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LETTER TO THE EDITOR

Leukemia and rosiglitazone

The article titled "Can an oral antidiabetic (rosiglitazone) be of benefit in leukemia treatment?" written by Cetinkalp et al. and published in one of the recent issues of your journal was quite interesting (Cetinkalp et al., 2015). Here, we would like to emphasize some points.

Pan et al. (2012) showed that, metformin and rosiglitazone enhanced daunorubicin-induced apoptosis. Rosiglitazone increased etoposide-induced and vincristine-induced apoptosis. In this study, results suggest that use of insulin to control hyperglycemia in acute lymphoblastic leukemia patients may contribute to anthracycline chemoresistance, while metformin and thiazolidinediones may improve chemosensitivity to anthracycline.

In another study it was concluded that rosiglitazone induced apoptosis in K562 leukemia cells in vitro, and that rosiglitazone-induced apoptosis in K562 leukemia cells was highly correlated with activation of caspase-3, decreasing telomerase activity, down-regulation of the anti-apoptotic protein Bcl-2, and up-regulation of the pro-apoptotic protein Bax (Liu et al., 2009).

In our previous study, while atorvastatin and rosiglitazone did not affect the expression of CD38 and the level of bcl-2, these drugs significantly increased the level of Annexin V (chronic lymphocytic leukemia patients-CLL-) when compared with control group ($p < 0.001$). Both drugs significantly decreased the expressions of CD5 ($p = 0.03$) and ZAP-70 ($p < 0.05$) compared with control group. Atorvastatin and rosiglitazone increased apoptosis in lymphocytes of CLL in vitro (Yavasoglu et al., 2013).

In conclusion, in the light of these studies, rosiglitazone can provide additional support in the classical treatment of both acute and chronic leukemia.

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