**Abstract**

The epithelial-to-mesenchymal transition may play a role in adenomyosis. GRIM19 expression is downregulated in adenomyotic lesions, and the effects of this downregulation in adenomyosis remain relatively unclear. In this study, we aimed to explore whether aberrant GRIM19 expression is associated with the epithelial-to-mesenchymal transition in adenomyosis and found that the expression of both GRIM19 and WT1 was low, and epithelial-to-mesenchymal transition, which included significant changes in CDH1, CDH2 and KRT8 expression, occurred in adenomyotic lesions, as confirmed by Western blotting and quantitative real-time PCR. We provided novel insights into WT1 expression in adenomyosis, revealing that WT1 expression was increased in the endometrial glands of adenomyotic lesions by immunohistochemistry. In vitro, knockdown of GRIM19 expression by small interfering RNA (siRNA) promoted the proliferation, migration and invasion of Ishikawa cells, as measured by Cell Counting Kit-8, wound healing assay and Transwell assays. Western blotting and quantitative real-time PCR confirmed that WT1 expression increased and epithelial-to-mesenchymal transition was induced, including the upregulation of CDH2 and downregulation of CDH1 and KRT8after transfecting the GRIM19 siRNA to Ishikawa cells. Furthermore, WT1 expression was upregulated and epithelial-to-mesenchymal transition was observed, including downregulation of CDH1 and KRT8in GRIM19 gene-knockdown mice. Upregulation of Wt1 expression in the endometrial glands of Grim19 knockdown mice was also verified by immunohistochemistry. Taken together, these results reveal that low expression of GRIM19 in adenomyosis may upregulate WT1 expression and induce epithelial-to-mesenchymal transition in the endometria, providing new insights into the pathogenesis of adenomyosis.