

Zinc deficiency in senile purpura

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SUMMARY Fasting plasma zinc concentrations were lower in elderly people with senile purpura than in a control group matched for age. No significant difference was found in the mean serum concentration of albumin, which is the main binder of zinc. No other clinical or laboratory findings differentiated the two groups.

As the cause of the low plasma zinc values has not been found it is suggested that further studies of the related factors including input, output, and binding should be made before a therapeutic trial is launched.

Senile purpura is a well known and apparently benign but unsightly condition affecting the elderly.¹ It is quite common, but the aetiology remains obscure: theories include loose supportive tissue and damage to the small blood vessels as possible factors in its production followed by abnormal macrophage function, which delays resolution.²

Several studies have suggested that zinc is an important element in the proportion of rapid and sound tissue repair.³⁻⁸ This study was undertaken to measure plasma zinc concentrations in patients with senile purpura and to compare them with a control group.

Patients and methods

Forty inpatients from the geriatric unit were studied. Their ages ranged from 65 to 99 years. They were divided into two groups each comprising eight men and 12 women. Group A comprised 20 patients without purpura (mean age 82 years) and group B 20 patients with senile purpura (mean age 86 years). Senile purpura was defined by the presence of four or more ecchymoses at least 1 cm in diameter on the extensor surface of the hand or forearm.^{9,10} There were no significant differences in either the diagnoses or the treatment of the two groups. All had normal full blood counts, including platelets, and the Hess test yielded normal results. No other causes for purpura had been found. The patients had no pressure sores or other disorders of the skin.

Both groups of patients fasted overnight before venous samples were taken. The blood was anticoagulated with lithium heparin and centrifuged

without delay at room temperature. Plasma samples were stored at 4°C until the analysis could be completed later that day.

A single beam atomic absorption spectrophotometer equipped with a long path burner for air acetylene mixtures was used (Instrumentation Laboratory Ltd, Birchwood, Warrington, Cheshire, England). Calibration was carried out at 213.9 nm with an aqueous solution of zinc acetate. All solutions and dilutions were made in deionised glass distilled water. The plasma samples were diluted one in 10, mixed, and aspirated directly into the flame after centrifugation. A deep frozen pooled

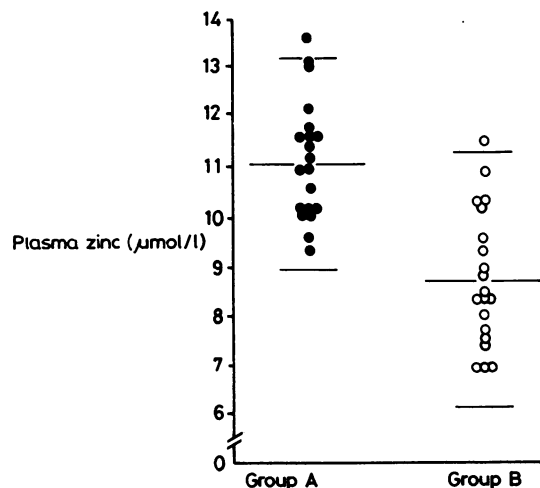


Fig. 1 Scattergram of plasma zinc concentration in people without (group A) and with senile purpura (group B). Horizontal bars represent means and 2 SD.

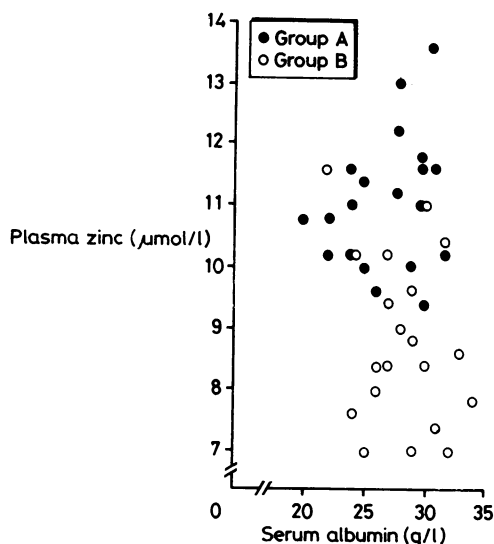


Fig. 2 Diagram showing plasma zinc v serum albumin concentrations in groups A and B.

serum gave a precision (between batch) of 3.3% (mean = 11.96; n = 9). The normal range for this laboratory is 12–19 µmol/l.

Serum albumin determinations were carried out by an automated bromocresol purple method (reference range 27–42 g/l).¹¹

Results

The results of this study showed (Fig. 1) that the mean (SD) plasma zinc value in group A was 11.1 (1.1) µmol/l. In group B the mean value was 8.8 (1.37) µmol/l. The group with purpura, therefore, had significantly lower concentrations ($t = 5.803$, $p < 0.001$).

Our results also showed (Fig. 2) that plasma zinc concentrations did not correlate with serum albumin concentrations in either group. The mean serum albumin concentration in group A was 26.95 (3.51) g/l and in group B 28.25 (3.24) g/l.

Discussion

Both groups of patients came from the same population in terms of reasons for admission, length of stay in hospital, and diet. The reason for the lower plasma zinc values in the patients with purpura is not clear. Possible explanations are malabsorption and increased requirements or losses of food, but it is less likely to be the result of diet.

The known clinical features of zinc deficiency, such as rashes, dwarfism, apathy, alopecia, delayed

surgical wound healing, and increased susceptibility to infection, have all been well described, as has the association of lower zinc concentrations in sickle cell anaemia and acrodermatitis enteropathica.^{12,13} Although to our knowledge senile purpura has not been reported in association with low plasma zinc concentrations, this may be because zinc has largely been studied in children and young adults rather than the elderly. It should be noted that the geriatric control values were clustered around the lower end of the normal range previously established in this laboratory in younger adults.

Zinc in plasma has been found to be bound largely to albumin (85%) and to an α^2 macroglobulin.¹⁴ As the binding to the α^2 macroglobulin is said to be remarkably constant¹⁵ it follows that variations are likely to be due to the zinc bound to albumin. In these patients, however, the low zinc value was not due to low albumin concentrations. Does the purpura develop, therefore, simply as a result of genuinely low zinc concentrations or in response to a related phenomenon? The possible combination of these with the aging processes of the skin, connective tissues, and small blood vessels should be considered.

We consider, therefore, that this new observation deserves further study, including consideration of a therapeutic trial. Senile purpura is not simply an unsightly indication of aging; its presence may be a good reason to suspect deficiency of zinc and, possibly, other trace metals in the bodies of the elderly.

We thank Dr OHD Portsmouth and Dr RM Kamalarajan for allowing us to study their patients, and Mrs Rae Williams for secretarial help.

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