Amifostine exerts anti-angiogenic activity and suppresses vascular endothelial growth factor secreted by hemopoietic stem/progenitor cells.

Akel SM¹, Atoum MF, Saleh SA, Awadallah SM.

Author information

• ¹Department of Medical Laboratory Sciences, Faculty of Allied Health Sciences, Hashemite University, Zarka, Jordan. sakel@hu.edu.jo

Abstract

OBJECTIVE:

To assess, in vitro, the effect of Amifostine (AMF, WR-2721) on angiogenesis and levels of vascular endothelial growth factor (VEGF) secreted from hemopoietic stem/progenitor cell populations.

METHODS:

We conducted the study in the research laboratories of the Hashemite University, Jordan between September 2003 and January 2005 where we took samples were from Myelodysplastic syndrome (MDS) patients and healthy donors attending Al-Hussein Cancer Center and We determined the proliferation of human umbilical vein endothelial cells (HUVECs) in cultures supplemented with media conditioned with AMF-treated and AMF-untreated pure hemopoietic cells [CD34+ cells, and erythroid, myeloid and megakaryocytic progenitors]. Furthermore, in the same conditioned media, we evaluated levels of elaborated VEGF by a sensitive enzyme linked immunosorbent assay.

RESULTS:

Biologically, media conditioned with AMF-treated cells reduced proliferation of HUVECs compared to media conditioned with untreated control cells (p<0.05). In cultures of AMF-untreated cells, elaboration of VEGF was higher (p<0.05) in media conditioned with cells from MDS patients compared to healthy donors. A 30 minutes pre-exposure of cells to AMF (500 mM) suppressed levels of VEGF secreted within 24 hours in 63 of 89 evaluated cultures. The percentage of reduction of VEGF in AMF-sensitive cultures was comparable in cultures of MDS cells (18%, 2-37%; median, range) and normal cells (12%, 2-45%).

CONCLUSION:

The results showed that AMF exerts an anti-angiogenic activity and suppresses the secretion of VEGF in hemopoietic stem/progenitor cells obtained from both healthy individuals and patients with MDS.

PMID: 16228049