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A Retrospective Study of Anaemia at Harari Hospital during 1973

I. Microcytic Anaemias

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Although anaemia is common it is rarely the primary cause of hospital admission. Patients are frequently admitted for other illnesses, and subsequently found to be anaemic. We have conducted a survey at Harari Hospital to assess the magnitude of this problem. The year 1973 was chosen as it was the first year that a Coulter counter was routinely used for blood counts on all hospital patients.

Methods

All folders of patients admitted to the five medical wards of Harari Hospital from January 1st, 1973, to December 31st, 1973, were obtained. Folders of all patients with haemoglobin concentrations of less than 12 Gm.% were extracted, and details of clinical presentation, special investigations and treatment recorded. This paper presents the findings on patients with microcytic anaemias (M.C.V. < 80 μ^3).

Results

The total number of patients admitted to the general medical wards at Harari Hospital in 1973 was 3 780. Of these 2 350 (62 per cent.) were male, and 1 430 (38 per cent.) were females, a ratio of 1,6 to 1,0.

Of the 3 780 patients admitted, anaemia (haemoglobin concentration of less than 12 Gm.%) was present in 454 (12 per cent.). One hundred and twenty-two (27 per cent.) of these were microcytic. The incidence of microcytic anaemia in all patients admitted was 3 per cent. The age and sex distribution of patients with microcytic anaemia is shown in Table I. The male-female sex ratio was 1:1,4, an almost complete reversal of the overall admission ratio (1,6:1,0).

The severity of anaemia in relationship to M.C.V. values is shown in Table II. Seven per cent. of patients had severe microcytosis (M.C.V. < 60), and the majority (8/9) of these were severely anaemic (haemoglobin concentration less than 8 Gm.%). Thirty per cent. of patients had moderate microcytosis (M.C.V. 61 to 70 μ^3) and 20/36 had a haemoglobin concentration less than 8 Gm.%. In the patients with mild microcytosis (M.C.V. 71 to 80 μ^3), 30/77 had a haemoglobin concentration less than 8 Gm.%.

Broad categories of illness precipitating hospital admission in these patients are shown in Table III. Thirty patients (25 per cent.) presented with acute bacterial infections. Thirteen had typhoid, four had pneumonia, three had bacterial endocarditis, three had acute bacterial gastro-enteritis, two had meningitis and there were single cases of acute otitis media, acute pyelonephritis, laryngotracheobronchitis, acute pelvic sepsis and septic arthritis.

There were eight patients (7 per cent.) with chronic bacterial infections. Seven of these had tuberculosis, and one patient had chronic bronchiectasis.

Twenty-three patients (19 per cent.) had parasitic infestation. Ten of these had major schistosomal disease, seven having cirrhosis and portal hypertension ascribed to schistosomiasis. Five patients had hookworm, four had malaria and four had amoebiasis.

Uraemia was the cause of admission in 14 (11 per cent.) of these patients. In five of these, obstructive uropathy secondary to schistosomiasis was present.

The remaining categories are self explanatory, except for the "others" group. Four of these patients were diagnosed as cardiomyopathies, three were homozygous sicklers, two had congenital heart disease, one had chronic pancreatitis with steatorrhoea, one had a subarachnoid haemorrhage secondary to an intracranial aneurysm, one had an unexplained

TABLE I

Age and Sex Distribution of Patients with Microcytic Anaemia

Age (Years)	*M.C.V.*						*Overall MCV<80*	
	< 61		61-70		71-80			
	Male	*Female*	*Male*	*Female*	*Male*	*Female*	*Male %*	*Female %*
10	0	0	3	2	6	3	7	4
11-20	2	0	6	5	9	13	14	15
21-30	1	2	0	2	5	11	5	12
31-40	0	1	3	6	5	8	7	12
41-50	1	1	0	6	1	5	2	10
50	0	1	2	1	7	4	7	5
Totals	4	5	14	22	33	44	42	58
Per cent.	3	4	12	18	27	36		

TABLE II

Severity of Anaemia in Relationship to M.C.V. in Subjects with Microcytic Anaemia

Haemoglobin Concentration (Gms.%)	*M.C.V.*					
	60		61-70		71-80	
	%	*No.'s*	*%*	*No.'s*	*%*	*No.'s*
4	1	1	3	4	2	2
4,1- 6.0	3	4	5	6	7	8
6,1- 8,0	2	3	8	10	15	20
8,1-10,0	1	1	14	16	34	41
10,1-12,0	0	0	0	0	5	6
Total	7	9	30	36	63	77

TABLE III

Illnesses Precipitating Admission to Hospital in Subjects with Microcytic Anaemia

Illness Category	*No.*	*%*
1. Acute bacterial infections	30	25
2. Chronic bacterial infections	8	7
3. Parasitic infestations	23	19
4. Uraemia	14	11
5. Rheumatic heart disease	11	9
6. Cirrhosis	7	6
7. Neoplastic disease	6	5
8. Peptic ulceration	5	4
9. Menorrhagia	5	4
10. Others	13	10

immune haemolytic anaemia and one had scleroderma.

This relatively simple primary presentation was complicated by a number of other factors contributing to morbidity in these patients.

Of the 30 patients presenting with acute bacterial infection, five had associated schistosomiasis, four had menorrhagia ascribed in each case to uterine fibromyomata, and single patients had cirrhosis with portal hypertension, uraemia, G.6.P.D. deficiency and well advanced pregnancy.

Two of the eight patients presenting with chronic bacterial infection had co-existent chronic pyelonephritis, one had schistosomiasis and one was uraemic. Four of the 13 patients

with parasitic infestation not due to schistosomiasis had co-existent schistosomal disease.

Of the 14 uraemic patients, five had severe schistosomal lesions, one presented with bleeding from the gastro-intestinal tract, and one had co-existent amoebiasis.

Three of the 11 subjects with rheumatic heart disease had schistosomiasis, and pyelonephritis, hookworm or shigellosis was present in three further subjects. Two of the seven cirrhotic subjects had schistosomiasis and one was a heterozygous sickler.

Overall, 25 per cent. of patients had significant schistosomal infestation, while in a further 22 per cent. some problem other than the discharge diagnosis contributed to morbidity.

The number of patients adequately investigated to assess blood loss or iron status was small. Of the 122 subjects, 89 (73 per cent.) were not investigated at all. Serum iron and iron binding capacity was measured in 20 (16 per cent.), and faecal occult blood in 21 (17 per cent.). Bone marrow aspiration and assessment of iron stores was performed in six (5 per cent.). On the basis of the above investigations, 23 out of 24 patients were shown to be iron deficient by standard criteria. The sole exception had normal bone marrow iron stores and was assumed to have anaemia secondary to his infection.

As so few patients were adequately investigated, the likelihood of bleeding from gastro-intestinal or genito-urinary sites was assessed in relationship to the clinical presentation. Twenty-four (20 per cent.) of patients had a lesion likely to cause upper gastro-intestinal bleeding while 11 (9 per cent.) could have bled from a lower gastro-intestinal lesion. Ten per cent. of patients had menorrhagia, 4 per cent. had hookworm. 2 per cent. had bladder lesions, and 2 per cent. were pregnant. A presumed reason to account for blood loss was present in 48 per cent. of the patients.

Discussion

The value of retrospective surveys can be questioned as all too often essential information is lacking. They do however, provide a platform upon which to base prospective studies.

Three per cent. of the total hospital admissions had a microcytic, usually hypochromic anaemia; in all the anaemic patients only 27 per cent. had this type of anaemia, a surprisingly low figure. This may reflect upon the cooking habits and high dietary iron intake of African patients. In a similar survey by Lunat and Gelfand (1970), 66 per cent. of patients admitted with a haemoglobin concentration of less than 9.5 Gm.% had a hypochromic anaemia. The disparity between this survey and theirs may reflect different criteria for study.

The majority of our patients were female, as opposed to the male predominance for overall admissions. This presumably reflects the precarious state of iron balance in females as a consequence of menstruation, pregnancy and lactation (Monsen *et al,* 1967; Pritchard and Mason, 1964; Scott and Pritchard, 1967).

There are a number of causes of microcytosis, but hypochromic microcytic anaemia is nearly always due to iron deficiency (Harris and Kellermeyer, 1970). Rare causes of microcytic anaemia include lead poisoning, refractory sideroblastic anaemias, chronic infection and inflammation (Moore C. V., 1963). On this basis we have assumed that the vast majority of patients in this study had underlying iron deficiency.

The diagnosis of iron deficiency is a relatively easy one to make. Microcytic hypochromic anaemia is the feature of established disease, but once this occurs iron stores are severely depleted (Zizza and Black, 1961; Pirzio Biroli and Finch, 1960; Conrad and Crosby, 1962). In the early stages there is usually normochromic normocytic anaemia, and other criteria are required to show iron lack. It has been shown that erythropoiesis is suboptimal below serum iron concentrations of 70 μ% and plasma transferrin saturations less than 15 per cent. (Hillman and Henderson, 1969). Values lower than this will reflect iron deficiency. Unfortunately a number of other factors influence serum iron and transferrin concentrations, particularly infection, inflammation and neoplastic disease, which usually depress both serum iron and transferrin concentrations (Beutler *et al,* 1954). The best method of assessing iron reserve is by measuring bone marrow or liver iron stores (Beutler *et al,* 1954; Beutler, 1959) or by measuring serum ferritin concentration (Lipschitz *et al,* 1974; Siimes *et al,* 1974; Addison *et al,* 1972).

Although it is important to establish that the anaemia is due to iron lack, it is more important to establish the reason for this. The usual cause is chronic blood loss, either from the genito-urinary or gastro-intestinal systems. In only 48 per cent. of the patients surveyed was this probable on the basis of their clinical illness, and only 25 (20 per cent.) of subjects had their faeces checked for the presence of blood. Gastro intestinal blood loss occurred in

35 subjects, uterine loss in 11 (almost invariably associated with fibromyomata), and two from bladder lesions.

Lunat and Gelfand (1970) found that 8 per cent. of their subjects had hookworm infestation, compared to 4 per cent. in this survey. These authors questioned the significance of hookworm as a cause of anaemia in this country where infestation is usually mild when contrasted with reports from Uganda and Nigeria (Gelfand and Warburton, 1967; Gelfand and Garnett, 1965). Unfortunately hookworm loads were not quantitated in these patients.

Twenty-five per cent. of all patients had schistosomal infestation, compared with 24 per cent. in the survey by Lunat and Gelfand (1970). This suggests that overall 25 per cent. of the population are infested with this parasite. However, seven patients had cirrhosis with portal hypertension thought to be due to schistosomal disease, and five of 14 uraemic patients were thought to have ureteric obstruction caused by schistosomiasis. It is difficult to be certain of how much blood loss can be caused by this parasite (Forsythe, 1970; Mahmood, 1966) and this subject deserves prospective study.

Thirty-two per cent of our patients presented with bacterial infections.

Uraemia and rheumatic heart disease were both associated with microcytosis. The anaemia of these disorders is usually normocytic (Editorial, *Lancet*, 1975). In 46 per cent. of uraemic and rheumatic subjects a second pathology was present, and it is probable that an underlying cause may have been found in the remaining patients had more intensive investigation been attempted.

This paper provides a basis for future prospective study of subjects with microcytic anaemia. It shows how frequently the anaemic patient is inadequately investigated, or the anaemia is "passed off" as being a consequence of the presenting illness. It also suggests that the role of schistomiasis in causing anaemia due to blood loss needs to be further investigated.

References

Addison, G. M., Beamish, M. R., Hales, C. N., Hodgkins, M., Jacobs, A., Llewellin, P. (1972). *J. Clin. Path.* **25,** 236.

Beutler, E., Drennan, W., Black, M. (1954). *J. Lab. Clin. Med.* **43,** 427.

Beutler, E. (1959). *Ann. Int. Med.* **50,** 313.

Conrad, M. E., Crosby, W. H. (1962). *Blood,* **20,** 173.

Forsythe, D. M. (1970). *Trans. Roy. Soc. Trop. Med. and Hyg.* **64,** 601.

Gelfand, M. and Garnett, P. A. (1965). *J. Trop. Med. Hyg.* **68,** 157.

Harris, J. W. and Kellermeyer, R. W. (1970). The Red Cell, p. 120, Harvard University Press, Cambridge, Massachusetts.

Hillman, R. S. and Henderson, P. A. (1969). *J. Clin. Invest.* **48,** 454.

Lancet (1975). **1,** 959. Edit.

Lipschitz, D. A., Cook, J. D. and Finch C. A. (1974). *N. Eng. J. Med.* **290,** 1213.

Lunat, M. and Gelfand, M. (1973). *C. Afr. J. Med.* **19,** 195.

Mahmood, A. (1966). *Trans. Roy. Soc. Trop. Med. and Hyg.* **60,** 766

Monsen, E. R., Kuhn, I. M. and Finch, C. A. (1967). *Am. J. Clin. Nutr.* **20,** 842.

Moore, C. V. (1963). *Aust. Ann. Med.* **12,** 16.

Pirzio Biroli, G. and Finch, C. A. (1960). *J. Lab. Clin. Med.* **55,** 216.

Pritchard, J. A. and Mason, R. A. (1964). *J.A. M.A.* **190,** 897.

Scott, D. E. and Pritchard, J. A. (1967). *J.A. M.A.* **199,** 897.

Siimes, M. A., Addiego, J. A. and Dallman, P. R. (1974). *Blood,* **43,** 581.

Zizza, F. and Black M. (1961). *Acta Haemat.* **25,** 1.