Study finds biomarker may boost ovarian cancer chemotherapy response

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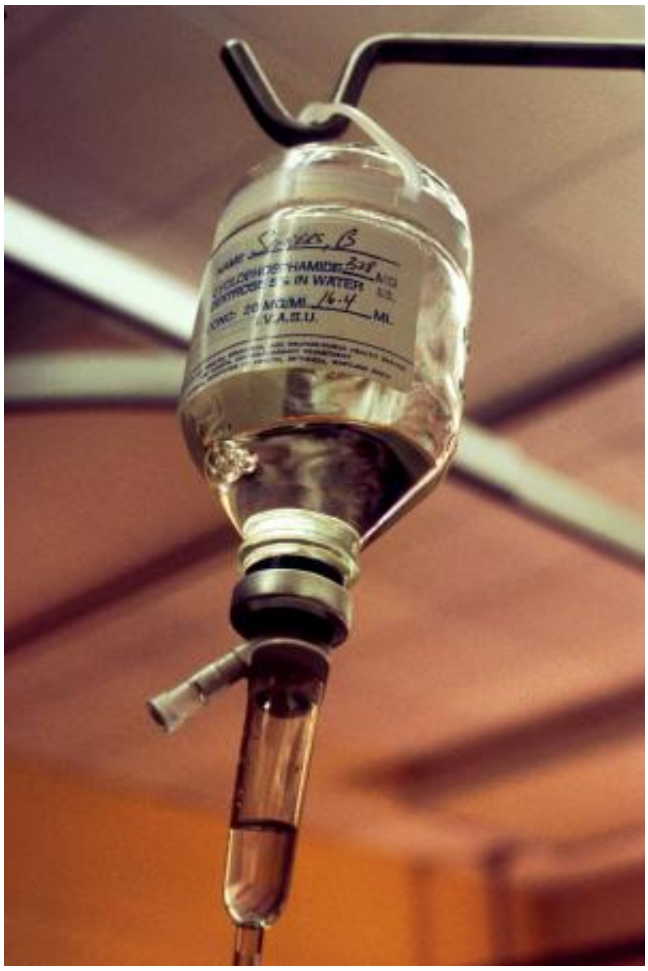

Shown is a close-up of an intravenous (IV) bottle. Credit: Linda Bartlett/public domain

A molecule that helps control gene expression may play a role in controlling chemotherapy resistance among patients with the most common form of ovarian cancer.

A study at The University of Texas MD Anderson Cancer Center has identified miR-506, a non-coding "micro" RNA molecule, as a likely robust clinical marker for epithelial ovarian cancer chemotherapy, and as a potential therapy due to its ability to sensitize cancer cells to chemotherapy. Epithelial ovarian cancer accounts for approximately 90 percent of all ovarian cancers. Ovarian cancer is the leading cause of death from gynecologic cancers in the U.S.

"MiR-506 was associated with better response to therapy and longer progression-free and overall survival," said Wei Zhang, Ph.D., professor of Pathology.

Zhang's study, which included data from The Cancer Genome Atlas and other independent clinical populations as well as study of mouse models, observed "statistically significant improved responses" to the chemotherapy drugs cisplatin and olaparib when miR-506 was added to the treatment. Standard treatments include surgery and platinum-based chemotherapy, and the five-year survival rates for patients with advanced ovarian cancer is 30 to 40 percent.

Chemoresistance is a major challenge in cancer treatment and this study may provide a means to overcome resistance, said Zhang. His team's findings are published online in the July issue of the *Journal of the National Cancer Institute*.

"Our previous study found that miR-506 is a potent inhibitor of a process known as epithelial-to-mesenchymal transition (EMT), which is also associated with chemoresistance," he said. "This study provides further insight into this molecule's role in augmenting chemotherapy responses by directly affecting the DNA repair process used by cancer cells to counter DNA damages caused by chemotherapy."

Prior investigations by Zhang's group observed that miR-506 suppressed cancer cell EMT. The current study showed that miR-506 also regulated RAD51, a protein involved in DNA repair that contributes to chemotherapy resistance when overexpressed.