VIEWPOINT

We advocate and prescribe certain therapeutic modality which is not always based on scientific basis. Therapeutic modality may be a point of debate. We have attempted to discuss in this section such topics of clinical relevance. We will put forward both points in favor and against the therapeutic approach in question and leave readers to make their conclusions. Comments and suggestions regarding this section should be addressed to Dr Deepak A Parikh, 1, Milan, Dixit Road, Vile Parle (East), Bombay - 400057.

Editor

CORTICOSTEROIDS : USE IN HERPES ZOSTER

Herpes zoster (HZ) is an acute viral infection involving particular dermatome. The aim of treatment in HZ is to

i) Limit the extent, duration and severity of the disease in the primary dermatome.

ii) Prevent disease eleswhere, and

iii) Prevent post-herpetic neuralgia, (PHZN).

First two goals can be achieved by limiting varicella-zoster viral replication using antiviral agents. Post-herpetic neuralgia once established is refractory to treatment. The possibility that post-herpetic neuralgia may be caused by inflammation, necrosis and subsequent scarring of the sensory ganglion provided the rational for use of corticosteroids.

Englstein et al[1] reported favourable results with use of corticosteroids early in the course of disease. They gave triamcinolone 8 mg, 2 tid for 7 days, 1 tid for 7 days, next 1 BD for 7 days and then stopped. They had also used lactose capsules on control group. They had not used acyclovir. Englstein et al concluded that in healthy patients more than 50 years old with painful zoster, the early use of oral corticosteroids significantly shortened the duration of post-herpetic neuralgia. Keezkes et al[2] compared role of corticosteroid and carbamazepine in prevention of post-herpetic neuralgia. They divided 40 healthy patients 50 years or older into two groups, one receiving carbamazepine 100 mg qid. The other group received 40 mg prednisolone tapered over 4 weeks. They did not observe any dissemination of the disease. Three in corticosteroid group and 13 in carbamazepine group developed post-herpetic neuralgia (PHZN). Neuralgia lasted only upto 6 months in corticosteroid group compared to 2 years in carbamazepine group.

Inflammation in sensory ganglion is initiated by VZV infection. Thus use of antiviral agent, acyclovir early in course of disease may help in minimizing inflammation and necrosis. Esmann et al[3] carried out randomised double-blind controlled study on 78 patients, 60 years and above. All patients were given 800 mg acyclovir 5 times a day for 7 days. Patients were devided into two groups of which one received prednisolone 40 mg/day gradually tapered and stopped over next 2 weeks. Nine patients in each group developed PHZN. Authors concluded that prednisolone had no effect on the development or the rate of disappearance of post-herpetic neuralgia.

Wood et al[4] conducted a double-blind, controlled trial in patients with acute HZ to determine whether either 21 days of acyclovir therapy or the addition of prednisolone offered

any improvement over 7 days of acyclovir therapy. Patients with a HZ of less than 72 hours duration were assigned to receive acyclovir (600 mg orally, five times daily) for 7 days with either prednisolone or placebo, or acyclovir for 21 days with either prednisolone or placebo. Prednisolone was given at a dose of 40 mg day and tapered over a 3 week period. They did not find any significant difference between any of the groups. However, they observed that steroid recipients had more adverse events. They concluded that acyclovir for 21 days or addition of steroid did not reduce the frequency of PHZN.

A contrary view has been expressed that suggests systemic steroid therapy might increase the severity of the eruption or lead to dissemination.[5] Miller[6] suggests that dissemination of the disease is possible only in those patients who are immunocompromised or on steroid prior to the onset of HZ. He recommends use of corticosteroid in healthy patients early in HZ.

References

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