

Effect of thiamine on defective chemotaxis of polymorphonuclear granulocytes

A preliminary report

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Chemotaxis of polymorphonuclear leukocytes (PMNs) plays an important role in protection of the organism against infection. A decrease in chemotactic capacity or a disturbance of chemotaxis has been demonstrated in the background of numerous body surface infections including purulent or mycotic processes of the skin and recurrent upper respiratory tract catarrhs.

Theron et al [4] have recently observed that thiamine stimulated PMN motility. On this basis we have treated two patients with chronic defective PMN chemotaxis. We were successful in normalizing the disturbed PMN chemotaxis and in moderating the clinical symptoms as well. In response to an i. m. dose of 5 mg/kg/day of thiamine for five days, PMN chemotaxis was restored

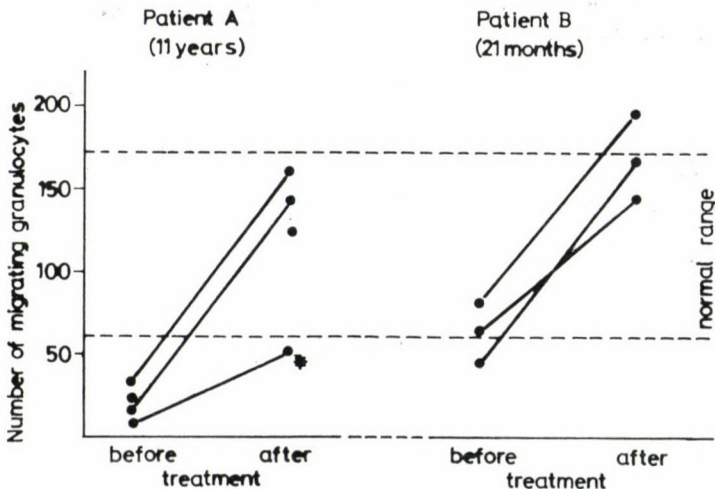


FIG. 1. Effect of thiamine (5 mg/kg/day for 5 days) on defective PMN chemotaxis. Chemotaxis was expressed as the mean number of granulocytes migrating across the filter; examinations in 5 different areas per visual field thiamine 1.0 mg/kg/day

in both patients (Fig 1). After treatment, the parents reported a catarrh-free 8–10 week period with cessation of the earlier dermal mycosis, stomatitis and paronychia. About 9 weeks after treatment the earlier complaints began progressively to return. Repeated treatment was also successful.

In an in vitro system the addition of 10^{-3} molar thiamine restored the motility of PMNs whose chemotaxis had been blocked with horseradish peroxidase. During the phagocytic activity of the cells, the coupling of leukoattractant and the PMNs, for instance, induced superoxide and H_2O_2 formation and degranulation with myeloperoxidase release [2]. This is an important regulatory mechanism in the process because H_2O_2 and the superoxide radicals cause reversible damage to the cell membrane and decrease the motility [1]. Thiamine is able to bind these aggressive radicals and exerts a scavenger effect on the cell membrane.

In our patients the defective chemotaxis was probably caused by the altered activity of the peroxidase (H_2O_2)-halide system of phagocytes.

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