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Oral echinacea for prevention of relapses of molluscum

contagiosum

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TEXT

We read with great interest the article by Dattola et al. published just a few weeks ago on this journal.¹ We wish to present the results of a multicenter, randomized, sponsor-free study on the efficacy and tolerability of an oral product containing *Echinacea angustifolia* (200 mg; titration of 4% of echinacoside) and *E. purpurea* (200 mg; titration of 4% of polyphenols) (Macrocea^R) for prevention of relapses of molluscum contagiosum (MC) in children.

Children previously affected by MC and successfully treated by curettage, were divided in two groups. The first group was treated with echinacea [1/2+1/2 sachet/ day (=200+200 mg/day) for two months in children with a weight <20 kg; 1+1 sachets/day (=400+400 mg/day) for two months in children with a weight >20 kg]. The second group was not treated. All children were examined at the beginning of the trial, one month later and at the end of the study. Follow up duration was six weeks.

The group treated with echinacea was made up of 36 evaluable children [24 males and 12 females, with an age ranging from 4 to 10 years (mean age: 6.3 years)]. The control group was made up of 38 evaluable patients [24 males and 14 females, with an age ranging from 4 to 11 years (mean age: 7.2 years)].

In the group treated with echinacea 14 relapses (38.9%) were observed. In the group which was not treated relapses were 26 (68.4%). By means of X² test, the difference of percentage of relapses was statistically significant (p <0.05). Two patients (2.7%) reported mild nausea, but it was unnecessary to stop the therapy.

Several approaches were suggested for the treatment of MC: cantharidine, salicylic acid, potassium hydroxide, sodium nitrite, povidone iodine, benzoyl peroxide, imiquimod, cidofovir, cimetidine, curettage, cryotherapy and laser. As far as imiquimod is concerned, it

stimulates the synthesis of cytokines by T helper 1 (Th1) lymphocytes, that are inhibited by MC virus. The latter produces chemokines analogous to epidermal growth factor, and viral interleukin-18-binding protein (vIL-18BP), that binds interleukin (IL)-18 and inhibits IL-18-induced interferon (IF)- γ synthesis. vIL-18BP inhibits the Th1 panel of cytokines, that are essential for cytotoxic T lymphocytes and natural killer proliferation.² We recently published our successful experience with a gel containing 1.8% hydrogen peroxide.³ However, according to the last Cochrane review, no single intervention showed to be convincingly effective.⁴

Echinacea spp. is a plant belonging to the *Compositae* family. Flowers, roots and seeds of *Echinacea* spp. are used in phytotherapy. *Echinacea* spp. contains several molecules; however, it is possible that the most important compound is arabinogalactan, a polysaccharide obtained by *E. purpurea*, with a molecular weight of 75.000. It was observed that arabinogalactan is effective in activating macrophages against tumor cells and protozoans, such as *Leishmania enriettii*. Arabinogalactan induced macrophages to produce tumor necrosis factor- α (TNF- α), IL-1 and IF- β -2. However, it did not activate B-cells and did not induce T-cells to produce IL-2 and IF- β , but it induced a slight increase in T-cell proliferation. *E. purpurea* was effective in activating peritoneal macrophages isolated from animals after administration of cyclophosphamide or cyclosporin. Macrophages treated with *E. purpurea* showed increased synthesis of TNF- α and enhanced cytotoxicity against tumor target WEHI 164 as well as against *L. enrietti*. After a cyclophosphamide-mediated reduction of leukocytes in the peripheral blood, arabinogalactan induced an earlier influx of neutrophils as compared to controls. *E. purpurea* treatment of immunosuppressed mice with cyclophosphamide or cyclosporin restored their resistance against lethal infections with the predominantly macrophage-dependent *Listeria monocytogenes* and predominantly neutrophil-dependent *Candida albicans*.⁵ Finally, in aging mice, *E. purpurea* increased NK

cell numbers in bone marrow and spleen. This increase in NK cell numbers was paralleled by an increase in their antitumor and cytolytic capacity.⁵

To our knowledge, literature data about the use of oral echinacea in prevention of relapses of MC are missing. The results of our study (as previously mentioned, it is multicenter, sponsor-free and based on a very large group of patients) must be confirmed. However, our observations would support the usefulness and safety of oral echinacea for prevention of relapses of MC. However, a careful medical history is necessary: as previously mentioned, echinacea belongs to the *Compositae* family.

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NOTES

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Supplementary Digital Material

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