PLASMA AND TISSUE ZINC IN TYPE 2 (NON-INSULIN-DEPENDENT) DIABETES MELLITUS

Sir.

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Low plasma zinc in diabetic patients is well documented (2,4). Surprisingly, a correlation between clinically recognized zinc deficient state and tissue zinc has not been attempted. Reports available pertain to study of autopsy material giving muscle zinc level as $243 \mu g/g$ (dry weight) in diabetics as opposed to $197-226 \mu g/g$ (dry weight) in other subjects (8,14). This led us to study the zinc level of muscle and adipose tissue (known to be markedly affected by insulin action in Type 2 (non-insulin-dependent) diabetes mellitus patients and to see if a correlation exists between plasma and tissue zinc levels and if control of diabetes affects the levels.

The study was carried out on 40 cases of overt Type -2 (non-insulin-dependent) diabetics (Table I) and 15 normal subjects of the same age group. Patients having signs of nutritional deficiency were not included in the study since zinc metabolism is known to be affected by malnutrition (7). The cases suffering from altered red cell function, chronic skin condition, pregnancy, and other disease states reported to alter plasma/serum zinc were also excluded from this study (3,4,12). The patients had no vascular, renal or retinal disease and were not taking steroid/diuretics. Care was taken to maintain patients on an adequate amount of protein (11). Zinc content (5) in diet was kept between 10-15 mg/day so as to ensure normal excretion of zinc (7,11) in urine (viz. 0.4-0.6 mg/day) and faeces (viz. 9-11 mg/day). To serve as good aged matches controls patients attending out patients department for various presentations of anxiety syndrome who were without any organic disease were selected.

The adipose tissue and skeletal muscle samples were obtained by biopsy under local anaesthesia, from anterior compartment of the thigh. The consent was taken from each patient individually after explaining him in details the objects of the study, before taking biopsy.
All glasswares, needles, syringes and biopsy instruments were prepared such as to eliminate any contamination with zinc (8). The plasma and tissue was acid-digested and zinc content was determined by atomic absorption spectrophotometric technique (1,8). Statistical analysis was done by using Student’s ‘t’ test. Findings are summarized in Table I.

### TABLE I: Plasma and tissue zinc levels (Mean±SEM) in diabetic subjects (Controlled or uncontrolled) and in non-diabetic subjects.

(Tissue levels are expressed as µg/g of wet weights)

<table>
<thead>
<tr>
<th>Subjects</th>
<th>Number in brackets</th>
<th>Plasma (µg%)</th>
<th>Muscle (µg%)</th>
<th>Adipose tissue (µg%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non-diabetic (Control)</td>
<td>15</td>
<td>111.15±1.04</td>
<td>90.06±1.77</td>
<td>100.13±1.63</td>
</tr>
<tr>
<td>Diabetics (40)*</td>
<td></td>
<td>92.77±1.40*</td>
<td>74.12±1.09*</td>
<td>67.76±1.19*</td>
</tr>
<tr>
<td>Controlled (26)b</td>
<td></td>
<td>92.24±1.13*</td>
<td>73.27±1.27*</td>
<td>67.84±1.34*</td>
</tr>
<tr>
<td>Uncontrolled (14)*</td>
<td></td>
<td>94.14±3.9*</td>
<td>75.71±2.20*</td>
<td>65.21±1.99*</td>
</tr>
</tbody>
</table>

(a) The group comprised of 23 males and 17 females (25 of which were ≥ 45 years in age. and 20 had disease of 5 years duration or longer).

(b) 10 by diet alone 16 received chlorpropamide.

(c) Through uncontrolled; 6 were on diet and 8 on chlorpropamide.

*P < 0.001, in comparison with non-diabetics (values in controlled and uncontrolled diabetics did not differ statistically)

The mean plasma zinc in diabetic patients was significantly lower than controls which accords with earlier reports (2,4). Similarly, in the diabetic subjects the mean muscle and adipose tissue zinc was significantly lower than controls. There was no statistical correlation between the plasma zinc and muscle or adipose tissue zinc in the diabetic subjects (γ<0.5).

Though separate analysis has not been presented here, no significant difference in the plasma and tissue zinc was found in the various age groups. No difference was found between the plasma or tissue zinc values of those diabetics in whom the diabetes was controlled as compared to those who were uncontrolled at the time of the study.
Significantly low muscle and adipose tissue zinc in diabetics has been attributed to break-down of zinc in insulin responsive tissues, such as muscle and adipose tissue, or may be a consequence of hyperzincuria (9,10). Strikingly, hyperzincuria in diabetes does not disappear with the control of diabetes (2,7). It is suggested that hyperzincuria is a genetically inherited disorder and not directly related to metabolic alterations of diabetes (9).

Poor wound healing in diabetes is a well known phenomena. It be of interest to know if low plasma and tissue zinc, as seen in the present study could be a contributory factor in poor wound healing in diabetics, since role of zinc in wound healing has been proposed (10).

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REFERENCES


