Kaposi’s sarcoma (KS) is a richly vascular systemic neoplasm of endothelial origin which can be associated with AIDS. Cutaneous and/or mucosal lesions are painful when submitted to friction or pressure.

Folkman and Haudenschild [1] showed that the capillary endothelial cell expresses the information necessary to build a capillary tube provided that sufficient stimulation of the cell by neighboring angiogenic factors is produced. Angiogenic factors may be critical in the onset of KS lesions as well [2]. HIV aggression of certain key cells (macrophages, lymphocytes) may induce angiogenic factor release.

Although heparin is bound to endothelial cells and attracts growth factors to promote angiogenic-dependent cell proliferation [3], in the presence of corticosteroids, it acts to inhibit angiogenesis. Dimethyl sulfoxide (DMSO) has the ability to cross biological membranes carrying with it substances unable to penetrate the membranes spontaneously [4]. Moreover, DMSO has been shown to reduce pain when applied to the skin locally [4] and may inhibit HIV production in vitro by altering viral assembly [5]. We tested the potential ability of DMSO to transport low molecular weight heparin and corticosteroids directly into KS lesions.

Eight male AIDS patients (stage 4-D, CDC classification), 23-43 years old, were treated after full consent had been obtained. KS lesions measured 4-12 mm in diameter. During the 5- to 10-month trial period, only azidothymidine (AZT), antibiotics or anti-parasitics were given as medically required. The preparation consisted of: 1 ml of 90% DMSO, 1 ml of 23 mg of hydrocortisone acetate in aqueous solution (in a water base of benzyl alcohol, carboxymethylcellulose sodium, sodium chloride, polysorbate 80; Roussel Labs, Paris, France) and 0.3 ml of 7.300 AXa.IC units of fraxiparin (Fraxiparin, Laboratoires Choay, Paris, France). One unit of antifactor Xa.IC is the amount of gly-cosaminoglycan that produces the same inhibiting effect as 1 IU of heparin. Each dose is increased 6 fold for daily application. Patients were instructed to liberally apply the preparation on painful skin lesions 2-3 times a day using a sterile gauze pad. No rebound or side effects were noted during or after the treatment period. The following positive results were obtained after 8 weeks of treatment: all patients reported that the pain disappeared and the size of the lesions remained either static or regressed to a smaller size than prior to treatment. Normally, KS lesions will increase in size without treatment. Measurement of lesion sizes prior to and during treat-
ment confirmed that growth arrest or actual lesion shrinkage occurred. Two weeks after treatment had been discontinued, patients reported return of pain and lesion size increase at a rate slower or the same as prior to treatment. We conclude that topical use of DMSO, heparin and corticosteroids is beneficial in blocking KS-associated pain and slowing or arresting growth of these lesions in 8 male AIDS patients.

References

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