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Successful Treatment with Targeted Therapy of Metastatic Refractory Pediatric ETV6-NTRK3 Fusion-Positive Secretory Breast Carcinoma

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P-glycoprotein (P-gp) expression/efflux activity in cytoplasmatic membrane can induced obstacles in the effective treatment of cancer by chemotherapy. Moreover, independently on its efflux activity, this protein can secure the resistance also to non-P-gp substrates and through affecting of several cell´s regulatory processes (1). In previous paper, we have described a reduced sensitivity of P-gp positive variants of L1210 cells (R & T) to endoplasmic reticulum (ER) stressors thapsigargin and tunicamycin (Tun) as compared with P-gp negative parental L1210 cells (S). Here we have studied the mechanism of P-gp positive cell resistance against N-glycosylation inhibitor Tun. We found that Tun at 0.1 µM induces an increase in expression of ER stress markers CHOP and spliced variant of XBP1 in S cells but not in R & T cells. Higher expressions of chaperones Hsp70 and Grp78/Bip were observed in R &T than in S cells. Transfection of S cells with the plasmid encoding Grp78/Bip, resulted in reduction of CHOP expression after ER stress inducted with Tun. Therefore we suppose, that overexpression of Grp78/Bip is responsible for less response of R & T to ER stressor Tun. To understand this feature in details requires further investigation.

Keywords: MDR, ER stress, Grp78/Bip.

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