

Thiamine Responsive Ankle Oedema in Detention Centre Inmates

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Summary

Twenty-seven inmates from a detention centre in Perak were evaluated for possible causes of their ankle oedema. Physical examination and biochemical evaluation did not show any evidence of renal or hepatic dysfunction. The cardiac origin of their problem was suggested by the presence of other signs of heart failure in three of them and by radiological evidence of cardiomegaly in 40% of them. All the patients who returned for review demonstrated a prompt clinical response to thiamine replacement therapy.

Key Words: Ankle oedema, Detention centre inmates, Thiamine deficiency, Beri-beri

Introduction

The common causes of bilateral painless ankle oedema in adults are congestive cardiac failure and hypoproteinaemic states due to malnutrition, liver or renal disease. Dietary deficiency of thiamine leading to wet Beri-beri is not a common cause of ankle oedema in Malaysia today, though it was common in the pre-World War Two Era^{1,2}.

According to the Straits Settlements Medical Reports³, there were 2,287 deaths due to Beri-beri in the Straits Settlements in 1904. However, since Independence in 1957, Beri-beri has become a rarity in Malaysia.

This paper describes the clinical, biochemical and chest radiographic features of a group of illegal immigrants who developed ankle oedema while in a Detention Centre in Perak, Malaysia. The prompt response to thiamine replacement therapy in the 16 patients who returned for review a week later, suggests the strong possibility of thiamine deficiency as a cause of their ankle oedema.

Materials and Methods

Twenty-seven detainees from the Detention Centre for

illegal immigrants at Langkap, Perak, were brought to Teluk Intan District Hospital between 10/05/93 and 08/07/93 for treatment of ankle oedema. None of the patients had any history of previous episodes of ankle oedema or of heart, liver or kidney problems. As thiamine deficiency had been previously diagnosed in cases from the same centre in December 1992⁴, a presumptive diagnosis of Beri-beri was made at presentation for all these 27 cases.

The cardiovascular and neurological of these patients were assessed clinically, and their weights were recorded to the nearest 0.5kg with them standing bare-foot on a bathroom scale (Kubota, Japan, reading 0-130kgs). Baseline investigations including urine albumin, blood urea, serum albumin and a chest X-ray were obtained for each of these patients. Blood samples were also sent to the Institute for Medical Research (IMR) in Kuala Lumpur for thiamine level estimation.

Sixteen patients were reviewed a week later to assess response to therapy. They were examined for persistence of ankle oedema, and their weights were re-checked using the same weighing scale as during their first visit. If the first chest X-ray had revealed cardiomegaly, a repeat X-ray was done at follow up.

Results

The majority of the patients were young Bangladesh men aged between twenty and thirty years who had been detained for periods between five and eight months (Tables I & II).

Table I
Ages and nationalities of the illegal immigrants with ankle oedema

Age	Nationality	
	Bangladeshi	Indonesian
≤ 20 years	1	0
21 – 30 years	20	3
31 – 40 years	2	1
Total	23	4*

* 3 of them were Achehnese

Table II
Duration of detention at presentation

Duration of detention (months)	Bangladeshi	Indonesian
0 – 2	0	0
3 – 4	4	1
5 – 6	10	0
7 – 8	1	0
9 – 10	0	0
>10	1	3
Not recorded	7	0
Total	23	4

On examination all 27 patients had definite pitting oedema over the ankles. Several of those managed as outpatients also had pitting oedema up to the thigh level. None were jaundiced and all were normotensive. Three of these patients gave a history of marked reduction in effort tolerance. On examination they had tachycardia, elevated jugular venous pressures, basal pulmonary crepitations and pitting oedema on the

trunk as well. On direct questioning many of the detainees complained of numbness of limbs but no objective evidence of neurological deficit was found.

None of the patients assessed had proteinuria or an active urinary sediment. Six of the nine patients who had hemoglobin estimations done were mildly anaemic with hemoglobin levels of between 10 and 12 grams per decilitre. Out of the twenty patients who had serum albumin levels assessed, two had levels below 30 grams per litre and six others had levels between 30 and 35 grams per litre (Table III). Ten out of the twenty seven patients had a cardiothoracic ratio of 0.5 or greater (Table IV).

Blood for thiamine levels was sent to IMR KL for 21 of these patients before commencing thiamine replacement. However results have only been received for 15 patients. At IMR, red blood cell transketolase levels and the effect of adding thiamine pyrophosphate (TPP effect) were assayed. Individuals with thiamine deficiency should have transketolase levels below 50 international units (i.u.) with a TPP effect of more than 25%. The TPP effect is considered the more sensitive of the two parameters⁵ (Table V).

Going by the criteria outlined earlier only one patient had unequivocal evidence of thiamine deficiency.

Table III
Laboratory investigations at presentation

Investigation	Number of patients assessed	Mean	Range
Urine albumin	24	(nil for all patients)	
Blood urea (mmol/L)	22	31	1.4 – 4.6
Serum albumin (gm/l)	20	35.2	27 – 43
Prothrombin time (seconds)	4	14.3	14 – 15
Hemoglobin (g/100ml)	9	11.4	10.1 – 13.3

Table IV
Cardiothoracic ratio (CTR) in chest X-ray P.A.
view at presentation

Cardiothoracic ratio	Number of Patients	
	Outpatients	Inpatients
0.40 - < 0.45	5	0
0.45 - < 0.50	12	0
≥ 0.50	7	3
	24	3

Table V
Thiamine levels (n=15)

Thiamine Pyrophosphate Effect (%)	RBC Transketolase Levels (i.u)		
	<50	50 - <60	≥60
> 25	1	0	0
15 - ≤ 25	0	1	2
< 15	3	3	5

Another three had low levels of RBC transketolase levels but had normal TPP effect while another three patients had normal RBC transketolase levels but marginally elevated TPP effect. The remaining eight patients were found to have no evidence of thiamine deficiency.

The three patients who had other signs of congestive cardiac failure to pitting ankle oedema were admitted for intravenous thiamine replacement (25 mg tds). The remaining 24 patients were treated as outpatients with thiamine tablets (6 mg tds). None of the 27 patients received any diuretics or cardiac glycosides except for one of those admitted who was given intravenous frusemide for 5 days.

All these detainees were asked to come back one week later for a review. But only 16 of them did come back. Many of them still complained of weakness and

tiredness. However the ankle oedema had completely resolved in all 16 of them. Weight increased by 0.5kg and 4.0kg respectively in two of those treated as outpatients, remained static in one patient, but decreased in thirteen patients. The largest weight loss of 10kg was observed in the inpatient who had been given frusemide as well as intravenous thiamine, while another four patients had weight loss of 3 or more kilograms and five had weight losses of between 2 and 3 kg.

Eleven patients had a repeat chest X-ray done and six had a decrease in their cardiothoracic ratio (CTR) ranging from 0.2% to 8.6%. The greatest diminution in CTR occurred in one of those treated with oral thiamine. There was no obvious correlation between weight loss and diminution in the cardiac size on chest X-ray.

Discussion

The cardiac origin of ankle oedema in these patients is suggested by the presence of cardiomegaly in their chest X-rays at presentation. Ten of them had a cardiothoracic ratio (CTR) of 0.50 or greater, while another 12 had a CTR of between 0.45 and 0.50. In addition, three of them also had other clinical evidence of cardiac involvement such as elevated jugular venous pressures and pulmonary crepitations.

Their ages, the absence of a previous history of heart disease, the absence of hypertension and severe anaemia, and their rapid clinical response to thiamine replacement therapy strongly supports the presumptive diagnosis of thiamine deficiency in these patients. It is possible that borderline hypoproteinemia also played a contributory role in some of them.

This clinical impression is not supported by the results of blood specimens sent to the Institute for Medical Research (IMR) for bioassay of thiamine levels. This may be due to three factors. Firstly, this test was not done for the three inpatients who had the worst symptoms because it was felt that it would be dangerous to delay thiamine replacement therapy in them. The tests being offered in the Institute for Medical Research are sensitive enough to pick up cases of subclinical thiamine deficiency⁵ and should have shown up thiamine deficiency even in those treated

as outpatients. The effect of the long transit time of about 20 hours on thiamine estimation is another possible cause of this incompatibility of clinical and laboratory findings. Finally, one must also consider the laboratory error as these are tests that are not frequently performed.

The Ministry of Health was alerted regarding this cluster of suspected Beri-beri cases in early June 1993, and the Ministry requested that the nutritional and health status of inmates in all nine centres in Peninsular Malaysia be looked into. The investigation of the centre in Langkap was completed in July 1993 and the report has been submitted to the Director-General. The Langkap centre has started serving a vitamin B complex tablet to each detainee at breakfast since early July 1993 and there were no further cases of ankle oedema presenting from the centre over the

next six months. It is hoped that with the heightened awareness regarding the possibility of nutritional deficiencies and the remedial actions taken, Beri-beri will no longer occur in these detention centres.

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