A high proportion of women with advanced epithelial ovarian cancer (EOC) experience weakness and cachexia. This relationship is associated with increased morbidity and mortality. EOC is the most lethal gynecological cancer, yet no preclinical cachexia model has demonstrated the combined hallmark features of metastasis (spreading of cancer), ascites development, muscle loss and weakness in adult immunocompetent mice. With growing awareness that cancer itself can induce cachexia even in the absence of toxic cancer therapies, it is imperative to develop pre-clinical models for each type of cancer cachexia that captures critical phenotypic hallmarks of this disease in humans. In this study, we established a new mouse model that demonstrates the concurrent development of cachexia and metastasis that occurs in women with EOC. The model provides physiologically relevant advantages of inducing tumour development within the ovarian bursa in immunocompetent adult mice. Moreover, the model reveals that muscle weakness in both TA and diaphragm precedes severe metastasis while weakness also precedes atrophy in the TA. An underlying mitochondrial bioenergetic stress corresponded with this early weakness. Collectively, these discoveries highlight that muscle weakness can occur earlier during cancer than previously discussed. In this way, the findings can direct new research towards the development of therapies that target pre-atrophy and pre-severe-metastatic weakness during EOC in addition to therapies targeting cachexia.