**Abstract**

**Introduction:** Endometriosis is a risk factor for low-grade serous, clear cell, and endometroid ovarian carcinoma. In both endometriosis and ovarian carcinoma, immunological factors are associated with clinical outcome. Chronic inflammation in endometriosis may be linked to tumorigenesis, but exact processes contributing to endometriosis-associated ovarian carcinoma remain unknown. This review aims to describe potential immunological factors involved in the malignant transformation of endometriosis into ovarian carcinoma.

**Methods:** PubMed and Embase were searched from inception up to October 2020 for studies comparing immunological processes in endometriosis and endometriosis-associated ovarian carcinoma.

**Results:** Detailed analysis of immune components in the malignant transformation of endometriosis into endometriosis-associated ovarian carcinoma is lacking. Altered levels of chemokines and cytokines as IL-6, IL-8, IL-10, and TNF-α are reported and the function, number and polarization of NK cells, dendritic cells, and monocytes differ between endometriosis and associated ovarian carcinoma compared to healthy tissue. In addition, altered inflammasome and complement systems, indicate a role for the immune system in the carcinogenesis of endometriosis.

**Conclusion:** Chronic inflammation in endometriosis may potentially drive inflammation-induced carcinogenesis in endometriosis-associated ovarian carcinoma. Exact immunological pathways and cellular processes remain unknown and require more thorough investigation.