ZINC AND SUDECK'S ATROPHY

O. SAHAP ATIK*
ILHAMI TELLI*

SUMMARY: Zinc ion levels in bone tissue of patients with Sudeck's atrophy were determined by use of Perkin Elmor Model 103 Atomic Absorption Spectrophotometer and Parker's method. Zinc in bone tissue was 76.6 ± 6.2 μg/g in the patients with Sudeck's atrophy and 92.5 ± 3.8μg/g in the control group. The difference between groups was found to be significant (p<0.05). On the basis of experimental and clinical evidences, we suggest that zinc depletion leads to the increase of the endogenous heparin and prostaglandins, which are probably cofactors of parathormone, and may have a role in the pathogenesis of regional osteoporosis in Sudeck's atrophy.

Key Words: Zinc, Sudeck's atrophy.

INTRODUCTION

Sudeck's atrophy (reflex sympathetic dystrophy; post-traumatic painful osteoporosis) is spotty decalcification, developing after trauma, and associated with pain, edema, tenderness, cyanosis, coldness, sweating and stiffness of the part. Many theeries regarding the pathogenesis of this condition have been proposed, but no single theory has been provent (1,2).

The irritative stimulus, often traumatic in origin (fracture, incomplete nerve injury, etc.), reflex provokes continuous vasospasm of the terminal arterial channels. It still remains to be demonstrated how a minor injury can cause severe, persistent pain after the injured tissues have healed (3).

The pathogenesis of regional osteoporosis (spotty rarefaction) is also unclear. The present study was designed to determine the zinc ion levels in bone tissue of patients with Sudeck's atrophy.

*From the Department of Orthopedic and Traumatic Surgery, Gazi University Medical School, Ankara, Turkiye.

MATERIALS AND METHODS

All 34 patients in the study were from the Department of Orthopedic and Traumatic Surgery of Gazi University Hospital. Their ages varied from 10 to 80 years. The mean age of control group was 30 years, and it was 39.5 years for the patients with Sudeck's atrophy (the mean ages of the groups were not significantly different). 22 patients had sudeck's atrophy. The others served as control subject.

Each bone specimen was obtained from lateral malleol or styloid of ulna according to in which region Sudeck's atrophy is.

Zinc levels were determined by use of the Perkin Elmer Model 103 Atomic Absorption Spectrophotometer and Parker's method (4).

RESULTS

The results are summarized in Table 1. The values obtained from specimens of the patients with Sudeck's atrophy (76.6 ± 5.2 μg/g) were lower than those of control group (92.5 ± 3.8 μg/g). The difference between groups was found to be significant ($p<0.05$).

DISCUSSION

Several reports have been published suggesting the role of zinc ion in bone metabolism (5-7). Zinc is essential for bone formation.

Table 1

	Sudeck's Atrophy Group (μg/g)	Control Group (μg/g)
1	78.3	76.0
2	102.2	72.0
3	76.6	115.0
4	58.4	69.3
5	115.2	86.4
6	93.8	92.0
7	68.1	143.0
8	57.9	93.3
9	79.7	92.0
10	72.2	91.6
11	68.4	87.3
12	83.3	93.0
13	45.6	
14	82.5	
15	89.5	
16	68.2	
17	54.7	
18	105.0	
19	45.0	
20	75.7	
21	82.5	
22	73.5	
Mean ± S.E.	76.6 ± 3.8	92.5 ± 6.3

In the present study, zinc levels in bone tissue of the patients with Sudeck's atrophy were lower than those of the control group. These findings supports the provious observations of Calhoun *et al.*, which suggest an important role for zinc in osteogenesis.

The role of zinc deficiency in osteoporosis is, however, unclear. Zinc also helps to stabilize the cell membrane structure and prevents the release of endogenous heparin-containing granules (8). On the other hand, zinc inhibits the metabolism of prostaglandin. So, zinc depletion probably causes increase of the PGE_2-like activity (9).

There are also several reports indicating the role of prostaglandins and heparin in bone destruction (10,11). It was demonstrated that the addition of PGE_2-like activity or heparin to the medium in which bone tissue is cultured, enhances the action of parathormone (12,13).

On the basis of experimental and clinical evidences, we suggest that zinc depletion leads to the increase of the endogenous heparin and prostaglandins.

These are probably cofactor of parathormone and may have a role in the pathogenesis of regional esteoporosis in Sudeck's atrophy.

ACKNOWLEDGEMENTS

We thank Naci M. Bor, M.D. for his technical support.

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Correspondence:
O. ÞSahap Atik
Dept. of Orthopedic and Traumatic Surg.
Gazi University Medical School,
Ankara-TÜRKIYE.