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EFFECT OF BROMELAIN ON CORONARY HEART DISEASES AND ANGINIA PECTORIS

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INTRODUCTION

Since 1972, we have been treating cancer patients with high dosages of oral bromelain, as adjunctive therapy, generally between 400-1000mg per day. This treatment intends to expose the surface antigens of malignant cells (deshielding) and to mobilize lymphocytes and macrophages. Bromelain is available in Germany today as a supportive cancer medicine and in the last few years we have treated over 600 cancer patients, most of them as outpatients (Gerard 1972, Nieper 1976, Goldstein et al. 1975).

ANGINA PECTORIS AND BROMELAIN

Among these were 14 patients with angina pectoris. These were relapsing anginas, four of them with infarct-anamnesis. Right from the start of the bromelain treatment, the angina pectoris disappeared in all patients. Only four of them are still under hypertensive therapy, combined with Mg-orotate (Nieper 1974). Otherwise, no coronary therapy is required. The latency period between start of high dosage bromelain therapy and disappearance of angina pectoris is between 4 to 90 days. It seems to increase with the severity of coronary sclerosis.

With four additional non-cancer patients, high bromelain dosages yielded also dramatic therapeutic results. We have been using bromelain also for several years continuously for protective therapy of thrombosis and thrombophlebitis with definite success.

It is known that bromelain has a potent inhibitory action on blood platelet aggregation. It possesses, also, fibrinolytic activity. One mg of bromelain in serum is equivalent to about 4000 units of streptokinase (Taussig, et al. 1975).

MECHANISM HYPOTHESIZED

It is unlikely that the long-term effect of bromelain on coronary diseases is connected with dissolving of coronary thrombi. Also, the prevention of cell wall connected platelet aggregation by bromelain does not seem relevant. The most likely explanation to me is that the fibrinous possibly also organized plaques are eliminated to a large extent by bromelain. The long latency periods plead for this hypothesis.

In connection with this view, I would like to refer to the basic work of Roberts (1976). According to him, fibrinous deposits are the basis for development of plaques. These cannot only narrow the lumen but prevent flow of nutrients.

1) "Bromelain 200"-Nadrol

Recent Publications (Benda, et al, 1976) could be also interpreted by this fibrinolytic hypothesis: continuous streptokinase treatment has a positive effect upon angina pectoris, otherwise resistant to therapy.

Bromelain therapy seems more efficient, because it can be applied orally and, therefore, more frequently, it is also less expensive.

SUMMARY

Although the above are only limited retrospective observations, without controlled coronary diagnoses, I find them very important and feel they deserve further investigation.

REFERENCES

Benda, L., Kloesel, W., Redtenbacher, L., Spiess, A. and Zenz, W., Wien. Med Wschr., 126:1, 1976.

Gerard, G., Rev. Agressologie, 13:4, 261-274, 1972.

Goldstein, N., Taussig, S., Gallup, J. and Koto, V., Hawaii Med. Journal., 34:3, 91-94, 1975.

Nieper, H. A., Krebsgeschehen, 8:1, 9-15, 1976.

Nieper, H. A., Zschr. Praejkub. Geriatrie, 2:200-204, 1974.

Roberts, W.C. Proc. Europ. Cong. Cardiol., 68, 1976.

Taussig, S., Yokoyama, M., Chinen, A., Inari, K., Yomakido, M. and Nishimoto, Y., Hiroshjima H. Med. Sci., 24-2, 185-193, 1975.

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