Reactive stress in kg1a cells. our findings

Reactive Oxygen Species (ROS) and oxidative stress with a subsequent increase in apoptotic cells (after 48 and 72h), the activities of enzymes were decreased.

Furthermore, 4 -HBTC induces an increase in cellular GSH levels at earlier stages (24 h), whereas, GSH oxidation and apoptotic cell death occur at 48 and 72h. According to these findings, 4 -HBTC may be an appropriate candidate for Acute myeloid leukemia treatment by reducing the activity of antioxidant enzymes and increase in oxidative stress levels. In our study, 4 -HBTC induced apoptosis via increasing intracellular ROS production and induction of oxidative stress.

The induction of apoptosis is known to be an appropriate strategy for therapy of cancer16. We used notable methods for confirming the occurrence of apoptosis in the KG1a cells by 4 -HBTC. The assay of 4 -HBTC treated KG1a cells by fluorescence microscopy demonstrated the signs of apoptosis (Fig. 2).

Translocation of phosphatidyl serine (PS) to the external side of the plasma membrane of cells is also a hallmark of apoptosis. Thus, the Annexin V/PI double staining method was used. The result presented in Fig.

3B indicated a time-dependent increase in the population of apoptotic cells. Moreover, because of the close relation between cell cycle and apoptosis, cell cycle of the KG1a treated-cells after 24 -72 hr was investigated. The flow cytometry analysis showed that reduction in proliferation was associated with sub- G1 phase arrest (Fig. 3A). The results showed that an increase in

the number of the cells in the sub-G1 phase indicating of the apoptotic event (Fig. 3A).

Based on these data, 4 -HBTC possesses remarkable antitumor activity through regulation of ROS and free radicals generation and can be proposed as impressive factors for more evaluations in future. Conclusion our study indicated that compound of 4 –HBTC exhibits an anti-proliferative effect on KG1a human leukemia stem-like cells. Herein, we evaluated apoptosis induction activity of 4 –HBTC depends on oxidative stress in KG1a cells. Our findings suggest that this compound can be considered as a great candidate for pharmaceutical evaluations. Annexin V/PI double staining method was and fluorescent microscopy demonstrated that 4 -HBTC induces apoptosis in KG1a cells in a time-dependent manner.

4 -HBTC enhanced cellular ROS and MDA levels and also decreased total thiol and the activities of CAT and SOD.