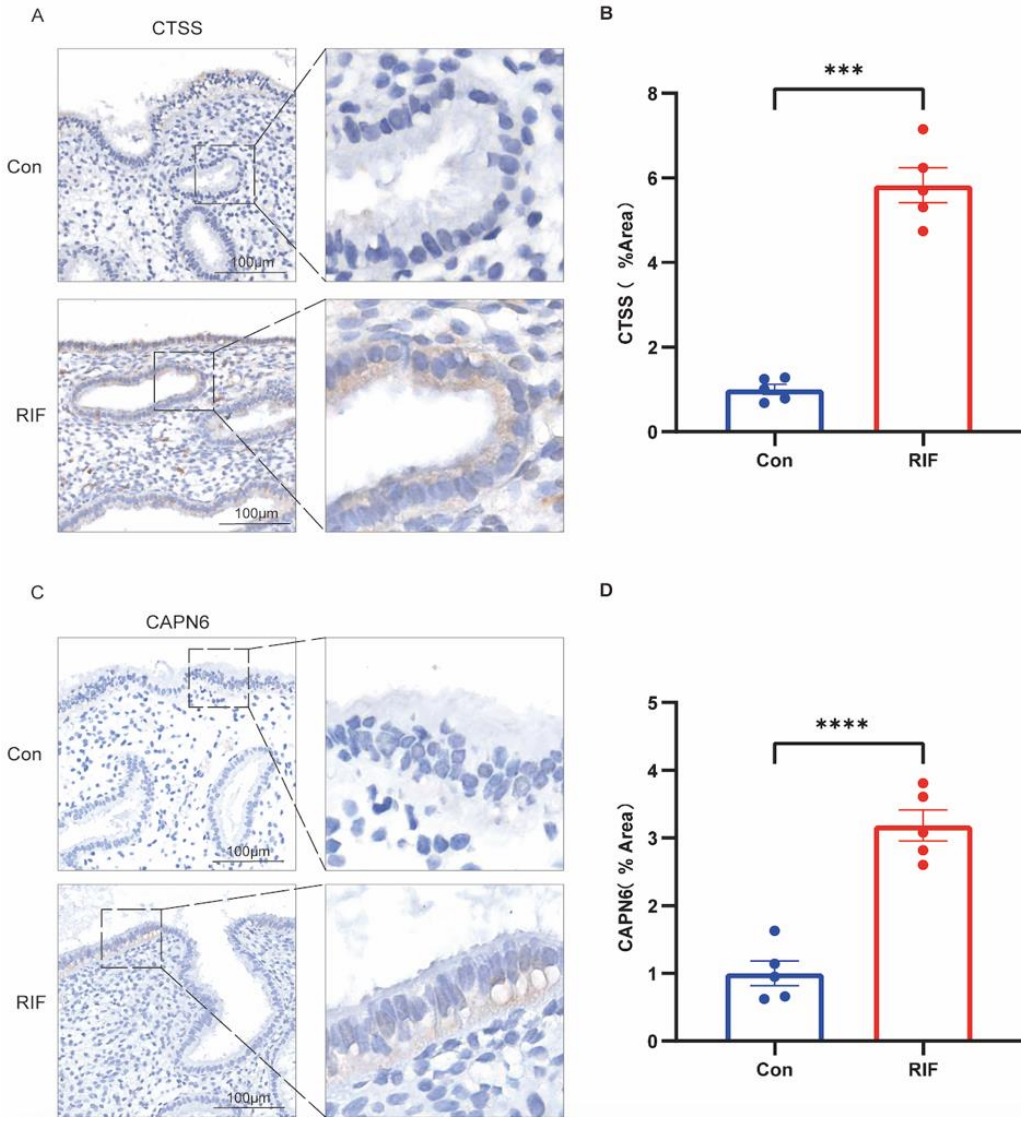

Figure 4: Experimental validation of CTSS and CAPN6 protein expression by IHC. (A-D) Representative IHC images of CTSS and CAPN6 in control and RIF endometrial tissues (400×). (E-F) Quantitative analysis confirms significant upregulation of both proteins in the RIF group.

4. Discussion

In this study, we identified CTSS and CAPN6 as key RIF-associated genes, which may play pivotal roles in RIF pathogenesis through various cellular processes, including immune response, cell

adhesion, and proteolysis. Our results suggest that CTSS and CAPN6 may be potential biomarkers for predicting RIF in women undergoing in vitro fertilization and provide a perspective on the molecular mechanisms underlying RIF, promising avenues for future research and treatment.

Although the pathogenesis of RIF is not fully elucidated, immunologic disturbances [14], embryo adhesion [15,16], and proteolysis [17] have been proposed to be involved. We identified 651 DEGs in RIF-endometrial samples, and the top 50 were primarily involved in immune responses, including the regulation of immune activation and cytokine production. Significantly, two of them, CTSS and CAPN6, were determined as key genes associated with RIF. CTSS, a lysosomal cysteine protease, regulates the activation of immune cells such as T cells, macrophages, dendritic cells, and neutrophils, and has been implicated in immune-related diseases, including Sjögren's syndrome, cancer, and infectious diseases [18]. CAPN6, a calpain protease, regulates cell adhesion, invasion, and migration, which are essential for implantation and placentation [19,20]. Dysregulation of CTSS and CAPN6 may contribute to RIF by disrupting these crucial processes, making them promising biomarkers. This is supported by ROC analysis: AUCs of CTSS and CAPN6 were 0.802 and 0.744, respectively, and their combination improved prediction (AUC = 0.835). Similarly, using GSVA and machine learning, eight RIF-characteristic genes (SRD5A1, POLR3E, PPA2, PAPSS1, PRUNE, CA12, PDE6D, and RBKS) were identified in endometrium tissues [21]. Their individual AUCs ranged from 0.614 to 0.765, and the combined AUC was 0.902, indicating that bioinformatic tools are powerful for biomarker discovery.

No protein can fulfill its biological functions in isolation; rather, protein–protein interactions and networks govern cellular processes and determine phenotypes. In this study, CTSS and CAPN6 were found to interact with proteins involved in immune response, cell adhesion, and proteolysis. CTSS-related networks regulate immune cell activation, such as CD4+ and CD8+ T cells, indicating a role for CTSS in modulating immune responses. CAPN6 networks are integral to cell adhesion and migration, involving proteins such as integrin beta 1 and focal adhesion kinase, suggesting a role for CAPN6 in regulating cell–cell interactions. These findings reinforce that CTSS and CAPN6 regulate immune cell activation and cell adhesion [18-20], and dysregulation may contribute to RIF [15-17,22]. The identified protein–protein interactions provide a foundation for further research into RIF mechanisms and therapeutic strategies.

A typical pathological feature of RIF is immunologic disturbances, characterized by unbalanced immune cell infiltrations in diseased endometrium [8,23,24]. A recent transcriptomic and single-cell sequencing study from the GEO database revealed increased M0 macrophages and decreased γδT cells, M2 macrophages, and dendritic cells in RIF samples, with diagnostic genes PDIA4 and PGBD5 correlated with suppression of B cells and T cells, respectively [12]. Our immunoinfiltration analysis corroborated these findings, showing increased M0 macrophages and decreased monocytes and mast cells in RIF endometria. These results again suggest a role for immune dysregulation in RIF pathogenesis. CTSS and CAPN6 may contribute to this dysregulation, especially by mediating the compositions of resting NK cells, M0 macrophages, monocytes, and resting mast cells. Targeting immune dysregulation may be a promising treatment. Drug enrichment analysis identified glyoxal, fucose, boceprevir, bleomycin, telaprevir, methylglyoxal, valsartan, toxoflavin, and fumonisin B1. Among them, fucose has been reported to affect core fucosylation in the immune system [25,26]; boceprevir and telaprevir are protease inhibitors for Hepatitis C virus [27]; bleomycin is involved in pulmonary fibrosis and associated with CAPN6[28,29]; and methylglyoxal inhibits inflammatory cell accumulation and cytokine production in autoimmune encephalomyelitis[30]. These findings indicate the importance of immune-focused considerations when developing RIF therapies.

There are several strengths to our study, including the use of a large dataset and multiple bioinformatic approaches. However, limitations include reliance on bioinformatic analysis; findings should be validated in larger, multi-center cohorts, and the therapeutic potential of CTSS and CAPN6

should be explored. Further research is needed to clarify their roles in RIF mechanisms and identify targeted therapeutic agents.

5. Conclusion

This study identifies CTSS and CAPN6 as novel biomarkers and therapeutic targets for recurrent implantation failure (RIF), driven by their roles in immune dysregulation and impaired endometrial receptivity. Experimental validation confirms these associations and clarifies their pathogenic mechanisms. Although clinical translation requires further validation and functional studies, the findings provide a strong basis for targeting CTSS and CAPN6 in RIF therapy.

Acknowledgements

Transcriptomic data were downloaded from the Gene Expression Omnibus (GEO). Endometrial tissues for validation were collected at the Affiliated Hospital of Guilin Medical University under ethics approval (No. 2025KJJLL-15). We thank the GEO contributors, patients, and staff of the Reproductive Medicine Center.

The present study was supported in part by the grant from Guangxi Key Laboratory of Molecular Medicine in Liver Injury and Repair (No. GXLIRMMKL-K202404).

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