THE SIGNIFICANCE OF LOW SERUM IRON IN TROPICAL PYOMYOSITIS

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SUMMARY: The serum iron (SI), total iron binding capacity (TIBC), transferrin saturation (TS) and serum ferritin (SF) levels were studied in 16 Nigerian patients with tropical pyomyositis (TP) and in 16 control subjects (CS). It was observed that the mean SI (μmol/L) and TS (%) values were significantly lower in TP than in CS (SI:7.32±2.33, 14.41±3.02, p<0.02; TS:14.24±4.03, 24.34±8.53, p<0.02). There were, however, no significant differences in the TIBC (μmol/L) and SF (μg/L) values between TP and CS (TIBC: 51.4±9.6, 56.2±8.5, p>0.5; SF:269±108, 239±113, p>0.5). The low serum iron level in tropical pyomyositis is therefore considered as reflection of sequestration into the reticuloendothelial system rather than an absolute iron deficiency. It seems unlikely that iron deficiency is a contributory factor in the aetiology of tropical pyomyositis.

Key Words: Tropical pyomyositis, serum iron, TIBC, transferrin saturation, serum ferritin.

INTRODUCTION

Tropical pyomyositis is a clinical entity involving the formation of skeletal muscle abscesses in various parts of the body. The clinical and epidemiological features of this disease has been well described from any tropical countries (3,11). Recently the disease has been increasingly reported from temperate areas of the world (8) and hence there is yet no agreement on the nomenclature of the disease (14). The precise aetiological factor responsible for this disease has still remained unknown. Even though *Staphylococcus aureus (S. aureus)* is the cultured microorganism in most cases (7), it is generally believed that this organism is only a secondary invader. Various other factors including trauma (10), malnutrition (6), vitamin deficiency (19), viral infection (15) and muscle damage by migrant helminthic larvae (12) have all been blamed for the primary assult to affected muscles. Iron deficiency, a common clinical condition in the tropics may predispose to bacterial infections (1). In addition, iron deficiency may lead to poor polymorph function especially phagocytosis and intracellular bacterial killing (17). Apart from the low serum iron observed in an isolated report by Gibson *et al.* (8), the role of iron in this disease has not been studied and hence it is not clear whether or not his element plays any significant role in the aetiology of tropical pyomyositis. It is with these views in mind that we studied the serum iron status of 16 Nigerian patients with tropical pyomyositis (TP) as well as in 16 normal subjects as controls (CS) and the results obtained from the details of this communication.

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PATIENTS AND METHODS

All patients in this study were, children or adults, admitted to Ahmadu Bello University Hospital, Zaria, with tropical pyomyositis during a prospective study by us as reported earlier (7,9). Patients were clinically examined on admission and regularly assessed until discharged. About 10 ml of blood was taken for estimation of the level of serum iron (SI), total iron binding capacity (TIBC), transferrin saturation (TS) and serum ferritin (SF) before any form of therapy. The SI and TIBC were measured spectrophotometrically by using 'Iron BP Test Reagents' and 'TIBC Test Reagents' respectively from Roche Diagnostica, Switzerland. The TS (%) was calculated from the equation: SI+Unbound Iron Binding Capacity=TIBC. The SF was determined by radio immunoassay with reagent Kits from Amersham International PLC, UK. The statistical analysis of the results was made by Student's t-test.

RESULTS

The ages of the 16 patients, 11 males and 5 females, ranged from 7 to 65 years. The details of the skeletal abscesses and microbiological culture patterns of pus and blood were described in our previous reports

Table 1: Serum iron status in patients (TP) and controls (CS) and their statistical analysis by Student's t-test.

Subjects	Serum iron status			
	SI (μmol/L)	TIBC (μmol/L)	TS (%)	S (μg/L)
TP:				
n	16	16	16	16
Mean±SD	7.32±2.33	51.41±9.61	14.24±4.03	269±108
CS:				
n	16	16	16	16
Mean±SD	14.41±3.02	56.21±8.51	24.32±8.53	239±113
*CS and TP:	S	NS	S	NS

* S: Significant ($p < 0.05$); NS: Not significant ($p > 0.05$)

(7, 9). Fourteen of 16 pus specimens (88%) and 3 of 16 blood specimens (19%) were positive for *S. aureus.* Proteus species was isolated from 1 speciman (6%). The mean SI (μmol/L) and TS (%) levels were significantly lower in TP than CS and there were, however, no differences in the TIBC (μmol/L) and SF (μg/L) values between TP and CS (Table 1).

DISCUSSION

There has been an increased interest in tropical pyomyositis especially with recent reports from non-tropical areas (8,13). Even though *S. aureus* is grown in 88% of cases (7), this organism is generally regarded as a secondary intruder and the primary underlying causative factor(s) continue to be uncertain. Trauma has long been suggested as a possible primary aetiological agent (10), but its precise role is inconclusive. Coxsachie B and other area viruses have been postulated to precede bacterial invasion (15), but evidence for this is not definite. Other reports (6,19) have suggested that malnutrition and vitamin deficiency may predispose to tropical pyomyositis. However, in two separate reports from Ugnada (19) and Nigeria (4), malnutrition and avitaminosis were not considered important aetiological factors in tropical pyomyositis.

The level of iron in the body has important bearings on both the body's immune response and susceptibility to infection (17). Waterlot *et al.* (16) observed that dialysis patients with iron over load had more infectious episodes than those with normal serum iron. In a similar way, iron deficiency also may impair the hosts immune response and predispose the subject to bacterial infections (1).

The impairment of neutrophil (PMN) function has been observed in mildly iron deficient children and depressed intracellular bactericidal action of neutrophils has also been reported in iron deficient subjects (2). Iron deficiency is a common clinical condition in the tropics because of nutritional deficiencies and parasitic infections. The question therefore arises: could the poor PMN function observed in pyomyositis be due to iron deficiency. Our observation that the ability of PMN to reduce NBT was similar in TP and CS suggested that there may not be any significant metabolic abnormality in PMN (9). However, our patients had low SI and TS with normal TIBC and SF levels as reported by others (8). This observation is an indication that our patients were probable not iron deficient since SF level is a sensitive marker of the body's iron store (5). We therefore propose that the low serum iron observed in our patients is an acquired one. This may be due to the hosts nonspecific defense response to sequestrate iron into the body's reserves so that this element is not available to the invading micro-organisms (17, 18). Therefore, iron deficiency may be ruled out as a probable cause for the inefficient killing of *S. aureus* by PMN in TP (13). In the presence of metabolically normal PMN and adequate amounts of antibody, *S. aureus* is readily phagocytosed, killed and eliminated (4). However, phagocytosis of *S. aureus* by PMN may be defective in TP due to non-availability of opsonising antibodies, particularly of IgM class, as proposed by Giasuddin *et al*. (7). This defective phagocytosis may therefore explain the inefficient clearance of *S. aureus* by PMN in TP (13).

In conclusion, it seems unlikely that iron deficiency is an aetiopathogenic factor in tropical pyomyositis. In support of this is the rarity of tropical pyomyositis in patients with hookworm anemia which is a common clinical condition leading to iron deficiency anemia in the tropics.

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