

TWO CASES OF HYPOPLASTIC OR APASTIC ANAEMIA

by

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Introduction :

Aplastic or hypoplastic anaemias usually are rarely seen in this part of the world. Over the period of three months (i.e. from Bajshak '028 to Asad '028) we came across two cases only. Both of them died. We could not account for the cause of their illness. Various possibilities are discussed below. A short account of each patient is presented as follows.

Case No. I.

Nirmal Gurung aged 14 years, male, was admitted first in Kanti hospital on 28/2/028 and died on 28/3/028 during his second admission.

The boy was admitted first with the history of increasing pallor and recurrent epistaxis of one month's duration. He used to get attacks of sore-throat with fever off and on. Previous to this he had been noticing "mosquito bites" (petechiae) over different parts of his body since 10 months. No history of drug ingestion was available. He had no significant illness in the past nor the family members had any blood diseases. He was of poor socio-economic group and was maintaining himself by tailoring.

On examination : The boy was of average weight and height, very pale with petechial rashes all over his body. Apart from the soft systolic murmur in his heart no abnormal findings were detected. His Hess' capillary fragility test was normal.

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Blood examination at this stage showed Total W.B.C. count to be 2300/mm³ with the Differential of Polymorph 18%, Lymphocytes 80%, Monocytes 2%; P.C.V. 12%, Haemoglobin 5.5 gm/100 ml. blood. The blood film showed considerable reduction of white blood cells, red blood cells and platelets. He was Rh + with group o.

A diagnosis of hypoplastic/aplastic anaemia was done and he was treated with bed rest, high protein diet, antibiotics (eg. penicillin), anabolic steroid (eg. oraboline), corticosteroids (eg. prednisolone 10 mgm tds for 7 days; then 10 mgm B.D.), haematinics (eg. iron, folic acid 10 mgm tds) and vitamin B complex.

Table IA

Date	Total WBC/mm ³	Differential WBC Count				Haemoglobin	Other
		Polymorph %	Lymphocyte %	Monocyte %	Eosinoph %		
28/2/028	2,300	18	80	0	2	5.5 G.	
31/2/028	4,100	20	79	0	1	4.1 G.	E.S.R 79mm. 18 hr
		Blood Transfusion (1 pint) given					
4/3/028						4.0 G.	Platelets 62,000/mm ³
21/3/028						3.7 G.	
25/3/028	5,100	43	51	3	3	4.3 G.	

A repeat blood examination done a few days later showed a fall of haemoglobin (i.e. 4.1 G./100 ml.) with differential W.B. count having polymorph of 20%. Unfortunately bone marrow examination could not be done at this stage. And in view of the progressive fall of haemoglobin a blood transfusion (1 pint) was given with some general improvement. But at the weekend the child deteriorated again. Repeat haemoglobin (i.e. 4.0 G/100ml.) did not improve and the platelet count as low as 62,000/mm³. Bone marrow examination done later showed gross aplasia.

Table IB.

Report on Bone Marrow (10/3/028)

Site — Iliac fossa

Consistency — Normal

Fragment — Acellular and fatty

Leucocyte / Erythrocyte ratio — Depressed

Erythropoiesis — Markedly Depressed

Leucopoiesis — Markedly Depressed

Megakaryocyte — not seen

No Malignant cell or parasite seen

Diagnosis : Aplastic anaemia.

Course — The patient rapidly went downhill and suddenly died of acute heart failure on 28/3/028 one month after his first admission.

Case No. 2.

Vidya Laxmi aged 4 years, female, was admitted first on 20/1/028 with 3 weeks' history of gradually increasing weakness, giddiness, anorexia and severe epistaxis for 8 days.

On Examination : She was ill, very pale with tachycardia. No other abnormality detected. There was no history of any toxic drug ingestion. There was nothing significant in her past illness nor in the family. She too was of poor socio-economic group.

Her blood picture during admission showed — Haemoglobin 3.7G/100ml, Total W.B.C. count 4,350/mm³ with the differential count of 28% polymorphs, 70% lymphocytes, 2% eosinophils; P.C.V. 10% and platelet count of 185,000/mm³. The blood film showed hypochromia with reduction of R.B.C., W.B.C., series (vide Table IIA). She also had upper urinary tract infection with E-coli sensitive only to streptomycin and chloramphenicol. Her blood group was of Group A and she was Rh+.

Table IIA

Date	Total W.B.C. Count per mm ³	Differential WBC Count				Haemoglobin G/100ml.	PCV	Others
		Poly- morph	lymyho- cyte	Monocyte	Eosin- ophil			
22/1/028	4,350	28	70	0	2	3.7	10%	Platelets 185,000/ mm ³
31/1/028	5,000	3	97	0	0	3.2	10%	90,000/ mm ³ Occult Blood in Stool
21/2/028	3,800	10	89	1	0	2.45	6%	Blood Film Showed Gross reduction of WBC, RBC, & Platelets.
29/2/028		Blood Transfusion Given (27.2.028)				4.30		

Table IIB

Report on Bone Marrow (17/2/028)

Site— Iliac crest

Consistency— Normal

Erythrocyte/leucocyte ratio— Depressed

Erythropoiesis— Depressed

Leucopoiesis— Depressed

Megakaryocytes— Few seen

Malignant cell or parasites— Not seen

Diagnosis— Hypoplastic Anaemia

She was treated with haemostatics (eg. clauden, calcium, vitamink), antibiotics (eg. penicillin), haematinics (eg. iron, vitamin, folic acid), corticosteroids and anabolic steroids.

Progress however was unsatisfactory. The child developed severe recurrent epistaxis and inspite of nasal packing and other treatment had severe melaena probaly due to swallowed blood.

Blood transfusion was recommended. But in view of poor economic status the party could not afford blood nor the free blood was available. However blood transfusion could be given on 27/2/028. The patient responded to treatment slightly (her post-transfusion haemoglobin was 4.3 G/100 ML). But she was taken to home on 1/1/028 where she deteriorated rapidly and was again admitted on 2/4/028 with severe bleeding from gums, coffee-group vomiting; and melaena and widespread purpura and echymoses all over her body. Two blood transfusions were given separately as a desperate attempt. But the child developed severe broncho pneumonia after 4 days and died.

Discussion

Aplastic or hypoplastic anaemia is not uncommon among Nepalese children below 16 years of age in all the cases (including the present two) which we have encountered. It has been very difficult to find any definite cause. No history of exposure to toxic drug is available although in a few previous cases have been given preparations of gold and mercury to the unfortunate patients. These preparations are claimed to be safe, but in view of their indigenous preparation (which we do not know at all) we are rather ignorant and therefore uncertain of their efficacy. All we can say is that the bone marrow has failed to function.

Regarding treatment—all of these cases of primary aplastic anaemia are given only palliative and symptomatic treatment only. Blood transfusion raises the haemoglobin to a level which is just comfortable to the patient corticosteroids and even testosterone are given to stimulate the bone marrow. When these fail even splenectomy's done as a desperate measure, but how far this is justifiable in a case of primary marrow failure one cannot say. Oxy Methalone or anabolic hormones are also given. Their mode of action is not known; but it is claimed to produce remission in 50% of cases. It has been reported that some of these cases treated with oxymethalone may develop acute myeloblastic leukaemia.

One fact to be emphasised here is that cases of normochromic anaemia without obvious cases should be thoroughly investigated haematologically.

Summary

Two cases of primary aplastic anaemia in children are reported. Their cause could not be found. Treatment was unsatisfactory. Blood transfusion was not available at short notice because the party could not afford; and unfortunately free blood usually was not available. The disease has very bad prognosis. Both of our patients died.

References

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