SERUM P53 ANTIBODY DOES NOT PREDICT CHEMORESISTANCE IN METASTATIC COLORECTAL CANCER TREATED WITH FOLFOX (XELOX) PLUS BEVACIZUMAB AT 1ST LINE CHEMOTHERAPY

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Background: P53 gene is one of the famous tumor suppressor genes. Some studies showed accumulation of p53 protein and p53 gene mutation had close relation with serum p53 antibody. The effect of p53 mutation on chemosensitivity in colorectal cancer is a controversial issue. Thus serum p53 antibody could theoretically predict chemoresistance in mCRC treated with FOLFOX (XELOX) plus Bevacizumab at 1st line chemotherapy. This study aimed to evaluate the predictive significance of serum p53 antibody in mCRC patients who were treated with FOLFOX (XELOX) plus Bevacizumab.

Methods: Ninety patients treated with FOLFOX (XELOX) plus Bevacizumab were enrolled, and seventy-seven patients whose KRAS gene status was revealing at the beginning of treatment. Before the first administration serum p53 antibody level was quantified by enzyme-linked immunosorbent assay utilizing MESAQCUP™ anti-p53 test kits. Cutoff value for positivity was 1.3 U/ml. KRAS genotyping using tumor samples was analyzed by Luminex® assay. We investigated whether anti-tumor effect of the treatment was relative with serum p53 antibody and KRAS gene status.

Results: There was no significant difference in clinical baselines between two groups. The positive rate of serum p53 antibody was 40.0% (36/90). KRAS gene mutation was detected in 40.2% (31/77). Both KRAS gene mutation and positive rate of serum p53 antibody were detected in 13.3% (12/90). Overall response rates of RECIST criteria were 74.4% (67/90) in total, 77.7% (42/54) in serum p53 antibody negative and 69.4% (25/36) in serum p53 antibody positive. Median progression free survivals were 17.9 months and 16.7 months (no significant statistic difference $P = 0.738$), respectively.

Conclusion: Serum p53 antibody did not predict chemoresistance in mCRC treated with FOLFOX (XELOX) plus Bevacizumab at 1st line chemotherapy.