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Study of Anemia below 15 Years of Age

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Abstract

Aims and Objectives: To study the etiology and clinico-haematological co-relation of anemia in Pediatric age group. *Methodology:* Cross Sectional analyticalstudy done on children between 6 months to 15 years admitted in Pediatric ward whose hemoglobin levels were below the WHO cut off for age from 1st August 2013 TO 31st July 2015. *Results:* We studied 557 anemic children admitted in pediatric ward aged 6 months to 15 years. Out of 557 subject 332 were males and 225 females. Maximum number of children (52.5%) were present in the preschool age group followed by school going (31.4%) and adolescent (16.3%). Amongst preschool (63.2%) and school going (60.6%) children, male were more in number whereas females were more in adolescents (53.8%). Easy fatigability (26%), irritability (25%), Lack of concentration (17.6%) and breathlessness (16%) were the few common clinical symptoms. Children with moderate anemia were more symptomatic.Palmar pallor (81.5%), Icterus (13.2%), knuckle pigmentation (12.2%) were few common clinical signs observed. Clinical signs were more common in patients with moderate anemia. Nutritional anemia (59.6%) and hemolytic anemias and haemoglobinopathies (32.7%) wwere the commonest etiological types and Iron deficiency anemia (88.5%) was the commones cause of nutritional anemia. Out of 557 children 56.2% were moderately anemic followed by 25.8% who were severly anemic. Palmar pallor was found to be 81.5% sensitive and conjunctival pallor was 89.9% sensitive. Palmar pallor was most sensitive for severe anemia (98.6%) followed by moderate (80.2%) anemia. *Conclusion:* Nutritional anemia was the commonest etiological type of anemia, with Iron deficiency being the most frequently observed sub type, followed by hemoglobinopathies and hemoatological anemia, which was mainly constituted by sickle cell anemia and thalassemia.

Clinical features like easy fatigability, irritability, lack of concentration, breathlessness, headache and palpitations were frequently observed in children with moderate anemia, where as children with mild anemia were relatively symptom free.

Palmar pallor was found to be 81.5% sensitive and conjunctival pallor was 89.9% sensitive, sensitivity was in more in severe forms of anemia hence we conclude that palmar pallor and conjunctival pallor can be used as a simple diagnostic tool for moderate and severe form of anemias, and reduce the morbidity and mortality associated with anemia.

Keywords: Anemia; Iron Deficiency; Palmar Pallor; Severe Anemia; Hemolytic Anemia; Hemoglobinopathies.

Introduction

Anemia is a condition in which the number of red blood cells (and consequently their oxygen-carrying capacity) is insufficient to meet the body's physiologic needs[1]. Anemia is defined as a reduction of the hemoglobin concentration or red blood cell (RBC) volume below the range of values occurring in healthy persons. "Normal" hemoglobin and hematocrit

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(packed red cell volume) vary substantially with age and sex.[2]Anemia is related to impaired physical growth and mental development[3]. It is also associated to a higher risk of infant and child mortality, particularly when it co-exists with malnutrition and other risk factors[4].Symptoms may include weakness, fatigue, difficulty concentrating, or poor work productivity.[5]Children may have issues with mental and motor development.[67] Some may present with irritability or pica (in iron deficiency), jaundice (in hemolysis), shortness of breath, or palpitations, tachypnea, tachycardia, and heart failure.[8]

Anemia is extremely common in Indian children. According to the National Family Health Survey (NFHS-3)[9], (2003-05) nearly 70 percent of children are anemic, including 26 percent who are mildlyanemic (10.0-10.9 g/dl), 40 percent who are moderately anemic (7.0-9.9 g/dl), and 3 percentwho are severely anemic (less than 7.0 g/dl). (MOHFW, 1998-1999)

Anemias may be classified on the basis of physiology ormorphology. Causes of anemia due to functional disturbances:

1. Disorders of effective red cell production, in which thenet rate of red cell production is depressed. This canbe due to disorders of erythrocyte maturation, inwhich erythropoiesis is largely ineffectual, or to anabsolute failure of erythropoiesis.
2. Disorders in which rapid erythrocyte destructionor red cell loss is primarily responsible for theanemia.

Anemias may also be classified on the basis of redcell size and then further subdivided according to red cellmorphology i.e. microcytic, normocytic, and macrocyticanemias.[10]

Although the primary cause is iron deficiency (approximately 50%). More frequently it coexists with a number of other causes, such as malaria, parasitic infection, nutritional deficiencies, and haemoglobinopathies.[11,12] Other causes of anemia include othermicronutrient deficiencies (e.g. folate, riboflavin and B12), acute and chronic infections (e.g. malaria, cancer, tuberculosis and HIV), and inherited or acquired disorders that affect haemoglobin synthesis, red blood cell production or red blood cell survival (e.g. haemoglobinopathies).[13,14]

In 2002, iron deficiency anemia (IDA) was considered to be among the most important contributing factors to the global burden of disease.[15] Anemic women and their infants are at greater risk of dying during the perinatal period; children's mental and physical development is delayed or impaired by iron deficiency.[16]

Megaloblastic anemia is fairly common in Pediatric population of the underdeveloped countries.It is a macrocytic anemia caused by thedeficiency of folic acid, vitamin B12, or both.Vitamin B12 and/or folic acid deficiency arethe commonest causes of megaloblastic anemia.[17]Neurological deficits are also associated with vitaminB12 deficiency.[18]

In India, the gene frequency of hemoglobinopathies is 4.2%, with a population over 1 billion and over 12000 infants born each year have a clinically significant hemoglobinopathies.[19,20] Within this overall disease classification, a 1989 WHO Working Group on guidelines for the control of haemoglobin disorders estimated a 3.9% carrier frequency for â-thalassemia in India, encompassing all types of â-thalassemia trait.[21]Sickle cell disease prevalence has ranged from 9.4 to 22.2% in endemic areas.Based on the surveys, prevalence of sickle gene is found to be 0-18% in north eastern India, 0-33.5% in western India, 22.5-44.4% in central India and 1-40% in southern India and the gene frequency of Hb-S varies between 0.031- 0.41.[22]

The autoimmune hemolytic anemias (AIHA) have an incidence estimated to be between 0.6 and 3 cases per 100,000 persons.[23]

Anemia is a common presentation in patientswith newly diagnosed childhood acute lymphoblasticleukemia (ALL).[24,]

Acquired aplastic anemia usually has anautoimmune basis. In some cases radiation, medicaldrugs and chemicals, and viruses cause depletion ofhematopoietic stem cells by direct toxicity.[26]Anemiaof chronic disorder is a mild to moderate anemia thatoccurs in many infections and inflammatory disorders.It is a frequent finding in chronic kidneyinsufficiency(CKI), dialysis patients, congestive heart failure (CHF) and renal transplantation.[27]

The initial laboratory tests should include determination of a complete blood count, measurementof erythrocyte porphyrin and serum ferritinconcentration, supravital staining of erythrocytes, hemoglobinelectrophoresis, a screening test for the presenceof unstable hemoglobins, a direct and indirect Coombstest, a screening test for glucose-6-phosphate dehydrogenase deficiency, and examination of bone marrow.[10]The use of the mean corpuscular volume to classify the anemia as microcytic, normocytic or macrocytic is a standard diagnostic approach.[28]

Palmar creases give a clue to the degree of anemia. When they are as pale as the surrounding skin,the

patients usually have severe anemia, a hemoglobin (Hb) level <7 g/dL.[29]

This study was done to know the etiology, clinical manifestations of anemia, and its correlation with the hematological profile so that the problem can be tackled in a better way and steps can be taken to minimize the suffering of the children.This study also tested the accuracy of palmar pallor compared to Hemoglobin levels which is also used in IMNCI programme.[30]

Aims and Objectives

Aim

"To study the etiology and clinico-haematological co-relation of anemia in Pediatric age group."

Primary Objectives

1. To classify anemia etiologically based on clinical features and investigations.
2. To correlate clinical features of anemia with level of haemoglobin.
3. To correlate color of palm and palmar creases (method adopted by IMNCI) with level of haemoglobin.

Materials and Method

Place of Study

Department of Pediatrics, Acharya Vinoba Bhave Rural Hospital, Sawangi (M), Wardha.

Study Population

Children between 6month to 15yrs of age

Study Design: Cross Sectional analyticalstudy

Duration of the Study: 1st August 2013 TO 31st July 2015.

Inclusion Criteria

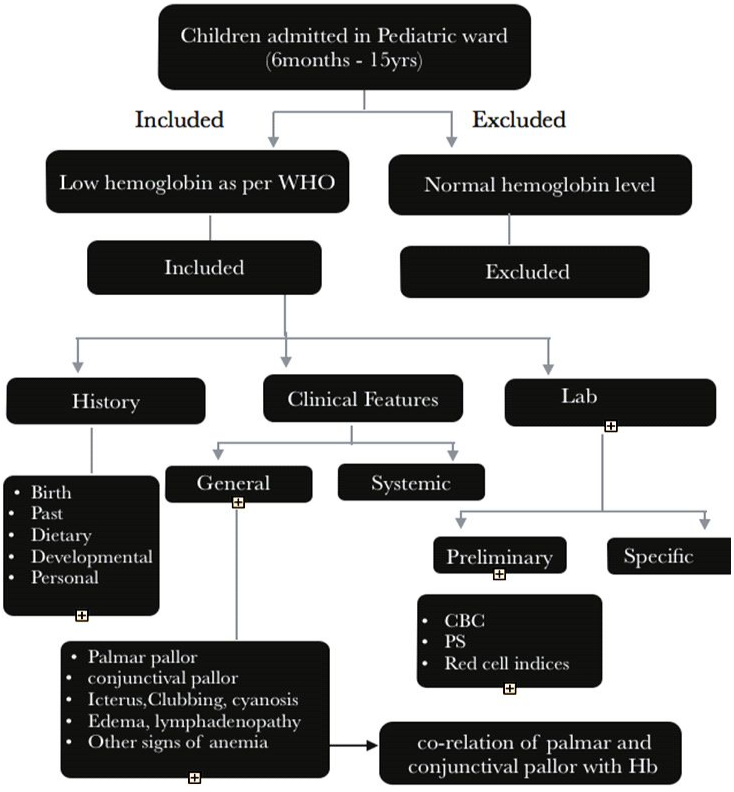
- Children between 6 months to 15 years admitted in Pediatric ward whose hemoglobin levels were below the WHO cut off for age.

Exclusion Criteria

- Parents refusing to enter the study and not willing to give consent.
- Patients admitted in the ICU.

Sample Size: 557 children with anemia.

Flow Chart



Important Definitions

Anemia was Defined as per WHO[31]

The patients were divided into 3 categories: Pre school (6mo-59months), School going (5-11.99yrs) and adolescent (12-15yrs), and the hemoglobin cut off were taken as follows:

Severity of Anemia was Defined According to WHO [31]

The anemia was further classified as mild, moderate and severe according to WHO as follows:

Age	Hemoglobin levels
6months – 4.99 years	<110
5 years – 11.99 years	<115
12-14.99 years	<120

Hemoglobin in grams per liter

Age	Mild	Moderate	severe
6months – 4.99 years	100-109	70-99	Lower than 70
5 years – 11.99 years	110-114	80-109	Lower than 80
12-14.99 years	110-119	80-109	Lower than 80

Hemoglobin in grams per liter

Normal Values

- MCV(80-100fl)Error! Bookmark not defined.:MCV <80 was considered to be microcytic and >100 as macrocytic.
- MCHC (31-35g/dL)Error! Bookmark not defined.: The MCHC is a measure of cellular hydration status. A high value (>35 g/dL) is characteristic of spherocytosis and a low value is suggestive of iron deficiency anemia.
- MCH: (26-34pg)Error! Bookmark not defined.: The MCH represents the mean mass of hemoglobin in the RBC and is ex- pressed in the mass unit, picograms.
- RDW (11.5-14.5%) [10]:RDW represents the coefficient of variation of the red blood cell volume distribution (size) and is expressed as a percentage[32].
- Serum Iron (50-120 mcg)Error! Bookmark not defined.: Serum iron was done were ever possible, in the absence of this test, we diagnosed IDA on the basis of red cell indices. Serum iron estimation as a measure of irondeficiency has serious limitations.
- Serum ferritin (upto 15 ng/ml)[2]: A low serum ferritin level is a very specific and early indicator of iron deficiency. Plasma iron concentrationsfall as iron is depleted
- TIBC (250-425 mcg/dl): The availability ofplasma iron binding sites, or TIBC, increases as ironstores fall. TIBC > 490 mcg/dl was considered as raised. Error!

Bookmark not Defined

- Transferritin saturation[2]: >15% considered normal.
- Serum folic acid 5-20 ng/ml: level below 4ng/ml[33] was considered as deficiency
- Serum B12: 200-900pg/ml:Error! Bookmark not defined Levels below 200pg/ml were considered as deficiency.

Iron Deficiency Anemia was Classified on the Basis of

- Low hemoglobin
- Low MCV (less than 80fl)
- Increased RDW (more than 14.5%)
- PS showing microcytic and hypochromic picture or both also anisocytosis and poikilocytosis were taken into consideration.
- *Wherever Possible-*
    - Serum Iron <50mcg
    - Transferritin saturation < 15%
    - TIBC >490 mcg/dl
    - Serum ferritin<15ng/ml was done

Megaloblastic Anemia was Classified on the Basis of the Following

- Low hemoglobin as defined earlier
- MCV > 100fl
- RDW> 14.5%
- PS showing macrocytes and or hypersegmented neutrophils
- Presence of thrombocytopenia and or leukopenia
- Serum B12 <200pg/ml or Serum folic acid < 5ng/ml
- *Whenever Possible* bone marrow showing macrocytes.

Aplastic Anemia was Diagnosed When

- Anemia with normal RDW
- PS: normocytic normochromic
- Leukopenia (TLC<4000 / cumm)
- Thrombocytopenia (platelets < 1.5lakhs / cumm)
- Bone marrow was considered *diagnostic*: hypocellular marrow
- Iron study if done: overload

Thalassemia

- Low hemoglobin
- PS: microcytic, hypochromic, increased normoblast.
- Reticulocyte count: decreased (<1%) in thalassemia major and elevated (3-6%) in thalassemia intermedia.
- Low MCV (<80fl)
- Normal RDW (11.5-14.5%)
- Osmotic fragility: reduced fragility
- *Diagnostic*: High performance liquid chromatography

  HbF: >20%

  HbA2: >3.5

  HbA: 0-80% (depending on genotype)

Sickle Cell Anemia/Trait

- Low hemoglobin
- PS: Normocytic, normochromic, presence of crescent shaped cells, poikilocytosis, anisocytosis, nucleated RBCs
- Positive sickling test
- Hb electrophoresis showing SS / AS pattern was considered diagnostic
- HPLC: with e / o HbS

Hemolytic Anemia

- Low hemoglobin
- Positive coomb's test
- Normal platelets and Total leucocyte count
- Elevated reticulocyte count >2%
- High RDW (>14.5%)

Result and Observation

In the present study, the main aim was to study the etiology and clinic-hematological co-relation of anemia in pediatric age group and we a total studied 557 anemic children admitted in Pediatrics ward aged between 6months to 15 years.

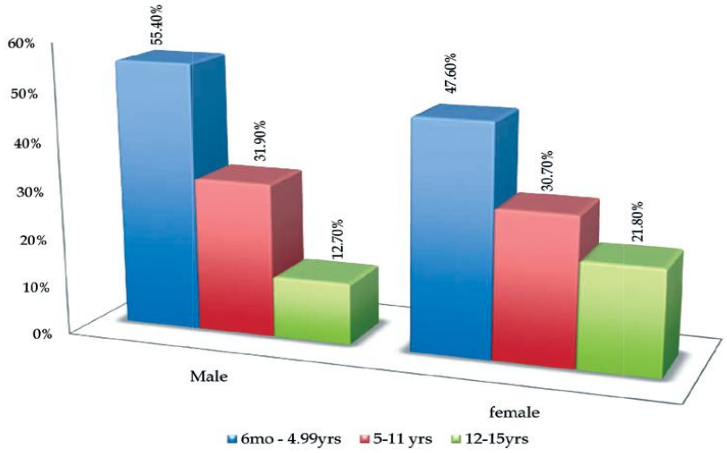


Chart 1: Age and sex distribution

Table 2(a): Clinical symptoms and severity of anemia

	Mild (100)	Moderate (313)	Severe (144)	Total (557)	P-value
Easy fatigability	13 (9%)	108 (74.5%)	24 (16.5%)	145	0.0003,s
Irritability	11 (7.9%)	97 (69.8%)	31 (22.3%)	139	0.002,s
Lack of concentration	10 (10.2%)	50 (51%)	38 (38.8%)	98	0.010,s
Breathlessness	9 (10.1%)	49 (55.1%)	31 (34.8%)	89	0.040,s
Headache	11 (12.8%)	45 (52.3%)	30 (34.9%)	86	0.13,ns
Palpitations	5 (6.6%)	47 (62.7%)	23 (30.7%)	75	0.030,s
Anorexia	9 (13.8%)	41 (63.1%)	15 (23.1%)	65	0.63,ns
Insomnia	3 (5.6%)	23 (42.6%)	28 (51.9%)	54	0.20,ns
Giddiness / syncope	3 (6%)	30 (60%)	17 (34%)	50	0.053,ns
Tinnitus	0 (0)	7 (58.3%)	5 (41.7%)	12	0.12,ns
Menstrual abnormality	0 (0)	0 (0)	1 (100%)	1	0.36,ns

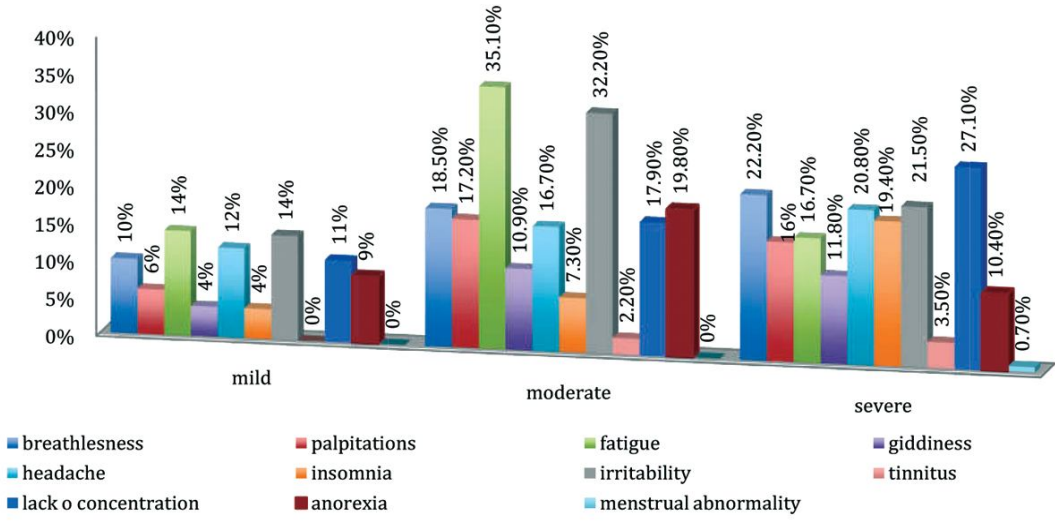


Chart 2 (a): Clinical symptoms and severity of anemia

In our study we found that males (59.6%) were more than females (40.4%), the ratio being 1.5:1. Majority of the children 291 (52.2%) were in age group of 6months to 5 years followed by 175 (31.4%) and 91 (16.3%) in age group 5-11years and 12-15years respectively. (Table 2)

Easy fatigability (26%) and irritability (25%) were the most common complaints while menstrual abnormality (0.2%) and tinnitus (2.2%) were less commonly observed feature in our study.Easy fatigability 108 (74.5%), irritability 97 (96.8%), lack of concentration 50 (51%), Breathlessness 49 (55.1%), Headache 45 (52.3%), palpitation 47 (62.7%), anorexia 41 (63.1%), insomnia 23 (42.6%), giddiness / syncope 30 (60%) and Tinnitus (58.3%) was seen more commonly in moderate anaemia. Menstral abnormality 1 (100%) was only seen in severe anemia

Table 2(b): Age wise clinical signs

	6mo-4.99yrs 289	5-11yrs 77	12-15yrs 91	Total (557)	P-Value
Easy fatigability	61 (42.1%)	54 (37.2%)	30 (20.7%)	145	0.004,s
Irritability	75 (54%)	48 (34.5%)	16 (11.5%)	139	0.0001,s
Lack of concentration	22 (22.4%)	55 (56.1%)	21 (21.5%)	98	0.0001,s
Breathlessness	27 (30.4%)	36 (40.4%)	26 (29.2%)	89	0.18,ns
Headache	19 (22.1%)	40 (46.5%)	27 (31.4%)	86	0.0001,s
Palpitations	16 (21.3%)	41 (54.7%)	18 (24%)	75	0.0001,s
Anorexia	32 (49.2%)	16 (24.6%)	17 (26.2%)	65	0.0002,s
Insomnia	6 (11.1%)	26 (48.2%)	22 (40.7%)	54	0.0001,s
Giddiness / syncope	20 (40%)	18 (36%)	12 (24%)	50	0.04,s
Tinnitus	0 (0)	7 (58.3%)	5 (41.7%)	12	0.0001,s
Menstrual abnormality	0 (0)	0 (0)	1 (100%)	1	0.0001,s

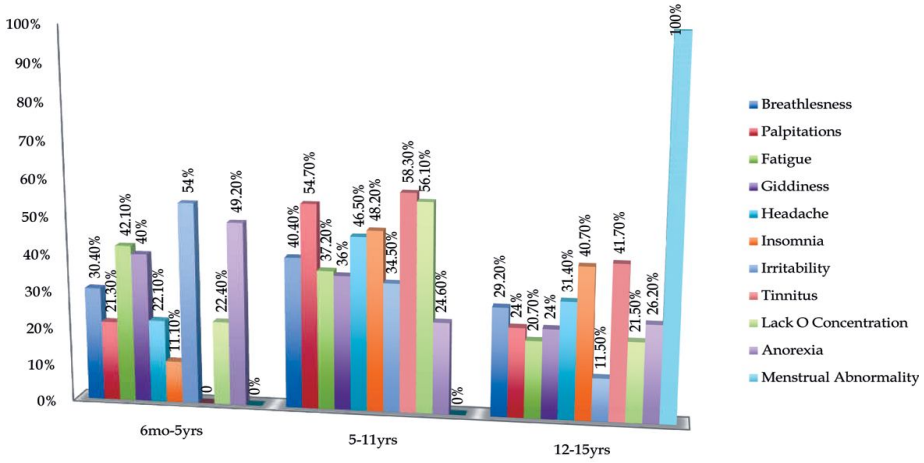


Chart 2(b): Clinical symptoms and age

Irritability (54%) was the commonest feature in children between 6mo-5years, followed by anorexia (49.2%), easy fatigability (42.1%) and syncope / giddiness (40%). Symptoms like lack of concentration (56.1%), palpitations (54.7%), Headache (46.5%), Insomnia (40.7%) and breathlessness (40.4%), were prevalent in children falling between 5-11 years of age. Adolescents were less symptomatic.

Table 2(c): Clinical symptoms and etiology of anemia

	6-59 months (291)	5-11yrs (175)	12-15yrs (91)	Total	P-VALUE
Palmar pallor	228 (50.2%)	146 (32.2%)	80 (17.6%)	454	0.0001,S
Icterus	21 (28.4%)	31 (41.9%)	22 (29.7%)	74	0.075,NS
Knuckle pigmentation	44 (64.7%)	11 (16.2%)	13 (19.1%)	68	0.0001,S
Glossitis	32 (49.3%)	19 (29.2%)	14 (21.5%)	65	0.0017,S
Hepatosplenomegaly	26 (51%)	16 (31.4%)	9 (17.6%)	51	0.0001,S
Platynychia / koilonychia	26 (66.7%)	6 (15.4%)	7 (17.9%)	39	0.0001,S
Angular stomatitis	13 (38.2%)	14(41.2%)	7 (20.6%)	34	0.32,NS

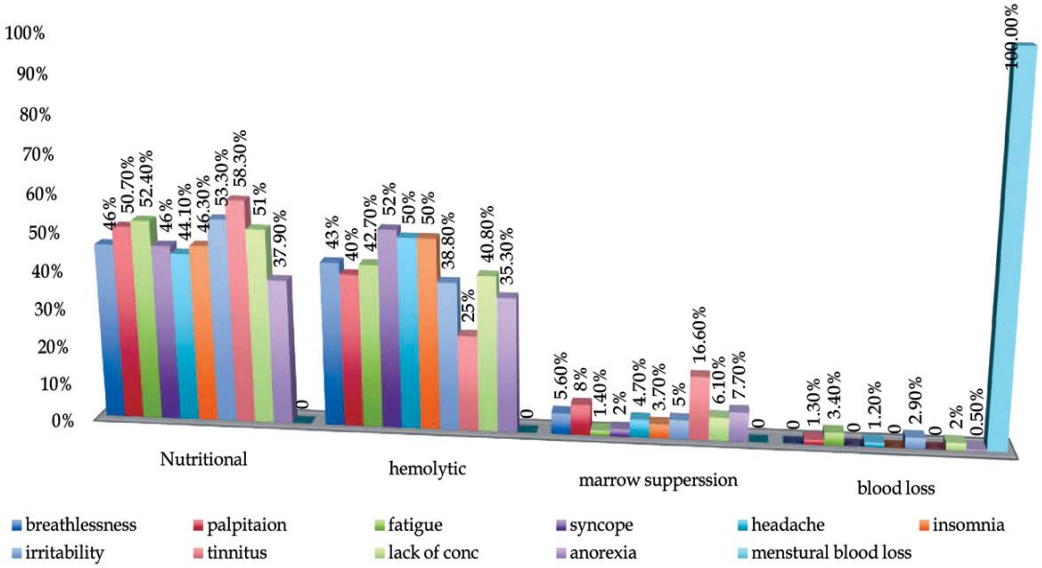


Chart 2(c): Clinical symptoms and etiology of anemia

Palpitation (50.7%), fatigue (52.4%), irritability (53.3%), lack of concentration (51%) and anorexia (55.3%) were common among children with nutritional anemia.Where else breathlessness (48.4%), Syncope / giddiness (52%), headache (50%) and insomnia (27%) were frequently seen in children with hemolytic anemia.

Table 3(a): Clinical signs and severity of anemia

	Mild	Moderate	Severe	Total	P-VALUE
Palmar pallor	61(13.4%)	251(55.3%)	142(31.3%)	454	0.0001,S
Icterus	4(5.4%)	41(55.4%)	29(39.1%)	74	0.0001,S
Knuckle pigmentation	13 (19.1%)	38(55.9%)	17(25%)	68	0.0001,S
Glossitis	15(23.1%)	33(50.8%)	17(26.1%)	65	0.0001,S
Hepatosplenomegaly	8(15.7%)	34(66.7%)	9(17.6%)	51	0.0001,S
Platynychia / koilonychia	8(20.5%)	18(46.1%)	13(33.4%)	39	0.0009,S
Angular stomatitis	7(20.5%)	17(50%)	10(29.4%)	34	0.0001,S

Table 3(b): Age wise clinical signs

	6-59 months (291)	5-11yrs (175)	12-15yrs (91)	Total	P-VALUE
Palmar pallor	228 (50.2%)	146 (32.2%)	80 (17.6%)	454	0.0001,S
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Hepatosplenomegaly	26 (51%)	16 (31.4%)	9 (17.6%)	51	0.0001,S
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Angular stomatitis	13 (38.2%)	14(41.2%)	7 (20.6%)	34	0.32,NS

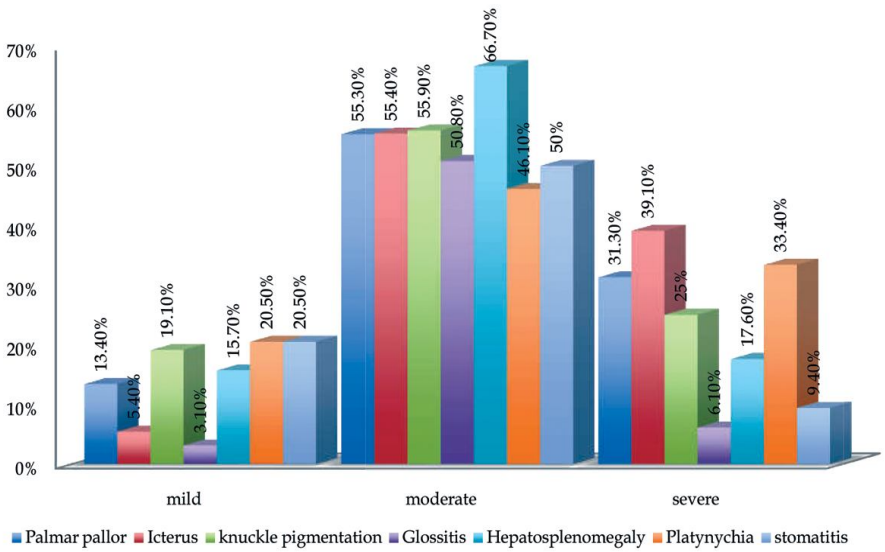


Chart 3(a): Clinical signs and severity of anemia

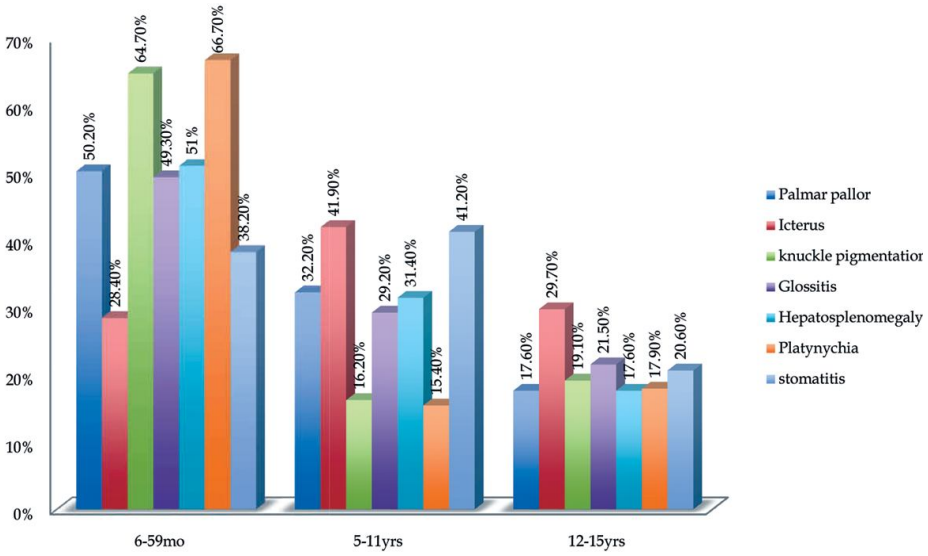


Chart 3(b): Age wise clinical signs

In the study out of 557 children 454(81.5%) had palmar pallor, 74 (13.2%) children had icterus, 68 (12.2%) had knuckle pigmentation, 65% (11.7%) had glossitis. Platynychia was observed only in 30 (7%) of the children.

The clinical signs were seen more commonly in moderate type of anemia followed by severe and mild. 251 (55.3%) children with palmar pallor had moderate anemia, followed by severe (31.3%) and mild anemia (13.4%). Similarly Icterus was most commonly seen in children with moderate anemia (55.4%) then severe (39.1%) and mild anemia (5.4%). Knuckle pigmentation was observed 55.9% in children with moderate anemia, severe (25%) and mild (19.1%). Glossitis observed in 50.8% in moderate anemia, 26.1% in cases of severe anemia and 23.1% of cases in mild anemia. Children with moderate anemia had highest number of clinical signs like hepatosplenomegaly (66.7%), Platynychia (46.1%), angular stomatitis (50%), followed by severe anemia 17.6%, 33.4% and 29.4% respectively and mild anemia 15.7%, 15.7% and 20.5% respectively.

Palmar pallor was most frequently seen in pre-school children (50.2%), followed by school going children (32.2%) and adolescents (17.6%). Knuckle pigmentation (64.7%), glossitis (49.3%), hepatosplenomegaly (51%), Platynychia (66.7%) and angular stomatitis (38.2%) were also most commonly observedseen in pre-school children.

Icterus was observed 41.9% in school going children, 29.7% in adolescents and 28.4% in pre school children.

Out of 124 children with hepatomegaly, 76 (61.3%)children were moderately anemic, 30 (24.2%)

were severly anemic and 18 (14.5%) were mildly anemic. Out of 114 cases of spenomegaly 64 (56.1%) children were moderately anemic, 32 (28.1%) were severely anemic and 18 (15.8%) were mildy anemic.

34 (66.7%)children with moderate anemia, 9 (17.6%) children with severe anemia and 8 (15.7%) children with mild anemia had hepatosplenomegaly (51).

Table 4: Organomegaly and etiology

	Nutritional	Hemolytic	Bone marrow suppression	Blood loss	Total	P-VALUE
Hepatomegaly	51 (41.1%)	65 (52.4%)	6 (4.8%%)	2 (1.7%)	124	0.0001,S
Splenomegaly	43 (37.7%)	62 (54.3%)	6 (5.3%)	3 (2.7%)	114	0.0001,S
Hepatosplenomegaly	23 (45.1%)	24 (47.1%)	2 (3.9%)	2 (3.9%)	51	0.0001.S

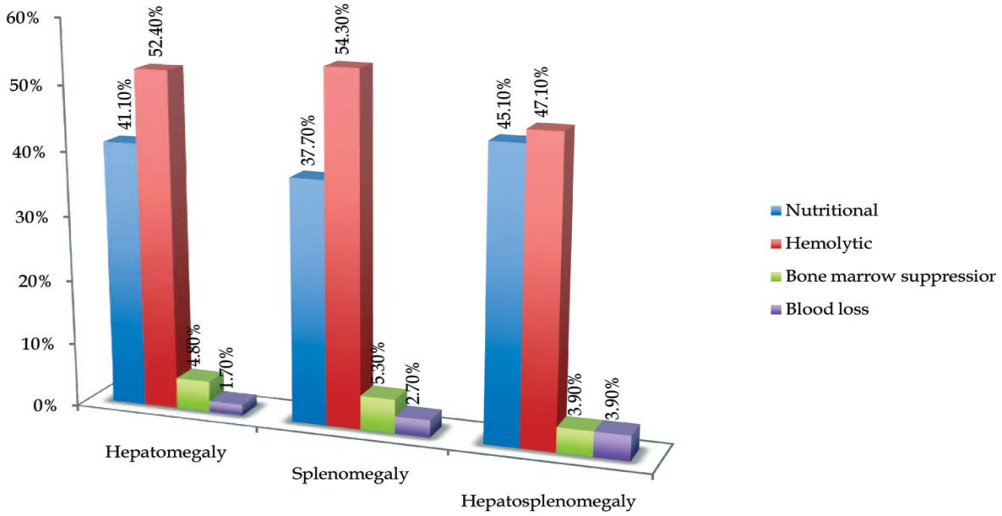


Chart 4: Organomegaly and etiology

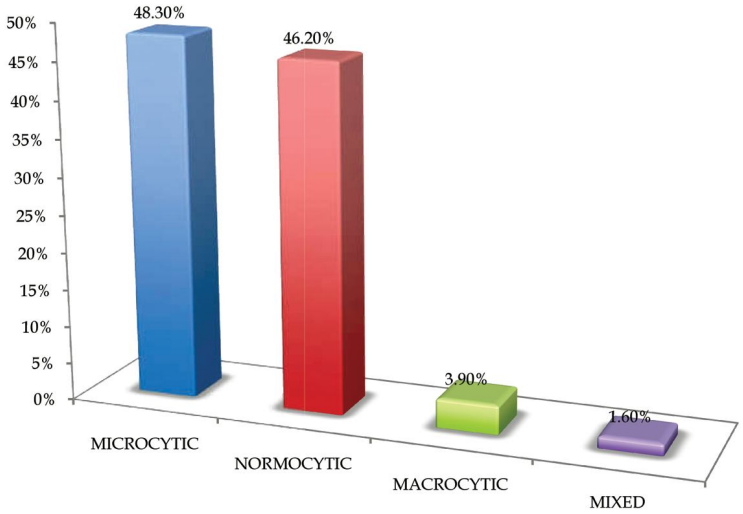


Chart 5: Morphological classification of anemia

Out of 124 cases of hepatomegaly 51 (41.1%) had nutritional anemia, 65 (52.4%) had hemolytic anemia, 4.8% had anemia due to bone marrow suppression and 1.7% had anemia due to blood loss. Out of 114 cases with splenomegaly 43 (37.7%) belonged to nutritional anemia group, 62 (54.3%) belonged to hemolytic anemia, 6 (5.3%) belonged to anemia due to marrow suppression and 3 (2.7%) had anemia due to blood loss. Hepatosplenomegaly was common in children with hemolytic (47.1%) and nutritional anemia (45.1%) and observed to be in 3.9% children with anemia due to blood loss and anemia due to

marrow suppression both.

Microcytic anemia (48.3%) was found to be the most prevalent type of morphological anemia, followed by normocytic (46.2%), macrocytic (3.9%) and mixed (1.60%) type.

Table 6: Etiological classification of anemia age wise

	Nutritional	Hemolytic and hemoglobinopathies *	Bone marrow failure	Blood loss	Total	p-value
6-59MONTHS	216 (74.2%)	60 (20.7%)	10 (3.4%)	5 (1.7%)	291	0.0001,S
5YRS - 11YRS	74 (42.3%)	80 (45.7%)	17 (9.7%)	4 (2.3%)	175	0.0001,S
12YRS-15YRS	41 (45%)	43 (47.3%)	4 (4.4%)	3 (3.3%)	91	0.0001,S
TOTAL	331 (59.6%)	183 (32.7%)	31(5.6%)	12(2.1%)	557	

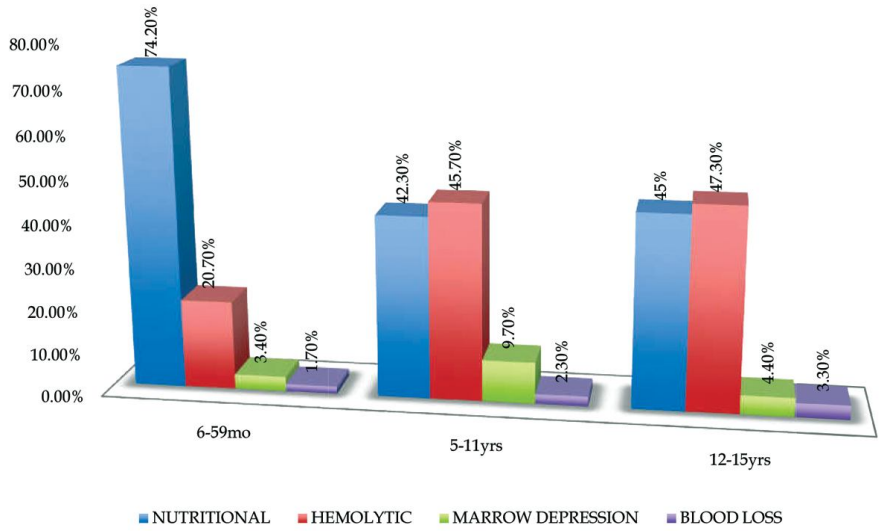


Chart 6: Etiology of anemia and age wise

Nutritional anemia was the most common anemia (59.6%), followed by hemolytic anemia (32.7%), anemia due to bone marrow suppression (5.6%). We observed nutritional anemia (74.2%) to be most common in children aged between 6months to 5years, followed by hemolytic anemia (20.7%), anemia due to bone marrow suppression (3.4%) and anemia due to blood loss(1.7%).Hemolytic anemia including hemoglobinopathies (45.7%) was the second common type, followed by nutritional anemia (42.3%), bone marrow suppression(9.7%) and anemia due to blood loss (2.3%) in children between 5-11years of age and similar result was observed in children between 12-15years of age.

Amongst nutritional anemia, iron deficiency anemia was 88.5% prevalent, followed by megaloblastic, 6.7% and mixed 4.8% (iron deficiency with megaloblastic)

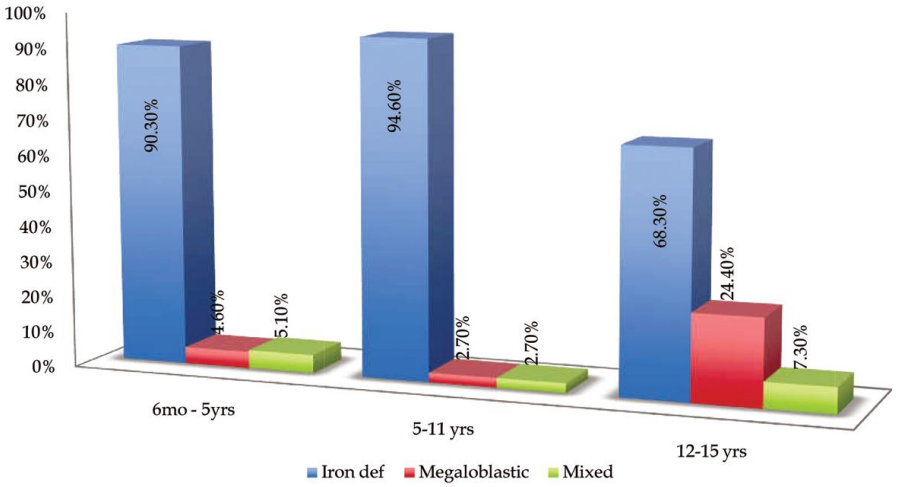


Chart 7(b): Age wise classification of nutritional anemia

Table 7(b): Age wise classification of nutritional anemia

	Nutritional	Hemolytic	Bone marrow suppression	Blood loss	Total	P-VALUE
Hepatomegaly	51 (41.1%)	65 (52.4%)	6 (4.8%%)	2 (1.7%)	124	0.0001,S
Splenomegaly	43 (37.7%)	62 (54.3%)	6 (5.3%)	3 (2.7%)	114	0.0001,S
Hepatosplenomegaly	23 (45.1%)	24 (47.1%)	2 (3.9%)	2 (3.9%)	51	0.0001.S

Out of 293 cases of iron deficiency anemia 66.5% were pre-school children, 23.9% were school going and 9.6% were adolescents. Megaloblastic anemia was equally observed in pre-school (45.5%) and adolescents (45.5%) and 9% in school going children. Mixed anemia was found in 68.7% pre school children, 18.8% adolescents and 12.5% school going children Out of 22 children with megaloblastic anemia 72.7% had vitamin B12 deficiency, 22.7% had folate deficiency and 1 patient (4.6%) had fanconis anemia.

Table 8(a): Classification of megaloblastic anemia based on the cause

	Numbers	Percentage
Vitamin B12 deficiency	16	72.7%
Folate Deficiency	5	27.3%
Fanconi's anemia	1	100%
	22	100%

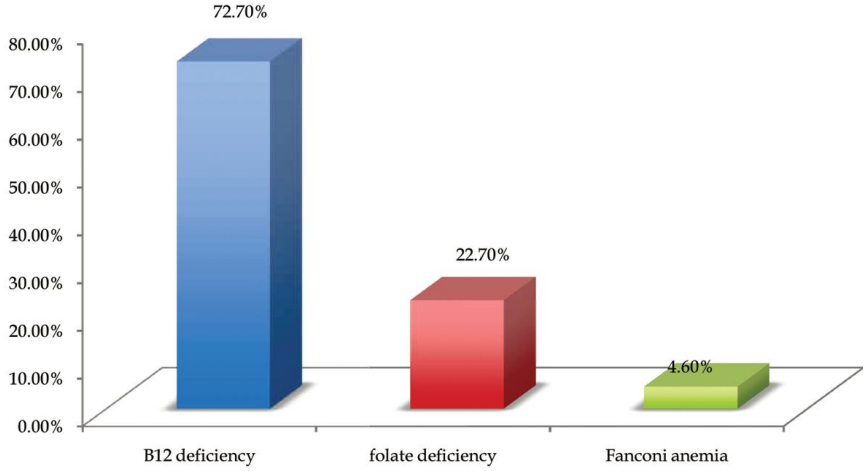


Chart 8(a): Classification of megaloblastic anemia based on the cause

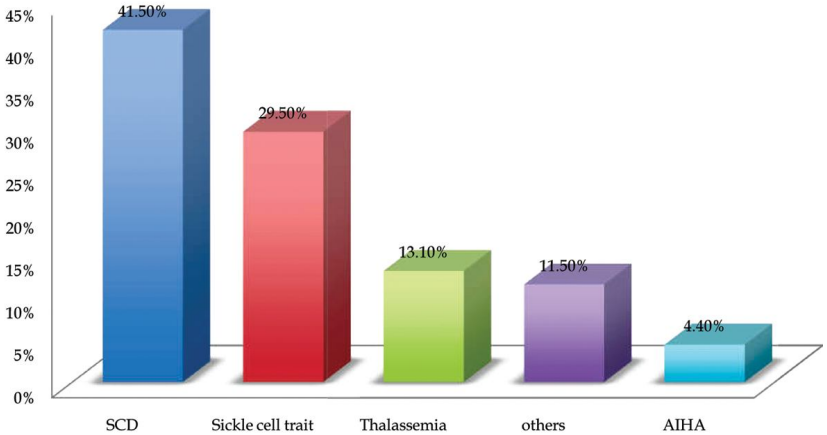


Chart 8(b): Classification of hemolytic anemias and hemoglobinopathies

Table 8(b): Classification of hemolytic anemias and hemoglobiniopathies

Type of anemia			Frequency	Percentage
Sickle cell anemia			76	41.5%
Sickle cell trait			54	29.5%
Thalassemia			24	13.1%
AIHA			8	4.4%
Others	HUS	2		
	Malaria	4		
	DIC	5	21	11.5%
	Hepatitis	7		
	Wilsons	3		
Total			183	100%

Table 9(a): Age wise classification of hemolytic anemias and hemoglobinopathies

	Sickle cell anemia	Thalassemia	Sickle cell trait	AIHA	Others*	Total	P-VALUE
6-59months	24 (40%)	12 (20%)	17 (28.3%)	4 (6.7%)	3 (5%)	60	0.0001,S
5 – 11 years	32 (40%)	5 (6.3%)	28 (35%)	4 (5%)	11(13.7%)	80	0.0001,S
12-15 years	20 (46.6%)	7 (16.2%)	9 (20.9%)	0 (0)	7 (16.2%)	43	0.0001,S
Total	76	24	54	8	21 (11.5%)	183	
	(41.5%)	(13.1%)	(29.5%)	(4.4%)			

*Others: DIC, hepatitis, HUS, Wilsons and malaria.

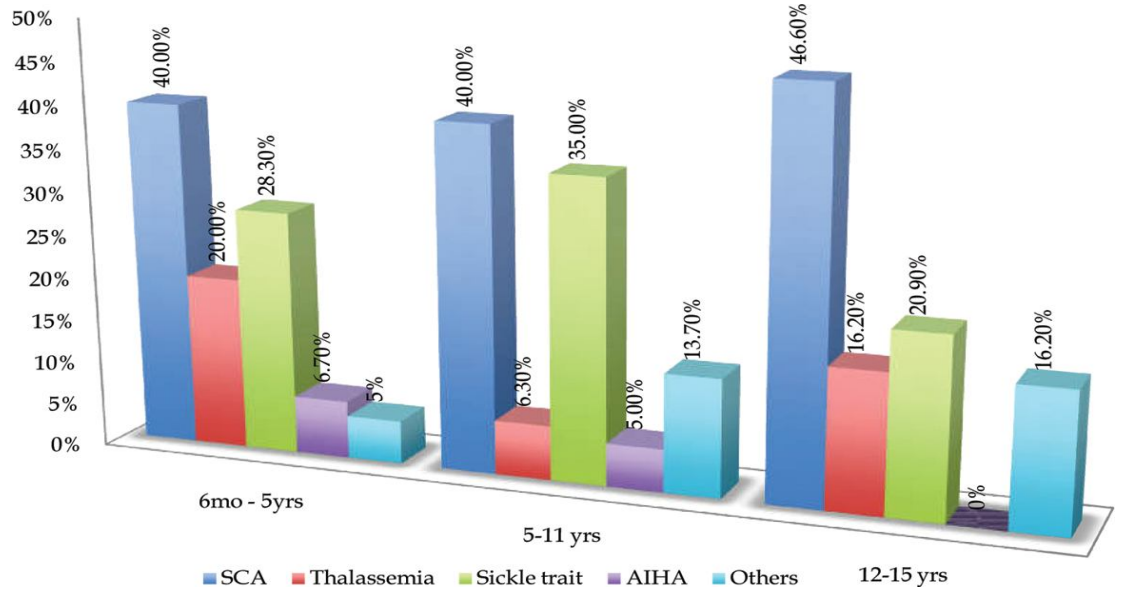


Chart 9(a): Age wise classification of hemolytic anemias and hemoglobinpathies

Amongst 183 children with hemolytic anemia, sickle cell anemia was 41.5% most prevalent, followed by sickle cell trait (29.5%), thalassemia (13.1%), other causes like HUS, DIC, Hepatitis, Wilsons and malaria (11.5%) and AIHA (4.4%).

In children aged 6-59 months sickle cell anemia (40%) was most common type, followed by sickle cell trait (28.3%), thalassemia (20%), AIHA (6.7%) and others (5%). Even amongst school going children sickle cell anemia (40%) was most common type, followed by sickle cell trait (35%), thalassemia (6.3%), others (13.7%) and AIHA (5%). Even adolescents followed same trend with 46.6% sickle cell anemia, 20.9% sickle cell trait, 16.2% thalassemia and others both and we did not find any case of AIIHA in adolescents.

Table 9(b): Severity of anemia as per age

	Mild	Moderate	Severe	Total	P-value
6-59MONTHS	83 (28.7%)	164 (56.8%)	42 (14.5%)	289	0.0001,S
5YRS - 11YRS	14 (7.9%)	101 (57.1%)	62 (35%)	177	0.0001,S
12YRS-15YRS	3 (3.3%)	48 (52.7%)	40 (44%)	91	0.0001,S
TOTAL	100	313	144	557	

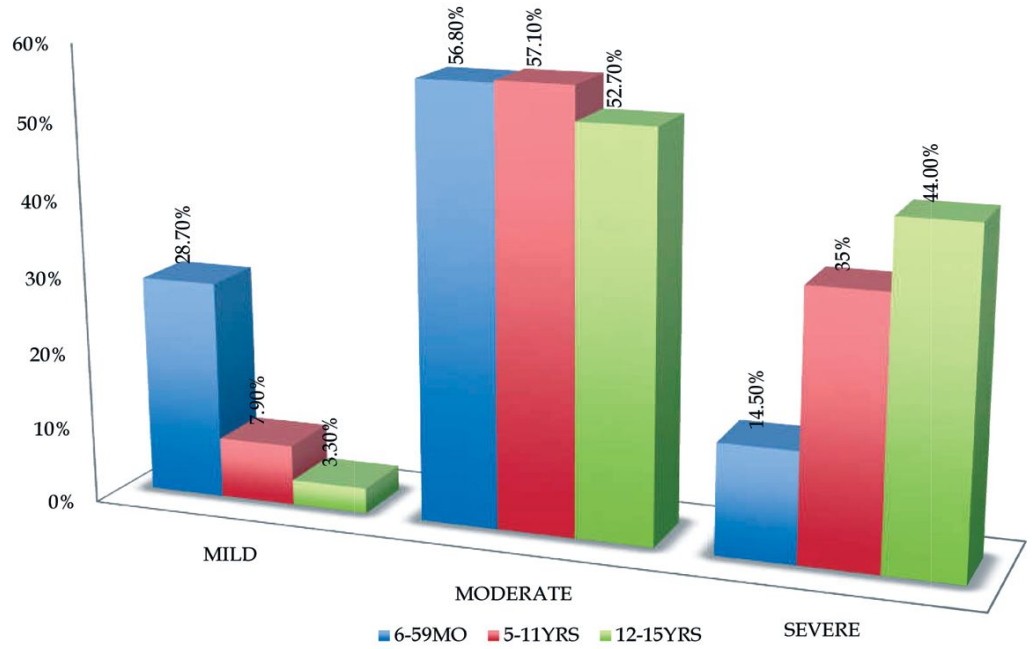


Chart 9(b): Severity of anemia as per age

Moderate anemia (56.2%) was found to be the most common type, followed by severe (25.8%).Out of 289 children in pre school age group 56.8% had mild anemia, 28.7% had mild anemia and 14.5% had severe anemia. Amongst 177 school-going children 57.1% had moderate anemia, 35% has severe anemia and 7.9% had mild anemia. Similarly 52.7% of adolescents had moderate anemia, 44% had severe anemia and 3.3% had mild anemia.

Table 10(a): severity and etiology of anemia

	Mild	Moderate	Severe	Total	P-Value
Nutritional	84 (25.4%)	187 (56.5%)	60 (18.1%)	331	0.0001,S
Hemolytic	9 (4.9%)	98 (53.6%)	76 (41.5%)	183	0.0001,S
Bone marrow suppression	5 (16.1%)	20 (64.5%)	6 (19.4%)	31	0.0001,S
Blood loss	2 (16.7%)	8 (66.1%)	2 (16.7%)	12	0.0001,S
	100	313	144	557	

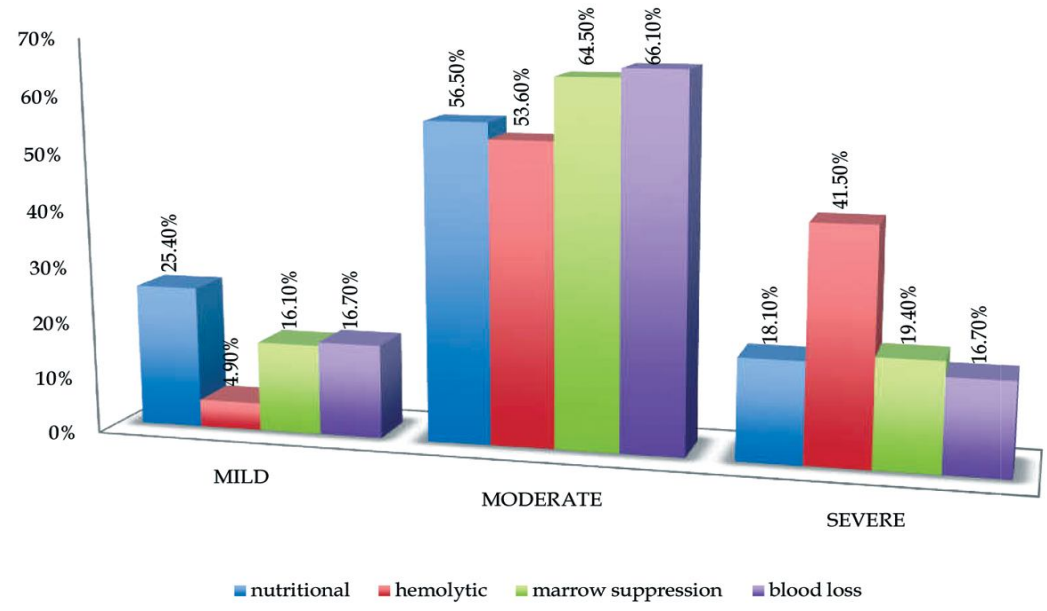


Chart 10(a): Severity and etiology of anemia

Out 331 children with nutritional anemia 187 (56.5%) had moderate anemia, 84 (25.4%) had mild anemia and 60 (18.1%) had mild anemia.

Where as amongst 183 children with hemolytic anemia 98 (53.6%) had moderate anemia, 76 (41.5%) had severe and 9 (4.9%) had mild anemia.

Amongst children with bone marrow suppression (31), moderate anemia was most prevalent (64.5%), followed by severe (19.4%) and mild (16.1%)anemia.

Moderate anemia was seen in 8 (66.1%) out of 12 children with anemia due to blood loss and 16.7% had mild and severe anemia.

Total 136 (24.4%) children out of 557 required blood transfusion.Out of 12 children with anemia due blood loss 10 (83.3%) blood transfusion. Out of 183 hemolytic anemias 93 (50.8%) required blood transfusion. Where as in children with nutritional anemias (6%) the need for transfusion was the least.

Table 10(b): Blood transfusion in anemia

	N	Frequency	Percentage
Hemolytic anemia	183	93	50.8%
Nutritional anemia	331	20	6%
Bone marrow suppression	31	13	41.9%
Blood loss *	12	10	83.3%
	557	136	24.4%

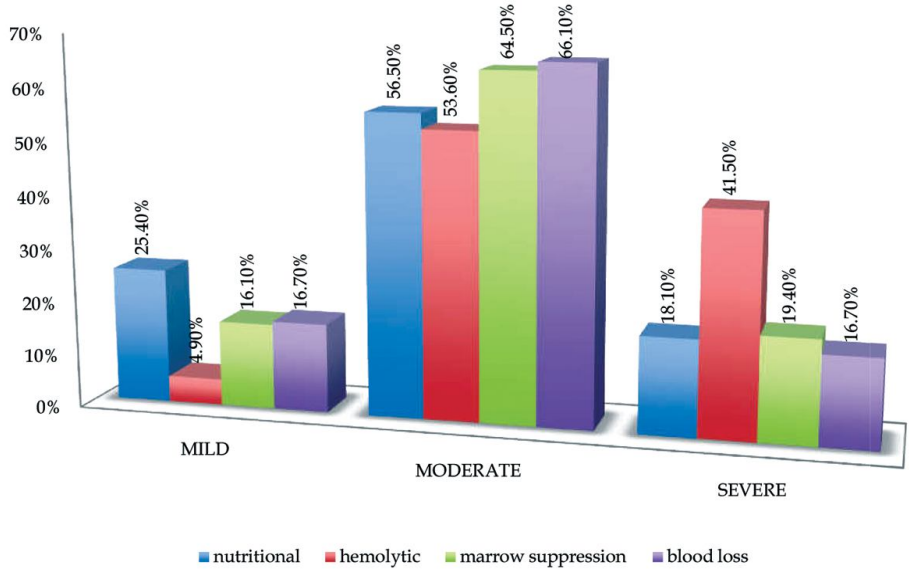


Chart 10(b): Blood transfusion in anemia

Total 136 (24.4%) children out of 557 required blood transfusion.Out of 12 children with anemia due blood loss 10 (83.3%) blood transfusion. Out of 183 hemolytic anemias 93 (50.8%) required blood transfusion. Where as in children with nutritional anemias (6%) the need for transfusion was the least.

All patients were examined for palmar pallor and out of 557 children 335 (60.1%) had pallor, 119 (21.4%) had severe pallor and in 21 patients the examination was non- conclusive due to icterus in some and dark skin color in others.

33% of patients with mild anemia had no pallor, 61% had detectable pallor.No severe palmar pallor was observed, and 6% of the patients had a non-conclusive result due to dark skin color and icterus.

In patients with moderate anemia 15.7% had no detectable palmar pallor, 72.5% had pallor, 7.7% had severe pallor and in 4.2% the examination was non – conclusive due to the same reasons.

Table 11(a): Palmar pallor in anemia

	Number	Percentage
No pallor	82	14.7%
Pallor	335	60.1%
Severe pallor	119	21.4%
Couldn't be detected	21	3.8%
Total	557	100%

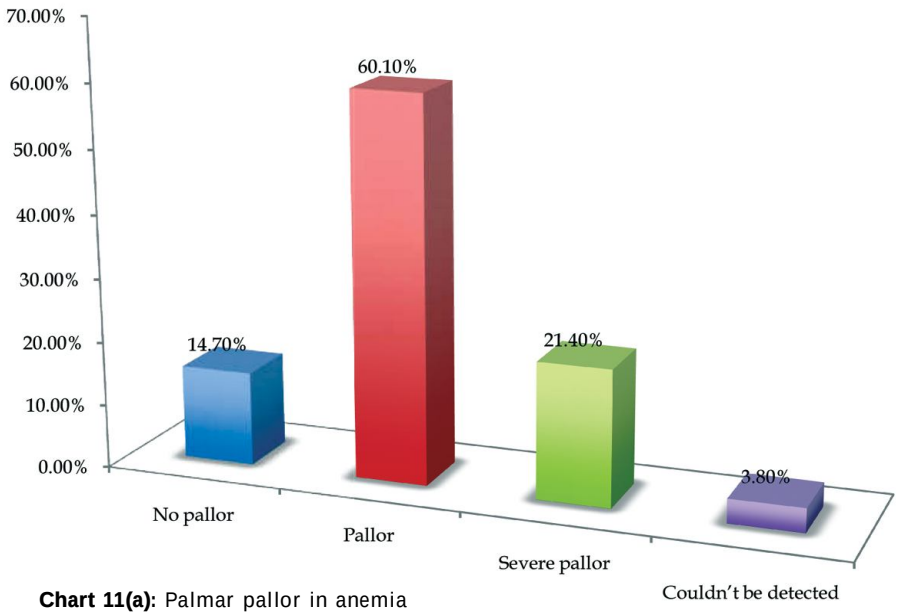


Chart 11(a): Palmar pallor in anemia

Table 11(b): Palmar pallor with severity of anemia

	No Pallor	Pallor	Severe Pallor	Not Detected	Total	P-Value
MILD	33 (33%)	61 (61%)	0 (0)	6 (6%)	100	0.0001,S
MODERATE	49 (15.7%)	227 (72.5%)	24 (7.7%)	13 (4.2%)	313	0.0001,S
SEVERE	0 (0)	47 (32.6%)	95 (66%)	2 (1.4%)	144	0.0001,S
TOTAL	82	335	119	21	557	

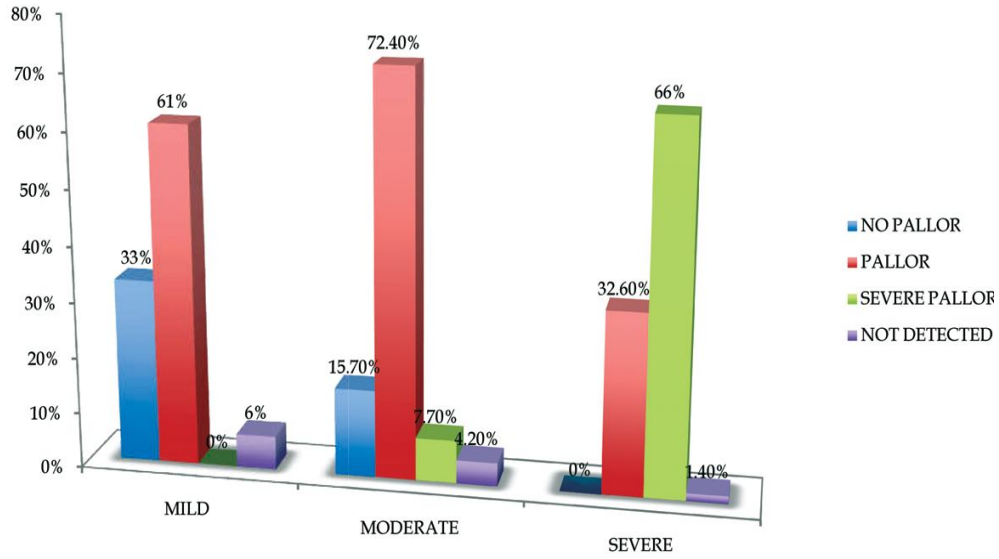


Chart 11(b): Palmar pallor and severity of anemia

Palmar pallor could detect anemia in all patients with severe anemia, except in those where it was non conclusive (1.4%). Pallor was observed in 32.6% and severe pallor was observed in 66% of all the severely anemic children.

Out of 331 children with nutritional anemia palmar pallor was observed in 224 (67.7%), severe pallor in 50 (15.1%), no pallor in 48 (14.5%) children and examination was non conclusive in 9 (2.7%) children.

Similarly in Children with hemolytic anemia (183), 80 (43.7%), 65 (35.5%), 27 (14.8%) had pallor, sever pallor and no pallor respectively.

77.4% patients with anemia due to marrow failure had pallor, 12.9% had no pallor and 16.7% had severe pallor.

58.3% patients with anemia due to blood loss had pallor, 25% and 16.7% had no pallor and severe pallor respectively.

Table 11(b): Palmar pallor and etiology of anemia

	Nutritional	Hemolytic	Bone Marrow Failure	Blood loss	Total	P-Value
No pallor	48 (14.5%)	27 (14.8%)	4 (12.9%)	3 (25%)	82	0.10,NS
Pallor	224 (67.7%)	80 (43.7%)	24 (77.4%)	7 (58.3%)	335	0.0001,S
Severe pallor	50 (15.1%)	65 (35.5%)	2 (6.5%)	2 (16.7)	119	0.0001,S
Not detected	9 (2.7%)	11 (6%)	1 (3.2%)	0 (0)	21	0.10,NS
Total	331	183	31	12	557	

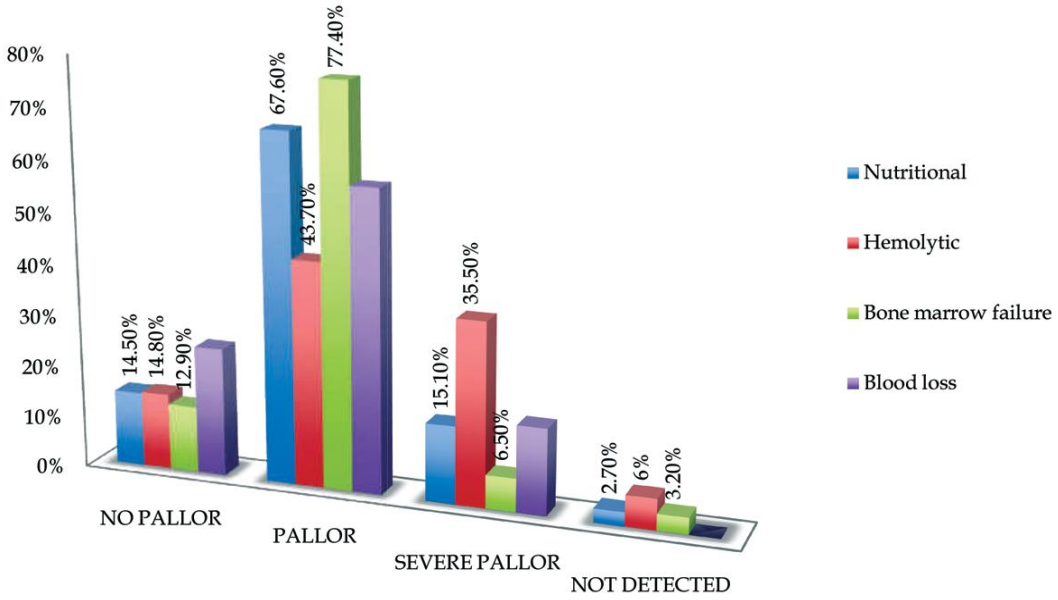


Chart 11(b): Palmar pallor and etiology of anemia

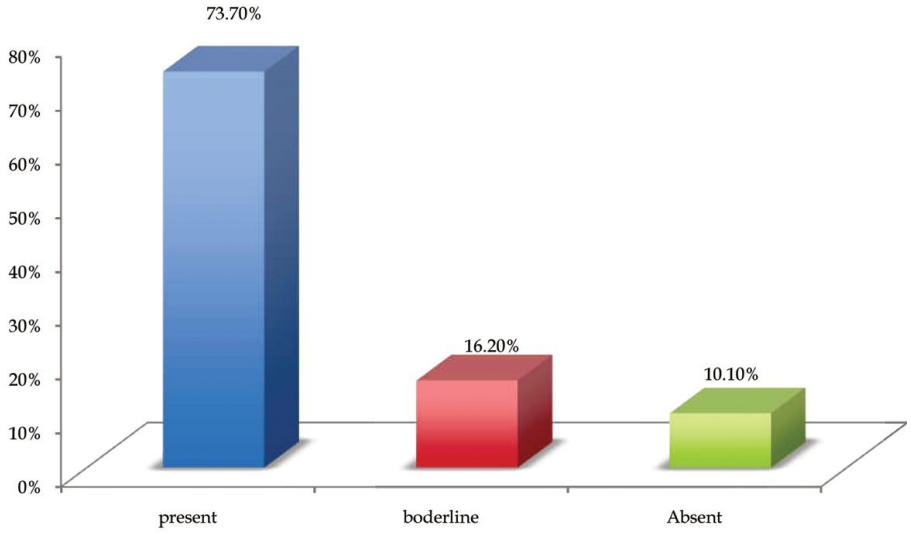


Chart 12: Conjunctival pallor

Table 13: Mean hemoglobin value in different age groups

	Min Hb (gm %)	Max Hb (gm %)	Mean Hb (gm %)
6mo-5years	2.3	10.9	8.69±1.61
5-11years	3.2	11.2	8.51±1.66
12-15years	4.3	11.4	8.21±1.48

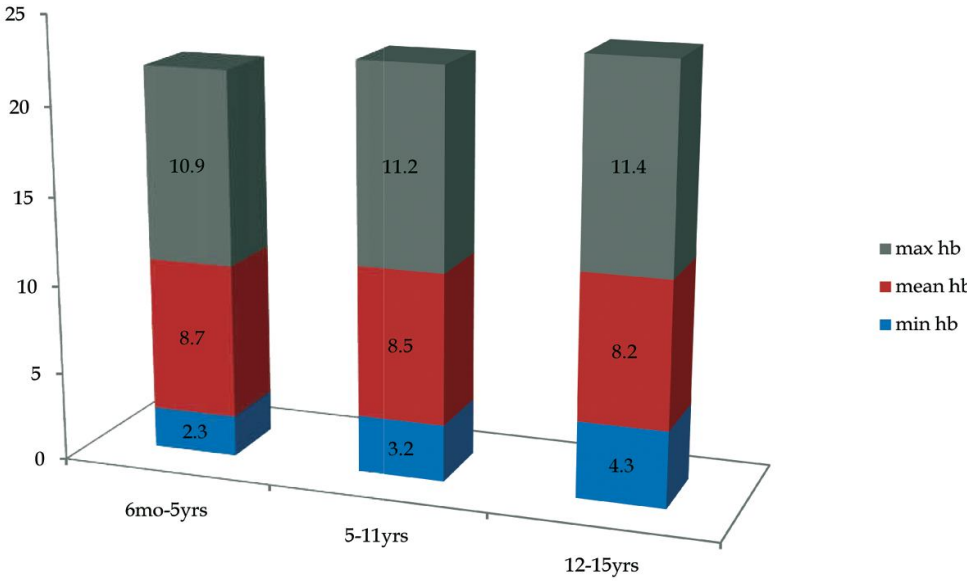


Chart 13: Mean hemoglobin value in different age groups

Table 14: Risk factors associated with nutritional anemia

	Iron deficiency (293)	Megaloblastic (22)	Mixed* (16)	Total
Decreased calorie intake	240 (82%)	16 (72.7%)	14 (87.5%)	270 (81.9%)
Cows milk >400ml/day	101 (34.5%)	7 (41.2%)	6 (37.5%)	114 (34.4%)
Prolonged breastfeeding (>2yrs)	50 (17%)	7 (41.2%)	7 (43.8%)	64 (19.3%)
Vegetarian diet	190 (64.8%)	17 (77.27%)	10 (62.5%)	217 (65.6%)
P-VALUE	0.0001,S	0.0001,S	0.0001,S	

*Mixed is iron deficiency with megaloblastic anemia

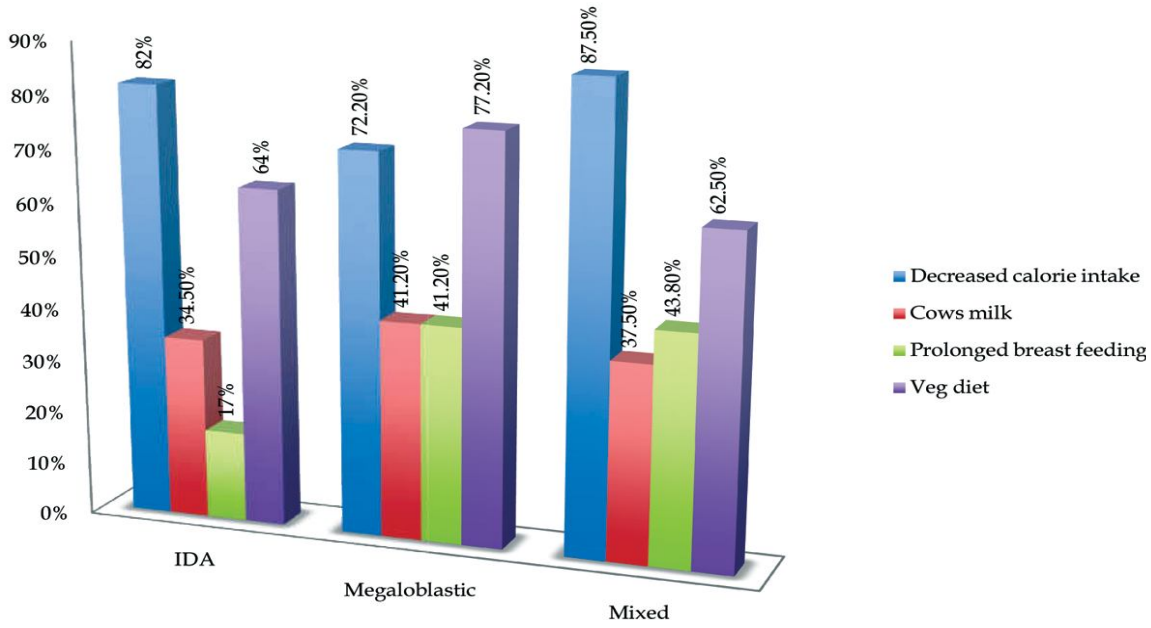


Chart 14: Risk factors associated with nutritional anemia

In the present study we found that conjunctival pallor was present in 73.7% children, borderline in 16.2% and absent in 10.1% of the total anemic patients.

The mean hemoglobin was found to be 8.7gm%, 8.5gm% and 8.2% in children between 6 months to 5years, 5-11 years and 12 to 15years respectively.

Decreased calorie intake (81.9%), vegetarian diet (65.6%), Cow's milk intake >400ml / day (34.54) and prolonged breastfeeding (19.3%) were certain risk factors identified in children with nutritional anemia, with decreased calorie intake being the most common risk factor in all the three types of nutritional anemia, i.e. 82% in children with iron deficiency anemia, 72.7% in megaloblastic anemia and 87.5% in mixed anemia.

Total 379 (68%) children out of 557 belonged to lower class, followed by 73, 13.1% in middle and 105, 18.9% (45+60) in Upper class.

Table 15: Body mass index

Scale	Number	Percentage
Severe thinness	95	17.1%
Thinness	335	60.1%
Normal	104	18.7%
Overweight	23	4.1%
Obesity	0	0
Total	557	100

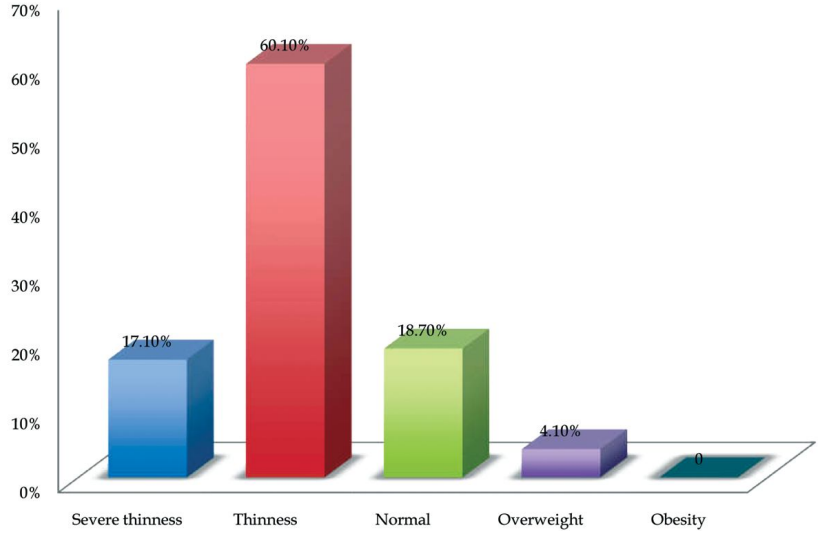


Chart 15: Body mass index

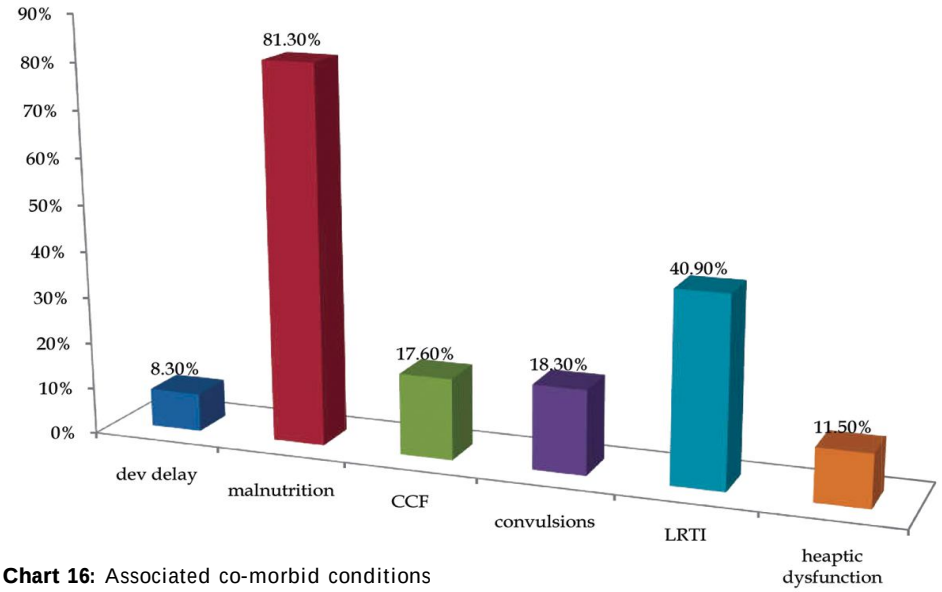


Chart 16: Associated co-morbid conditions

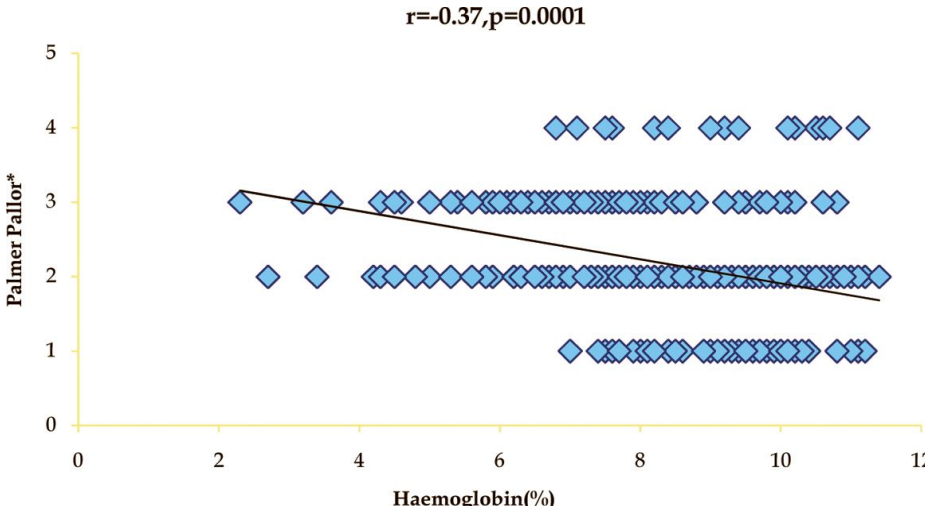
Table 16: Associated co-morbid conditions.

	Number	Percentage
Developmental delay	46	8.3%
Malnutrition*	453	81.3%
Congestive cardiac failure	98	17.6%
Convulsions	102	18.3%
Respiratory tract infection	228	40.9%
Hepatic dysfunction #	64	11.5%

Hepatic dysfunction – derranged liver function tests)
*Malnutrition includes thinness, severe thinness and over weight

Table 17: Correlation between Hb% and Palmar Pallor

	Mean	SD	Correlation r	p-value
Hb%	8.55	1.61	-0.37	0.000 S,p<0.05



Graph 17: Correlation between Hb% and palmar pallor

* Palmar pallor – 1: No pallor, 2: pallor, 3: severe pallor

We observed thinness in 60.1% children, where as 18.7% were normal, severe thinness was observed in 17.1% and 4.1% children were overweight. No obesity was observed.

Maximum numbers (285, 51.2%) of mother attended primary school, 32% were illiterate, 14.4% went to high school, only 1.9% were graduates and 0.5% were post graduates.

Most of the children with anemia were malnourished (81.3%).Amongst otherobservedco-morbidities were respiratory tract infections (40.9%), convulsions (18.3%), congestive cardiac failure (17.6%), hepatic dysfunction (11.5%) and developmental delay (8.3%).

In the present study we observed a negative co-relation between hemoglobin and palmar pallor, as seen the graph above (Graph no.20). With the in crease in hemoglobin levels the palmar pallor decreases.In the present study sensitivity of palmar pallor in the present study was found to be 81.5%6.

Discussion

Anemia is a global public health problem affecting both developing and developed countries with major consequences for human health as well as social and economic development.It occurs at all stages of the life cycle, but is more prevalent in young children and pregnant women. In 2002, iron deficiency anemia (IDA) was considered to be among the most important contributing factors to the global burden of disease.

Currently, the World Health Organization accepts that generally a little less than 50% of all anemias can be attributed to iron deficiency.[1]

The WHO categorizes the prevalence of anemia as a public health problem as follows:

<5% – no problem

5–19% – mild public health problem

20–39% – moderate public health problem

>40% – severe public health problem

Anemia is the world's second leading cause of disability. In terms of lost years of healthy life, iron deficiency anemia causes 25 million cases of Disability Adjusted Life Years (DALYs); this accounts for 2.4% of the total global DALYs.[15]

The main purpose of the study is know the risk factors, clinical manifestations, various etiological and morphological types in children so that the problem can be tackled in a better way and steps can be taken to minimize the suffering of the children. Total 557 children admitted in Pediatrics ward with anemia were studied.The discussion of the present study will be done under the following headings:

1. Demography
2. Clinical features and signs
3. Organomegaly
4. Morphological classification
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Demography

Age and Sex

In our study we included 557 children with anemia, maximum (52.5%) children were between 6months to 4.99 years (pre-school), followed by school going children who were between 5 to 11 years (31.4%) and adolescent between 12 to 15years(16.3%) (Table no.2).In our study out of 557, 332(59.6%) were males and 225 were females. The sex ratio was 1.5:1 (Table no.1). The sex ratio was different in all the three age groups. Males were more in pre school children (63.2%) and school going children (60.6%) than females, while in adolescent age group females (53.8%) were more than males (46.2%) (Table no.2)Though we could not calculate the prevalence of anemia as we chose to include children as per the sample size of our study, which was an added limitation to this study, various studies have observed anemia to be most prevalent in pre-school children.

Hanumante et al(2008)[2] studied 50 toddlers (1 to 3years), 25 males and 25 females in urban slums of Pune andreportedanemia in 66%.

Pasricha et alError! Bookmark not defined. (2011) also reported 75.3 % of anemia prevalence in children aged 12 to 23 months in 2 rural districts of Karnataka, India.Error! Bookmark not defined.

Prevalence of anemia was higher in Punjab pre school children 90.5 % as reported by Sharadasidhu (1996).[3]

N. Arlappa et al. (2012) [4]conducted a community-based cross sectional study on Prevalence of anemia among rural pre-school children (1-5years) of Maharashtra, reported prevalence rate of 59.2% (CI: 54.4-64.0) and the prevalence was significantly($p<0.001$) higher (76.5% with CI: 68.1-84.9) among 1-3-year children as compared to 53.6% in 4-5-year- children

Muthayya S et al(2007) [5]studied a total of 2030 boys and girls, aged 5–15 years, attending schools in the Bangalore district and reported low prevalence rate of anemia in 13.6%.

Comparison of the Prevalence of Anemia in Pre School Children between the Present Study and Previous Studies in Indian

Authors	Year of study	Region	Anemia prevalence
Present study	2013-2015	Wardha, Maharashtra	52.2%
Sharadasindhu et al	1996	Punjab	90.5%
Pasricha et al	2011	Karnataka rural districts	75.3%
Hanumante et al	2008	Pune	66%
N.Arlappa et al	2012	Maharashtra	59.2%
Muthayya S et al	2007	Bangalore,	13.6%

The differences in the prevalence of anemia between the studies may be due to different geographical location and other factors like sample size and selection of subjects.

A study done by Gomber et alError! Bookmark not defined. revealed that the prevalence of anemia in urban slums school children aged 5 to 10.9 years was 41.8 percent.

In a similar study carried out among 1138 children aged 5- 15 years in urban areas of Guntur by

PhaniMadhavi[6] et al (2013) the prevalence was found to be 28.92% which was similar to the result of our study in school going children.

ShardaSidhu (2005)[36] reported that prevalence of Anemia among 265 adolescent girls between the age group 11 and 15years old of scheduled caste community of Punjab was 70.57%.

In another cross sectional study by Sanjeev C et al (2008)[7]in Government Medical College and Hospital,amongst 296 adolescent females (10–19 years old) the prevalence of anemia was found to be 35.1%.

A study by Sabita et al[8] (2005) showed that the over all prevalence of anemia among 1120 school going adolescents (12 to 18 years) 48 of Chandigarh were 16.25%.

Similar study conducted by KP Baral and SR Ontain[9]308 adolescents in Nepal the overall prevalence of anemia among adolescents (male, female, urban and rural combined) was found very high with 65.6%.

SalujaN et al (2010) [10]for the purpose of study divided the urban area of Meerut district into four zones. A list of all government primary schools was taken and arranged according to the zones. Equal numbers of students were examined from the randomly selected school / schools from each zone 37.7% prevalence was reported in children between 5-11years.

Authors	Age group	Region	Prevalence
N.Arlappa et al (2011)	6-12 years	West Bengal	81.2%
ShardaSidhu (2005)	11-15 years, girls	Punjab	70.57%
Ruchika H et al	7-10 years	Allahabad, Uttar Pradesh	65.33%
Jain N et al (2012)	5-16 years	Rishikesh, Uttrakhand	56.5%
Verma et al	5-15 years	Punjab	51.5%
Gomber et al	5-10.9 years	Delhi	41.8%
Biradar SS et al (2012)	10-19years	Vantamuri, Karnataka	41.1%
Saluja et al	5-11 years	Meerut, Uttar Pradesh	37.7%
PhaniMadhavi et al	5-15 years	Guntur, Andhra Pradesh	28.92%

In a Study conducted by Mohamed Ag Ayoya[11] (2013) in 557 pre school children in Haitishowed that the prevalence of anemia was slightly higher among boys (42.1%) than girls (35.7%).

Kriviene et al[12](2006) reported that the overall prevalence of anemia among 6 to 16 years old children was higher in girls 17.8 % than in boys 3.4%.

Alain B et al (2012) has reported statistically significant genderdifferentiation in anemia in pre school children. The result revealed that theprevalence of anemia in pre schoolboys was higher 35.3 % than girls (30 %).

The prevalence of anemia in 5 to 15 years school children of an urban area of Guntur, India, was significantly higher in girls (65.35%) than in boys (34.65%) reported by PhaniMadhavi K.V. et al[39]in contrast to our study, where we found anemia to be more prevalent in males (60.6%) in school going children than in females (39.4%).

Clinical Symptoms and Signs

Rupali V et al [13](2013) studied 385 school going children of Mumbai and reported breathlessness, palpitations, fatigue and lack of concentration in 14.58%, 32.29%, 34.38% and 23.44% males and 21.76%, 42.49%, 44.04% and 19.69% females respectively.

Bhagwat AM [14] observed that out of 306 children studied, 183 never experienced dizziness, where as 123 felt dizzy and 136 experienced headaches.

In a study done by Venkatesh Get al[15] (2013) 202 severely anemic children (1-5years) were studied and among which Pallor was seen in 100% of patients, vitamin deficiency in 54.4%, knuckle pigmentation in 29.7%, edema in 21.7% and koilonychia in 10.8%.

Kapil et al[16](2002)reported that even mild iron deficiency results in poor attentiveness, memory and academic performance in the areas of vocabulary, reading and knowledge. Children with iron deficiency perform less well on standardized scholastic tests and have impaired motor development.

Shemesh Z et al[17](1993) reported that patients with tinnitus and noise-induced hearing lossNIHL exhibited vitamin B12 deficiency in 47% of cases (blood levels }* 250 pg / mL). This was significantly more (P < .023) compared with NIHL and normal subjects who exhibited vitamin B12 deficiency in 27% and 19%, respectively.

Y.C. Wu et al [51](2013) showed that anemic patients had significantly higher frequencies of all oral manifestations than healthy controls ($p < 0.001$ for all), in which burning sensation of oral mucosa (76.0%), lingual varicosity (56.0%), dry mouth (49.3%), oral lichen planus (33.3%), and atrophic

glossitis (26.7%) were the five leading oral manifestations.

In a study conducted by Onder et al [19](2005), depressive disorder was found to be a common disorder in patients with anemia.

A study done by Semiz M [20]et al (2015) a major portion (nearly 45%) of the patients had high anxiety and depression and 67.3% patients reported a bad sleep quality. In our study we found sleep disturbances only in 9.7% of the study population, the difference could be because in our study all the clinical features were based on the history whereas they used hospital anxiety and depression (HAD) scale and Pittsburgh sleep quality index (PSQI) for analysis.

Haq S et al[21] (2012) studied eighty patients with a megaloblastic change in bone marrow in Lahore. There were 32 males (40%) and 48 females (60%). The most common clinical presentation was pallor and fatigue (67 patients, 84%).

In the present study easy fatigability (26%), irritability (25%), lack of concentration (17.6%), breathlessness (16%), headache (15.4%) and palpitations (13.5%) were a few common symptoms observed in the study population, where as anorexia (11.7%), insomnia (9.7%), giddiness (9%), tinnitus (2.2%) and menstrual abnormality (0.2) were less common. We also observed that mildly anemicchildren were less symptomatic. The manifestations were more prevalent in children with moderate anemia followed by severe anemia. This can be explained because maximum number of children with sickle cell and thalassemia had moderate anemia and were more symptomatic because of the acute change in hemoglobin levels and acute symptoms, where as in nutritional anemia, the drop in hemoglobin is slow and chronic so they tend to be more adaptive. Based on etiology, clinical features were more commonly reported in nutritional and hemolytic anemia probably because we had more number of children in both the categories. Certain features like irritability (54%), anorexia (49.2%), easy fatigability (42.1%) and giddiness (40%)were more common in pre school children. Tinnitus (58.3%), Lack of concentration (56.1%), Palpitations (54.7%), insomnia (48.2%), headache (46.5%) and breathlessness (40.4%) were more common in school going children. We found menstrual abnormality only in 1 female child in adolescent category.

In the present study we observed Icterus in 13.2%, knuckle pigmentation in 12.2%, glossitis in 11.7%, Platynychia/koilonychia in 7% and angular stomatitis in 6.1% children. These signs were more common in children with moderate anemia. And when classified age wise we observed that pre-school children had more clinical signs than any other category because most of these signs were realted to iron deficiency anemia and it was found to be most prevalent in pre school children.

Organomegaly

Lucia F et al [22](1998) studied 89 children with hepatosplenomegaly and observed anemia in 70 children (79%).

Parmar D et al[56] reported that out of 95 cases of sickle cell anemia splenomegaly was found in 50 cases (52.63%) of which 42 cases (44.21%) were males while only 8 cases (8.42%) were females.

Somaiah G et al[24] (2014) studied a total of 150 cases, from One Month to Fifteen years of age with Hepatosplenomegaly. Anemia was observed in 22.67% of patients of the study population, forming the second major group. Out of which 11.33% had Thalassemia, 8.67% were due to Sickle cell disease, 1.33% due to hereditary spherocytosis and 1.33% due to hereditary persistence of fetal Hb.

Shahu et al[Error! Bookmark not defined.]also found splenomegaly in patients with sickle cell anemia in 24.72% of under fives, 35.1% of 5-9 years, and 13.64% of 10-15 years.

In the present study we found hepatomegaly in 124 children, splenomegaly in 114 children and hepatosplenomegaly in 51 children amongst the study population. In the our study we found splenomegaly (54.3%), hepatomeglay (52.4%) as well as hepatosplenomegaly (47.1%) to be most prevalent in hemolytic anemia, because this is a sickle cell belt and sickle cell anemia is the most common type of hemolyitc anemia and splenic crisis is rare hence autosplenectomy not seen commonly. Organomegaly was found to be strongly associated with moderate anemia as most of the children with sickle cell anemia and thalassemia had moderate anemia.Out of 331 cases of nutritional anemia 51(15.4%) had hepatomegaly, 43 (12.9%) had splenomegaly and 23 (6.9%)had hepatosplenomegaly.

Morphological Classification and Mean Hemoglobin

Gera et al[25](1991) reported an almost four fold rise in proportion of macrocytic anemia cases over less than a decade at one center-2 % in 1991 and 7.8% in 1999.

In a study done by Padmanabhan A et al[26](2001) in Oman in 256 children who were divided into two groups: 153 children between 3-5 years and 103

children between 5-10 years showed that 45.1% children in group A and 37.9% in group B were anaemic according to WHO criteria and all the anemic children had low mean corpuscular haemoglobin and 75% showed microcytosis which was much higher than the what we observed (48.3%) in our present study. The microcytic anemia in the study conducted by Padmananbhan et al could be attributed to the alpha-thalassaemia trait, which is highly prevalent in Oman.

Chaudhry et al[27] (2001) reported microcytic anemia in children with nutritional anemia in 27.1%, which was lower when, compared to our study. In their study they only included children with nutritional anemia, where as in our study we also had children with thalassemia, which added to the burden of microcytic anemia.

In contrast to our studySalah N et alError! Bookmark not defined. reported that out of 75 anemic children in Egypt 60% (45) had microcytic hypochromic anemia, 24% (18) children had normocytic normochromic and 16% (12) children had macrocytic hyperchromic. The variation may be becasuse of the ethinicity and sample size, thoudh the trend was the same, even we found maximum children with microcytic anemia followed by normocytic and macrocytic.

K. S. Lamsal[61](2009) studied 237 patients in a tertiary hospital in Nepal, the average hemoglobin was 7.8gm%, the lowest being 2.8gm%. Morphologically hypochromic picture was seen in 140, macrocytic picture in 26 and normocyticnormochromic in 71 cases.

In the present study mean hemoglobin was 8.7gm%, 8.5gm% and 8.2gm% and minimum Hb was 2.3gm%, 3.2gm% and 4.3gm% in pre-school children, school going children and adolescents respectively (table no.14). We found that microcytic anemia (48.3%) was the commonest type, followed by normocytic anemia (46.2%), macrocytic anemia (3.9%) and Dimorphic (1.6%).

Etiological Classification

Sunil Gomberet alError! Bookmark not defined.studied 95 children between 5 to 10.9 years for etiology of anemia, 51 were boys and 44 girls. Pure or mixed iron deficiency anemia was found in 68.42 per cent children followed by pure or mixed vitamin B12 deficiency in 28.42 per cent children. Pure iron deficiency was the commonest cause occurring in 41.05 per cent children.Similar study done in pre school children by Pasricha et al (2011) and Garcia-Casal et al (2008) iron deficiency anemia was noted in 61.9% and 56% of the studied population respectively.**Error!** Bookmark not defined.Error! Bookmark not defined. In a study by Chaudhry MW[60] (2001) observed B12 deficiency in 19.0%, folate deficiency in 20.0% and mixed in 14.0%.In the present study out of 557 children nutritional anemia was noticed in 331 i.e. 59.6% (Table no.4), and out of 331 children 293 (88.5%) had iron deficiency anemia and it was found to be most common in pre school children (195, 90.3%). Where as megaloblastic anemia was observed in 22 (6.7%) children with nutritional deficiency, and was equally observed in pre school children (10, 45.5%) and adolescents (10, 45.5%). Amongst 331 children mixed / dimorphic anemia was observed in 16(4.8%) and pre school children were most affected (11,68.8%) and the data was found to be statistically significant (p value <0.001) (Table no.5).

According to nutrition Examination Survey[29] (NHANES, U.S.,2002), the prevalence of anemia in stage 3 chronic kidney diseases was 5.2%, rising to 44.1% in stage 4, and becoming almost universal in stage 5. We also found 8 patients with chronic kidney disease having iron deficiency anemia, this was probably due to increased loss of iron due to dialysis.

A Study from Orissa byKar BC et al (1986)[30]reported SCD in hospitalized pediatric patients to be 6.42% (results based on positive sickling test) and 11.1% (results based on hemoglobin electrophoresis), which was similar our observation of sickle cell disease (13.5%).A study conducted by M kamble and P Chaturvedi(2000)Error! Bookmark not defined.in1753 children out of which 99 (5.7%) were diagnosed to have SCD. Of these, 61 (61.6%) had homozygous state (HbSS) whereas 38 (38.4%) had heterozygous state (HbAS).Shukla RM and Solanki BR[31] did a study in Central India and reported sickle cell trait (SCT) in 11.1%.Tariq HA[32]had screened 3980 children and found sickle cell trait in only 60, all being asymptomatic.Yadav et al[33]

[33]Rajiv Y, Gupta RB, Bharadwaj VK, et al. Morbidity Profile of Sickle Cell Disease in Central India. Proceeding of National Symposium on Tribal Health; 1999; 136-40.

found most of the patients of with sickle cell disease belonging to 10 to 15 years age group, 78(25.16%) in a group of 310 patients.In a study conducted by Jain D et alError! Bookmark not defined. in their study of sickle cell traits observed that 63.41% were males and 36.58% were females. Both the studies were done in Vidharbha region (Nagpur).In the present study we observed that 76 (13.5%) children out of 557 had sickle cell anemia and 54 (9.7%) had sickle cell trait and was most common inchildren between 5 to 11

years,the high prevalence in our study could be explained as the study was conducted in central India, which is an endemic for Sickle cell disease, where as the prevalence in our study was lower than what was observed in a study conducted in central india by Jain D et al. Central Maharashtra is reported to be in the sickle cell belt by Kar BC[34].

Pasricha et alError! Bookmark not defined.(2011) conducted a study in 401 children and found thalassemia in 1.3%.In a Study conducted by Balgir RS (2005) in Orissa thalassemia major observed in 5.3% patients out of 1015.A large study by Mulchandani et al [35] done among the Sindhis of Nagpur in Maharashtra had shown the prevalence of â-thalassemia trait to be 16.81 %.In a study Madan N et al[36] reported â-thalassemia trait as 2.7 % in Mumbai, 5.5 % in Delhi and 10.2% Kolkata, result of the present study was comparable to this study, we found thalassemia in 24(4.3%) children out of 557 anemic children.

PetzLD et al (2004) reported the incidence of autoimmune hemolytic anemia in adults of 0.8-3 per 105 / year, a prevalence of 17:100,000 and a mortality rate of 11%.Error! Bookmark not defined.Error! Bookmark not defined.In a study Buchanan et al (1976) found a 32% incidence of secondary AIHA.Error! Bookmark not defined.In our study we found autoimmune hemolytic anemia only in 8 out of 557 (1.4%) children, other causes of hemolytic anemia were malaria, DIC, hepatitis and HUS and constituted 3.8% (21 / 557) of total anemia.

Saima B et al (2011) studied 110 children admitted in a pediatric hospital in Pakistan and observed aplastic anemia in 6 (5.5%).Kevin K et al[37] reported that 7% of patients with Hodgkin disease had anemia and prevalence varied by cancer type and disease stage and nearly 80% of patients with advanced disease had anemia.Tesarova P (1995)[38] reported that upto to 30% of patients with tumors suffer from anemia. In the present study we found 3 (0.5%) children with aplastic anemia.Anemia due to bone marrow suppression was found in 31 (5.6%) of 557 children, out of which 14 had malignancies (45.2%). We found 7 cases of ALL, 1 case of AML, 2 non hodgkins lymphoma, 2 hodgkins lymphoma and 3 neuroblastomas.

In the present study anemia with blood loss was observed in 12 (2.2%) children, which included acute or chronic blood loss due to portal hypertension (3 / 12), blood loss in head injury and trauma (6 / 12) and menstrual abnormality (1 / 12), Hemophillia (1 / 12), Von wilibrand (1 / 12).

Severity

According to National family health survey 3Error! Bookmark not defined. (2003-05), 26 percent are mildly anemic (10.0-10.9 g / dl), 40 percent are moderately anemic (7.0-9.9 g / dl), and 3 percent are severely anemic (less than 7.0 g / dl).

A K Shina et al[39](2013) reported that 43.3 percent had mild anemia, 45.1 percent had moderate anemia and 11.6 percent had severe anemia out of 589 adolescents (10-19 years) in Nepal.

Sidhu S (1996)[36]observedmild, moderate and severe anemia in 6.33%, 75.75% and 8.42% respectively in pre schoolchildren of Punjab.

A study conducted by Verma A et allError! Bookmark not defined. (2004) amongst school going girls in Ahmedabad revealed that 55.2% were mildly anaemic, 44.9% were moderately anaemic and that 0.6% were severely anemic.

Sinha N et alError! Bookmark not defined. (2008) conducted a study on epidemiological correlates of nutritional anemia among children in Wardha, Central India. Seven hundred seventy-two children between 6 months and 35 months of age were studied for anemia by cluster-sampling method. They reported a mean hemoglobin level was 98.5 ± 12.9 gm / L. Prevalence of anemia was 80.3%. Only 1.3% children had severe anemia (hemoglobin <70 gm / L).

S. Jain et al (2000) in their study observedanemia in 59.9 % in 137 children of age 1-2 years in urban slums of Meerut. Of these anemic children, 24.3% had severe anemia, 49.8% children had moderate anemia and 26.8% had mild anemia

Similarly in our study moderate anemia was observed in 313 (56.2%) children among 557, 25.8% children had severe anemia and 18% children had mild anemia. 62 (43.1%) of patients presenting with severe anemia were found to be between 5 to 11 years of age. Where as 83(83%) of mildly anaemic and 164 (52.4%) of moderately anemic paitents were pre-school children. Moderate anemia was the most common in all the etiological types of anemia.

Blood Transfusion

K. S. Lamsal[61]reported that 84 patients were transfused blood of total 237 patients included in the study.

A study conducted in central India by Jain D etalError! Bookmark not defined. (2003) also found that severe anemiasrequiring blood transfusion was the most common reason for admission in the hospital.

In our study we found that the blood transfusion rate was very high in children with anemia due to blood loss and hemolytic anemia. 10 (83.3%) of

patients with anemia due to blood loss and 93 (50.8%) of patients with haemolytic anemia required blood transfusion. Of the haemolyticanemias, 66 (86.8%) of patients with sickle cell anemia and 20 (83.3%) required blood transfusions.

Palmar and Conjunctival Pallor

Santra G et al [40] reported that in severely anemic patients sensitivity of palmar pallor is only 12% but specificity is 100% and positive likelihood ratio is >1200.

Montresor et al[41] also reportedhigh specificity of clinical diagnosis (91% by observingpallor at three sites) but the sensitivity was as low as 20per cent.

Zucker et alError! Bookmark not defined. (1997) severe anemiawas best identified by the presence of severe nailbed orsevere palmar pallor as indicated by thehighest sensitivity (62% and 60%, resp.) comparedwith severe conjunctival pallor (sensitivity = 31%) severe tongue pallor (sensitivity = 13%) or nailbedblanching (sensitivity = 55%). Similarly, childrenwith moderate anemia were best identified by thepresence of nailbed or palmar pallor (sensitivity = 90% for both signs), compared with conjunctivalpallor (sensitivity = 81%), tongue pallor (sensitivity = 59%), or nail bed blanching (sensitivity = 58%) found that 60% of cases of severe anemia in children (Hb<5 g/dl) could be detected through clinical signs alone, and that such an evaluation could be used for identifying children with moderate or severe anemia.

Luby et al[42] (1995) recognized the validity of this method for the detection of severe anemia (93% sensitivity) and were able to identify 66% of children with moderate anemia, similarly in our study we found sensitivity of 98.6% in cases of severe anemia.

In contrast to our studyStoltzfus J Et al[43] (1999) reported that the sensitivity of pallor to detect low hemoglobin concentrationin individuals was low at higher cutoffs and increasedgreatly at lower hemoglobin cutoffs.

In a study by Kalter HD[44], lower sensitivity of palmar than conjunctival pallor was seen among children in Bangladesh due to increased palmar pigmentation. In the present study we couldn't detect palmar pallor in 21/557 children due to increased palmar pigmentation and icterus in some children.

In our study we found palmar pallor to be 81.5% sensitive. As all the children included in he study were anemic, we couldn't calculate the specificity of palmar pallor, which was the major limitation of this study. Sensitivity of palmar pallor increased with the severity of anemia, it was 61% sensitive for mild, 80.2% sensitive for moderate and 98.6% sensitive for severe anemia. Conjunctival pallor was 90.1% sensitive in diagnosing anemia.

Risk Factors

Cows Milk

Consuming >400 mL of milk/day is accompanied with less consumption of iron-rich foods and drinks, and these children are more prone to have a poor iron status.[45]

In a subanalysis by Lieke Uijterschout of 246 children >1 year of age, 92 children (37.4%) received follow-on formula and 12 of these children (13.0%) were iron deficient, whereas among 154 children (62.6%) not receiving follow-on formula, 47 (30.5%) were iron deficient (odds ratio 2.9, 95% CI 1.5 – 5.9). Intake of >400 mL of cows' milk per day occurred more frequently in children with ID than in those without ID[46]

In the present study 114 (34.4%) children out of 331 cases of nutritional anemia had history of cows milk intake >400ml/day, maximum number of these children were observed to have iron deficiency (101/114).

Oliveira A et al[47] (2005) reported that cow's milk has decreased iron density and bioavailability, excess protein and minerals, notably calcium, and thus interferes in the absorption of iron from other foods, and is also linked to small intestinal hemorrhage in young children.

Vegetarian Diet

Banerjee DK et al [48] (1960) in their study conducted in Calcutta stated that Serum vitamin B12 in the vegetarian group was in general; lower than that in the non-vegetarian group.

Antony AC[49] stated that only in the past 50 years was it recognized that vegetarians have consistently lower vitamin B-12 concentrations than do non-vegetarians and that vegetarians are at greater risk of vitamin B-12 deficiency than are non vegetarians. Because vitamin B-12 is produced in nature only by vitamin B-12–producing microorganisms, humans must receive vitamin B-12 solely from the diet, our present study also reflected an association between vegetarian diet and B12 deficiency, 17 (77.27%)on 22 children with megaloblastic anemia were vegetarians.

K.A. George et al (2000) conducted a study to analyze the anemia and Nutritional status of pre-

school children in Kerala, among 927 vegetarians, 86 (9.27%) were anemic and among 2706 non-vegetarian, 328(12.1%) were anemic.

In our study we found 217 (65.6%) lactovegetarians out of 331 children with nutritional anemia. Iron deficiency had 64.8% lactovegetarian and out of 22 children with megaloblastic anemia 17 (77.27%) were lactovegetarians. (Table no 15)

Breastfeeding and Anemia

Exclusive breastfeeding for more than 6 months has been associated with increased risk of IDA at 9 months of age.[50] We found similar results in our study that prolonged exclusive breastfeeding was associated with anemia. Out of 331 children with nutritional anemia, history of prolonged exclusive breastfeeding was present in 64 children, out of which 43.8% had mixed anemia, 41.2% had megaloblastic anemia and 17% had iron deficiency anemia.

Maternal Education

A study by Saluja et al[43] showed that the Percentage of anemia was significantly (p<0.001) higher in children of illiterate mothers compared to educated mothers, which may be attributed to their lack of knowledge about iron rich foods.

Bharathi S et al[51]also reported high prevalence of anemia in children of illiterate parents.

In the present study mothers of 178 (31.94%) children were illiterate, 283 (50.8%) went to primary school, 80 (14.4%) went to high school and 11 (1.97%) mothers were graduates and 5 (0.89%) were postgraduates and most of them were not aware of anemia as a disease and its consequences.

Malnutrition

Gomber S et al Error! Bookmark not defined. observed that among 406 anemic children, 205 (50.5%) had normal nutrition, 145 (35.87%) had grade I malnutrition, 51 (12.6%) had grade II malnutrition and 5 (1.2%) had grade III malnutrition.

In a study by S jain et al prevalence of anemia was also found to be significantly higher in children having low nutritional status (84.3%) as compared to children of borderline (51.4%) or normal nutritional status (52.9%).

Saluja N et al[43] (2010)studied 800 children (426 boys and 374 girls) out of which 542 children (67.8 %) were found to be suffering from one or more morbid conditions. total of 2532 morbidities were found to be present in 542 sick children accounting for 4.6 morbidities per sick child. Maximum children (93.4%) were having morbidity related to nutritional deficiencies.

M.E. Bentely et al (2003) studied 4032 women and found Fifty-two percent of thin, 50% of normal BMI, and 41% of overweight women were anemic.

In our study we found out of all the anemic children 17.1% had severe thinness, 60.1% children were thin, 18.7% were normal and 4.1% were overweight, we did not observe obesity in our study population.

Body mass index was the standard used in our study to assess nutritional status of the patient as the age groups coverd in our study ranged from 6 months to 15 years and BMI charts (as per WHO guidelines) were standardized across all the age groups. Thus comparision of the nutritional status of the patient was possible across all the age groups.

Socio Economic Status

Sharma P et al[52] found that the prevalence of anemia was found to be significantly more (p<0.001) in children belonging to socio economic class IV (35.71%) when compared to children belonging to socio economic class I (2.59%).

Saluja N et al [43] also observed higher prevalence of anemia (100%) in children belonging to Lower class V as compared to children belonging to upper middle class II (22.2%).

In the present study we found that maximum number of children (298, 53.5%) belonged to class IV of Kuppuswami classification, followed by 14.5%, 13.1%, 10.8%, 10.1% in class V, class III, class II, class I respectively.The higher prevalence of anemia in children from low socio economic status in our study can be attributed to the poor dietary intake, higher incidence of infection and infestation among them.

Co-Morbidity

Convulsions

Rehman N et al[53](2005) reported that Iron deficiency anemiawas significantly more frequent among the children with febrile convulsions as compared to the controls as evident from parameters studied i.e. hemoglobin <10 g/dl (p-value= <0.000), hematocrit <30% (p= <0.01), MCV <70 fL (p=<0.002), MCH<24 pg (p= <0.001) and serum ferritin <10 ng/ml (p= <0.000). Similarly in the present study we found that 18.3% (102) children had convulsions out of which 68 were febrile convulsions. (table no 19)

Developmental Delay

Antunes H et al[54] (2002) compared the development of 17 children with IDA and control. At 12 months children with IDA had significantly lower development scores—mean (sd)—than those without IDA: 112(5) vs. 121(7). At 15 months, after iron therapy, there were no significant differences between cases and controls. Non-IDA children showed significantly lower development scores at 15 months when compared with 12 months (121 vs 115).

In our study we found developmental delay in 46 children out of 291 pre school children had developmental delay.

Congestive Cardiac Failure

InderAnand et al (2004) reported that anemia is associated with heart failure and Anemia (Hb d"12.0 g / dL) was present in 12% of subjects.

In the present study we found that anemia was associated with CCF in 17.6% of children.

Conclusion

1. Nutritional anemia was the commonest etiological type of anemia, with Iron deficiency being the most frequently observed sub type, followed by hemoglobinopathies and hemoatologicalanemia, which was mainly constituted by sickle cell anemia and thalassemia.
2. Clinical features like easy fatigability, irritability, lack of concentration, breathlessness, headache and palpitations were frequently observed in children with moderate anemia, where as children with mild anemia were relatively symptom free.
3. Palmar pallor was found to be 81.5% sensitive and conjunctival pallor was 89.9% sensitive, sensitivity was in more in severe forms of anemia hence we conclude that palmar pallor and conjunctival pallor can be used as a simple diagnostic tool for moderate and severe form of anemias, and reduce the morbidity and mortality associated with anemia.

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Original Article

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Prevalence of Overnutrition among Late Adolescents

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Abstract

Introduction: Childhood obesity is one of the most serious public health challenges of the 21st century. [1]Overweight and obesity among the adolescents is in the rising trend that often begins in childhood. *Materials and Methods:* cross-sectional study carried out over a period of six months, from November 2013 march 2014. 240 adolescents, 16 to 18 years of age, of raichur city, Karnataka, India were included as subject in the study. *Results*: A total of 240 adolescents in the age group of 12 to 18 years were analyzed. Out of these 132 (55%) subjects were males. The mean BMI of the sample was 21.04 ± 2.07 kg / m^2 among boys and 23.25 ± 1.91 kg / m^2 among girls. The prevalence of overweight among adolescents was 13.18% (5.31% among boys and 15.74% among girls) and obesity was overall 1%. *Conclusion*: With this study we would like to place a take home message that the over weight and obesity is highly prevalent among adolescents. The onset of the same is noted as early as in childhood. The late adolescent is the best time for the primary prevention.

Keywords: Adolescents; Overweight; Obesity; BMI.

Introduction

Childhood obesity is one of the most serious public health challenges of the 21st century. The problem is global and is steadily affecting many low- and middle-income countries, particularly in urban settings. [1]Overweight and obesity among the adolescents is in the rising trend that often begins in childhood. Overweight for children is defined as a BMI at or above the 85th and less than the 95th percentile, and obesity is defined as a BMI greater than the 95th percentile for age and gender (NIH, 2012)[2]. Adolescent overweight and obesity is the leading cause of morbidity in adulthood. The prevalence of overweight and obesity have doubled among children and tripled among adolescent over past 30 yrs. (CDC, 2013)[3,4]. The prevalence has increased at an alarming rate. Prevalence varies within the country because of differences in the lifestyle, mainly in the dietary patterns, and physical activity. In addition to this urbanization and industrialization are the main culprits for the increase in the prevalence of childhood and adolescent obesity. No published literature can be found in this part of the country to assess the prevalence and determinants of obesity among adolescents.

Materials and Methods

This study was a community based cross-sectional study carried out over a period of six months, from November 2013 march 2014 . Study sample was calculated with a reference value of 11% of the general

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adolescent population are overweight in India.. A total of 240 subjects were selected for this study. A multistage stratified random sampling procedure was adopted. colleges were selected by a simple random technique.

The subjects were adolescents, 16 to 18 years of age, of raichur city, Karnataka, India. After reaching the concerned school, the classes were selected randomly. The adolescents were chosed by simple random techinique using student database of the school. The adolescents were weighed using a electronic weighting machine with an error of ±100 g by Trained investigators. The weighing scale was regularly checked with known standard weights. A portable anthropometric rod was used for measuring the height, with an error to the nearest of 0.1 cm, using standard procedures [6]. The BMI was calculated with the standard formula. The WHO reference, BMI for age were used to classify into overweight and obesity. Adolescents were categorized into two groups namely overweight (e" eighty-fifth percentile) and obese (e" ninety-fifth percentile). The socioeconomic status was assessed based on the Kuppuswamy classification. Analysis was done using SSPS version 11.0 . For all statistical tests, $P < 0.05$ was taken as the significant level.

Results

A total of 240 adolescents in the age group of 12 to 18 years were analyzed. Out of these 132 (55%) subjects were males. The mean BMI of the sample was 21.04 ± 2.07 kg/m^2 among boys and 23.25 ± 1.91 kg/m^2 among girls. The prevalence of overweight among adolescents was 13.18% (5.31% among boys and 15.74% among girls) and obesity was overall 1% [Table 1).

Table 1: Table displaying Mean weight and mean height for respective recorded in our study

Age	Sex	n	Mean weight	Mean height	Mean BMI
16 yrs	Male	60	55.98±4.03	166.83±4.60	20.19±1.82
16 yrs	Female	52	58.69 ± 3.40	158.92 ± 3.70	23.28 ± 1.96
17yrs	Male	48	61.70±3.16	169.98±5.18	21.44±1.86
17yrs	Female	35	59.62 ± 5.56	160.37 ± 3.98	23.10 ± 1.89
18yrs	Male	24	62.12±4.46	166.83±4.43	22.37±2.15
18yrs	Female	21	60.90 ± 5.43	161.38 ± 5.07	23.38±1.91

Table 2: Prevalence of overweight and obesity according to its determinants N = 240

Sex	Weight	Height	BMI	Underweight	Normal	Overweight	Obese
Male	59.18 ±4.79	167.98 ± 4.99	21.04 ± 2.07	14 (10.6%)	111 (84.09%)	7 (5.31%)	0 (0%)
Female	59.42 ± 4.65	159.87 ± 4.17	23.25 ± 1.91	1 (0.92%)	89 (82.42%)	17 (15.74%)	1 (0.92%)

Discussion

The overall prevalence of overweight adolescents among the study group was found to be 13.18%, which was consistent with a recent study.(2)[8] However, the National Nutrition Monitoring Bureau surveys in 2002, in rural areas, reported the prevalence of as little as 0.6%. the urban adolescents studied (7.2%) was 10 times higher then that of their rural counterparts[9] However, the prevalence was lower in this study compared with studies carried out in cities such as Ludhiana, Punjab, Pune, Maharashtra, Dehli, Chennai, and Tamil Nadu[13]. A similar study done in Hyderabad showed that the prevalence of overweight was 7.2% among the 12 to 17 year age group[10]. Although, some other studies done in India showed a higher prevalence of overweight and obesity [11-14]. A study in Delhi on affluent school children showed the prevalence of obesity to be 7.4%[15]. Another study among affluent girls in Delhi reported the prevalence of obesity and overweight to be 5.3 and 15.2%, respectively[16]. Similar studies had been conducted to assess the prevalence of overweight and obesity in India and the results are comparable to our study, with respect to the prevalence of obesity[15,17]. A study done in USA during 2001–2002 showed the prevalence of overweight and obesity as 31.5 and 16.5%, respectively, for the 6 to 19 year age group[18]. A clear socioeconomic gradient in the prevalence of overweight and obesity was observed in this study, which was consistent with other studies [10,11,20]. The prevalence was marginally higher among girls compared with boys ($p > 0.05$), as observed in many international studies. The results revealed that regular physical activity was an important factor in reducing the prevalence of overweight and obesity, which was consistent with other studies[10,11]. Overweight and obesity were marginally higher in

the pubertal age group, i.e., 13 to 15 years of age, as was observed in other studies in Delhi[13] and Chennai[13], perhaps because of increased adipose tissue and overall body weight in children during puberty. The prevalence of overweight and obesity were marginally less in the postpubertal period (16 to 17 years of age). It has been reported earlier that the number of fat cells increases during periods of rapid growth up to 16 years of age, after which increased fat ordinarily accumulates by increasing size of the fat cells already present[13]. Freedman et al.[11] showed the adverse effects of overweight in their 17-year follow-up study and reported that an early average increase of 0.5 kg/m^2 of BMI in children increases the risk for hypertension, dyslipidemia, and type 2 diabetes a decade later. It is interesting to note that <"8% of adolescents perceived that they were overweight, which indicates that the self-reporting of obesity could also be a good indicator of the problem. There is an urgent need to educate the urban community on the aspects of healthy food habits and desired lifestyles to prevent overweight/obesity and its associated ill effects.

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To Assess Determinants of Impact of Fast Food on the Present Scenario of Childhood Obesity

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Abstract

'Eat healthy and live healthy' is one of the essential requirements for long life. Childhood obesity is a serious epidemic, affecting children across the world. In our country alone, 17% of all children and adolescents are now obese, triple the rate from just a generation ago (Centers for Disease Control and Prevention [CDC], 2011). Fast food consumption is one potential cause that has received widespread attention. Many researchers have looked at the relationship between fast food and childhood obesity from various angles. Some of these include the influence of family, the media, and the proximity of fast food restaurants to schools and homes. Examining the interrelationships of these angles can lead to a better understanding of the relationship between childhood obesity and fast food, and from this multi-angle viewpoint, we can see that no single aspect is solely to blame. Diseases like coronary artery disease and diabetes mellitus have seen a profound rise in developing countries and such unhealthy junk food consumption is one of the notable factors to its contribution. This global problem of consuming junk food on a large scale and its impact on health needs emphasis and health education which can greatly contribute to its limited consumption and switching over to healthy eating habits for the better living. **Aim:** To examine the inter-relationship between childhood obesity and fast food & assess the various factor affecting childhood obesity. *Objectives***:** To Examine relationship between childhood obesity and fast food. *Materials & Methods*: children in various 8 community health centres .Inclusion criteria: Children from 2-12 years of age. Exclusion criteria: Children with obese parents: "Constitutional obesity" Children with chronic systemic diseases. *Result***:** the characteristics of the 312, Marathis, jains, Bengalis, and Sikhs children whose parents completed the survey. half the children were female. Children ranged from 2 to 12 years of age, 58% were younger than 7. Approximately 2/ 3rd of the children were Gujaratis, Marathis constituted the next largest group (17%). Parents' education ranged from second grade to a professional school degree, and (68%) had completed a high school degree (41.7%) or less (26%). Household income ranged from 10,000 - 75,000 per year, and most parents (70%) were in the category of less than 30,000 per year. Income and education differed across ethnicity: Sikhs had significantly lower levels of education than all other participants, and Gujaratis had the highest. Furthermore, Sikhs had the lowest income level, and Gujaratis had the highest. As we expected a high proportion of the children were overweight (23%) or at risk of becoming overweight (14%), which is higher than would be expected for children in this age range. In terms of overweight status across the various ethnic groups, 33% of the Gujarati children, 25% of the children identified as mixed, 18.5% of the Marathi, 18.4% of the child bengali, and 15.4% of the sikh children were overweight. *Conclusion***:** the results of this study show that fast-food influences parents' behavior with respect to feeding their children. Thus, for a more comprehensive understanding of approaches to reduce childhood obesity and related cardiovascular risk factors, research that assesses the influence of Fast food on children's eating behaviors and policy debates about food marketing to children should consider parents's exposure.

Keywords: Not Provided

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Introduction

The public health concern with fast-food marketing lies in the proposed relationship between fast-food consumption and obesity in both children and adults, as well as in the nutritional profile of most fast-food menus. The basic cause of obesity is an imbalance between the amount of energy taken in, through eating and drinking, and the amount of energy expended through metabolism and physical activity—and, in the case of children, through energy deposition for growth. It is estimated that, in children, a sustained imbalance of approximately 2% of energy results in the development of obesity over time.[1] Foods' energy density is a key determinant of energy intake, and most fast foods have extremely high energy density . Physiologically, humans are poorly able to differentiate between high- and low-energy density foods.Consequently, it is difficult for people to regulate energy balance, and passive overconsumption can occur . Research indicates that fast-food consumption leads to excess energy intake and, in turn, increased risk of overweight and obesity.Frequent fast-food consumption is also a health concern because most fast foods are rich in saturated fats, trans fats, simple carbohydrates, and sodium—all of which are nutrients associated with hypertension, cardiovascular disease, and type 2 diabetes (World Health Organization 2003).[3] We first review the relationship between fast-food consumption and obesity and the influence of 1)marketing on the relationship. 2) ethnic minority populations. We report the results of an exploratory empirical study that examines

1. The relationships among parents' perceptions of their exposure to fast-food promotion.
2. Their attitudes and normative beliefs about fast food.
3. Frequently their children eat fast food.

We then describe the limitations of the results in some detail to provide guidance for further research. Last, we discuss the implications of the results in terms of their relevance to public polices and the design of social marketing interventions for obesity prevention and, ultimately, for children's health.[4]

Fast-Food Marketing

In 2003, the fast-food market grew 2.6% to reach $148.6 billion in sales.The industry's marketing and promotional strategies emphasize the convenience, taste, and low cost of fast food. Product development is important to the industry because taste is so important to consumers.The increase in fast-food distribution to create ease of access for consumers is also a key marketing strategy.[5 Fast-food franchises are found in gas stations, department stores, zoos, schools, and other nontraditional outlets, which enables consumers to eat in the midst of performing other activities. Fast-food promotions, especially advertising and in-store promotions, are important components of fast-food marketing. Advertising creates overall awareness and establishes brand equity. Fast food accounts for almost 30% of food advertising, and this amount has been growing steadily over the years. Price promotions create awareness of specific menu items, provide purchase incentives, or create repeat purchases among frequent patrons.[6]

Parents' Role in Children's Fast-Food Consumption

Parents influence children's eating habits through their implicit and explicit modeling of food consumption behavior. For example, the children of parents who consume fruits and vegetables do the same. Likewise, the children of parents who consume large amounts of fast food may also do the same. Thus, parents influence children's eating habits through the foods they purchase for and serve in the household, as well as through their selection of places to eat and foods to buy. From this perspective, parents influence children's exposure to particular foods and potentially their habits and preferences. Children who develop particular habits and preferences in childhood may establish them as a lifelong pattern. Research on intergenerational influences demonstrates how information, beliefs, and resources are transmitted from one generation to the next and implies a particular mechanism by which parents' attitudes and beliefs related to fast food affect children's fast-food consumption [7].Parents' brand preferences create comfort in children and set the stage for compliance with their children's request for a brand. The formation of children's attitudes and beliefs about fast food in the con-text of family life may imbue the attitudes and beliefs with sustaining characteristics over time.

Ethnic Minority Populations

Beliefs related to fast food and fast-food consumption may also differ among various ethnic groups. Understanding any potential ethnic variation is important because, in our country, rates of child-hood and adult obesity, diabetes, and cardiovascular disease are significantly higher among certain ethnic minority populations.

Materials & Methodology

We conducted a cross-sectional study at eight CHCs in medically underserved communities. We selected the centers on the basis of distribution in urban and rural locations, interest in participation, and availability of adequate time and resources for data collection. For the initial subject-sampling strategy, we randomly selected parents of children aged 5 to 12 from the 8 CHCs using a centralized file of chart numbers of children within the age group. We provided each CHC with a table of numbers corresponding to each day and instructed the study administrators to approach all families for which a child of the eligible age had a chart number finishing with the numbers. Because of recruitment difficulties, it was necessary to expand efforts to include on-site recruitment of children using a randomized process with medical record numbers. Specific recruitment challenges involved unreliable or missing contact information, limited telephone access, lack of availability, mistrust of research, and limited transportation.

Measures

We designed measures to capture parents' self-reports of five key constructs:

- Fast-food access.
- Exposure to fast-food promotion
- Fast-food attitudes
- Social norms about fast food,
- Their children's fast-food consumption

We developed the fast-food access and expo-sure to fast-food promotion measures specifically for this study to reflect observed fast-food marketing strategies and tactics. We adapted the fast-food attitudes and social norm measures from those used in prior research on the influence of attitudes and norms on consumption. We measured the access, promotion, and social norms variables on five-point scales, where 1 = "disagree," and 5 = "agree."We measured attitudes on five-point semantic differential scales, where 1 = "negative," and 5 = "positive" We measured parents' perceptions of fast-food access by their agreement with two items: "I can easily walk to several fast-food restaurants", and "I can easily drive or take public transportation to fast-food restaurants" .Because the responses to access variables clustered at extreme ends of the distribution, we combined items for analytic purposes.

We created a three-level ordinal variable with the following categories:

1. cannot easily walk or drive,
2. can either walk or drive.
3. can easily both walk and drive.
4. We measured parents's perceived exposure to fast-food promotion by their degree of agreement or disagreement with the item "My local fast-food restaurants often have special deals."

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Source of Data

Children in various 8 community health centres.

Inclusion Criteria

Children from 5-12 years of age.

Exclusion Criteria

Children with obese parents : "Constitutional obesity"

Children with chronic systemic diseases.

Statistical Method Involved

- The data collected will be analyzed statistically

using descriptive statistics namely Mean, Standard Deviation, Percentage where ever applicable.

- Chi square and t-test are used for comparison.
- SPSS 17 software will be used for analysis.

Result

The characteristics of the 312, Marathis, jains, Bengalis, and Sikhs children whose parents completed the survey. half the children were female. Children ranged from 2 to 12 years of age, 58% were younger than 7. Approximately 2/ 3rd of the children were Gujaratis, Marathis constituted the next largest group (17%). Parents' education ranged from second grade to a professional school degree, and (68%) had completed a high school degree (41.7%) or less (26%). Household income ranged from 10,000 - 75,000 per year, and most parents (70%) were in the category of less than 30,000 per year. Income and education differed across ethnicity: Sikhs had significantly lower levels of education than all other participants, and Gujaratis had the highest. Furthermore, Sikhs had the lowest income level, and Gujaratis had the highest.As we expected a high proportion of the children were overweight (23%) or at risk of becoming overweight (14%), which is higher than would be expected for children in this age range. In terms of overweight status across the various ethnic groups, 33% of the Gujarati children, 25% of the children identified as mixed, 18.5% of the Marathi, 18.4% of the child bengali, and 15.4% of the sikh children were overweight.

Table 1: Result of the study

	Total sample	312	100%
	Female	162	52
	Younger than 7	181	58
Race	Sikh	52	17
	Gujarati	25	9
	Marathi	100	33
	Buddhist	114	37
	Mixed	12	4
INCOME	<30,000	190	70
	30-54000	48	17.6
	>54,000	35	12.4
Education	12th Grade	81	26
	High school	130	41.7
	College	87	27.9
	Urban	200	64
	Overweight	72	23
	Risk of overweight	44	14

Conclusion

The results of this study show that fast-food influences parents's behavior with respect to feeding their children. Thus, for a more comprehensive understanding of approaches to reduce childhood obesity and related cardiovascular risk factors, research that assesses the influence of Fast food on children's eating behaviors and policy debates about food marketing to children should consider parents's exposure.

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Review Article

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BCG Vaccine

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Abstract

Tuberculosis (TB) is still responsible for 2 million deaths every year despite being a treatable airborne infectious disease. "Consumption" and "Phthisis" were terms historically used to describe TB, which was responsible for one in four deaths in the 19th century. Due to its infectious nature, chronic progression and long treatment, TB is a great burden for society. Moreover the emergence of multi-drug resistant TB and the current TB-HIV epidemic has raised even greater concern. Treating and preventing TB has become a permanent challange since the ancient times. Bacille Calmette-Guérin (BCG) is the only vaccine available today and has been used for more than 90 years with astonishing safety records. However, its efficacy remains controversial. No universal BCG vaccination policy exists, with some countries merely recommending its use and others that have implemented immunization programs.

Keywords: Tuberculosis; BCG; Vaccine.

Introduction

TB infection is characterized by a complex immunologic response, which leads to a unique host-pathogen interaction therefore make it difficult to treat and control. Moreover TB is a poverty related disease and has severe social implications. The introduction of Bacille Calmette-Guérin (BCG) and chemotherapy in the past century marks an important advance in the history of tuberculosis (TB), which accounted for optimism to fight the disease especially in endemic area. To date, BCG remains as the most widely used vaccine worldwide and has been given to more than 4 billion individuals with astonishing safety records[1,2]. Next to BCG, no other vaccines are available for treating TB and of the many new candidates in the pipeline none is close to market use.

History

Mycobacterium Tuberculosis, the intracellular pathogen that causes TB, was discovered in 1882 by Robert Koch and is responsible for more human deaths than any other single pathogen today.[3,4,5]

BCG vaccine is derived from the bovine tuberculosis strain and was first developed in 1921. It was the result of painstaking efforts by the French microbiologist, Albert Calmette, and the veterinary surgeon, Camille Guerin, who performed 231 repeated subcultures over 13 years. It continues to be the only effective vaccine against tuberculosis.

Early last century, hopes were that TB could be conquered by vaccination with the newly developed M. bovis BCG vaccine, isolated by and named after Calmette and Guerin in Lille, France[6]. These hopes

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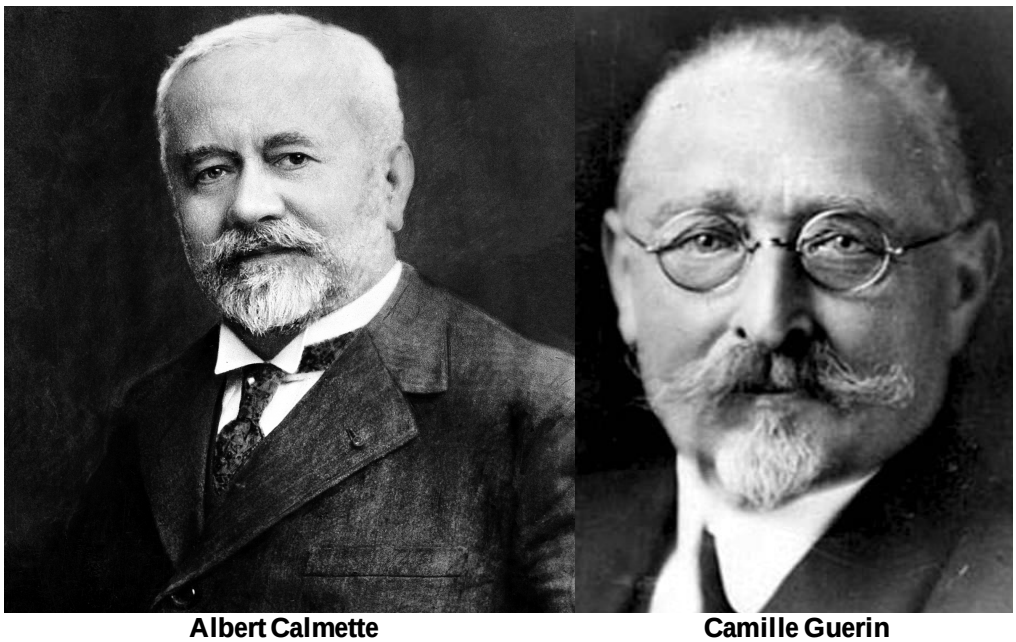

Albert Calmette (1863-1933) **Camille Guerin (1872-1961)**

Fig. 1: Albert Calmette & Camille Guerin)

were further boosted by the development of the first anti-tuberculous drugs during WWII by Selman Waksman, who discovered streptomycin bacteriostatic activity towards Mycobacterium Tuberculosis[7]. Initially, treatment with streptomycin appeared highly efficacious, but the tide turned when drug resistance rapidly developed, an early testimony of Mycobacterium Tuberculosis' ability to acquire drug resistance when treated by single antibiotics. Despite this early writing on the wall, the misconception that TB could be conquered by antibiotics and BCG vaccination led to complacency for several decades. This situation dramatically changed only in the early 1990s, when the World Health Organization (WHO) declared TB a global emergency[8].

In 1900 Albert Calmette and Camille Guérin began their research for an antituberculosis vaccine at the Pasteur Institute in Lille. They cultivated tubercle bacilli on a glycerin and potato medium but found it difficult to produce a homogeneous suspension of the bacilli. In an attempt to counteract their tendency to clump they tried the effect of adding ox bile to the medium and, to their surprise, they noted that subculture led to a lowering of the virulence of the organism. It was this fortuitous observation that led them to undertake heir long term project of producing a vaccine from this attenuated tubercle bacillus[9].

In 1908, starting with a virulent bovine strain of tubercle bacillus supplied by Nocard (originally isolated by him in 1902 from the udder of a tuberculous cow), they cultured it on their bile, glycerine and potato medium and then proceeded to subculture at roughly three weekly intervals. By 1913 they were prepared to initiate a vaccination trial in cattle which was interrupted by outbreak of World War I. Subculturing was continued throughout the German occupation of Lille, despite the greatly increased cost of potatoes and the difficulty of obtaining suitable ox bile from the abattoir. Yet, they managed to obtain this by grace of the veterinary surgeons of the German occupying force. By 1919, after about 230 subcultures carried out during the previous 11 years, they had a tubercle bacillus which failed to produce progressive tuberculosis when injected into guinea pigs, rabbits, cattle, or horses. At Guerin's suggestion, they named it Bacille Bilie Calmette-Guerin; later they omitted "Bilie" and so BCG was born[9].

In 1921, Calmette decided that the time was ripe for a trial of the vaccine in man. The first human administration of BCG was by Benjamin Weill-Halle (1875-1958) assisted by Raymond Turpin (1895-1988) at the Charité Hospital, Paris. A woman had died of tuberculosis a few hours after giving birth to a healthy infant. On 18 July 1921, Weill-Halle and Turpin gave a dose of BCG by the oral route to the infant. There were no undesirable sequelae. The oral route was chosen since Calmette considered the gastrointestinal tract to be the usual route of natural infection by the tubercle bacillus. Weill-Halle then tried the subcutaneous and cutaneous routes on other infants but local reactions were objected to by the parents, and so the oral method was continued, an emulsion of BCG prepared by Boquet and Negre being used. By 1924 they were able to report a series of 664 oral BCG vaccinations of infants[10]. The Pasteur Institute at Lille began the mass production of BCG vaccine for use by the medical profession. From 1924 to 1928, 114 000 infants were

vaccinated without serious complications[11]. In 1928, Calmette called Guerin to join him in Paris, since he did not feel it necessary for Guerin to continue the BCG experiments on animals in Lille. By 1931, there was a special laboratory for the preparation of BCG and Guerin was placed in charge.

Current Status in India

Globally, about 1 million cases of pediatric tuberculosis are estimated to occur every year accounting for 10–15% of all tuberculosis (TB)[12]. The exact burden of childhood TB in India is unknown due to diagnostic difficulties but it is estimated to be 10% of the total adult incidence[13]. The proportion of pediatric TB cases registered under RNTCP has shown an increasing trend, from 5.6% in 2005 to 7% in 2011[14]. Prevention of childhood tuberculosis is thus an important priority. However, in comparison to other EPI vaccines, efficacy of BCG vaccine is limited. Several new vaccines against tuberculosis are in development phase, and many are designed to boost pre existing immunity induced by BCG[15] and some candidates aim to ultimately replace BCG as the priming vaccine[16].

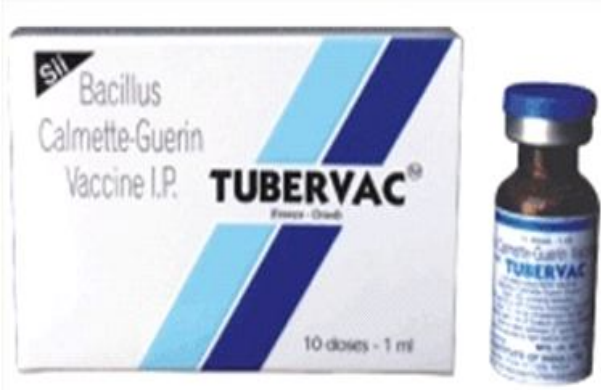

Fig. 2: BCG vaccine

Composition & Method of Administration

The two common strains in use are Copenhagen (Danish 1331) and Pasteur, of which the former was produced in India at the BCG Laboratories, Guindy, Tamil Nadu till recently. BCG induces cell-mediated immunity, but the protective efficacy is a matter of debate and is very difficult to quantify. It has an efficacy of 75–86 % for prevention of miliary and meningeal form of the disease. Protective efficacy for pulmonary tuberculosis is 50%[17].

The vaccine contains 0.1–0.4 million live viable bacilli per dose. It is supplied as a lyophilized (freeze-dried) preparation in vacuum sealed, multi-dose, dark colored ampoules or 2 ml vials with normal saline as diluent. The vaccine is light sensitive and deteriorates on exposure to ultra violet rays. In lyophilized form, it can be stored at 2 to 80 0 C for up to 12 months without losing its potency.

The long necked, BCG ampoule, should be cut carefully by gradual filing at the junction of its neck and body, as sudden gush of air in the vacuum sealed ampoule may lead to spillage of the contents. Diluent should be used for reconstitution. Sterile normal saline may be used if diluent is not available. As the vaccine contains no preservative, bacterial contamination and consequent toxic shock syndrome may occur if kept for long after reconstitution. The reconstituted vaccine should be stored at 2 to 8 0 C, protected from light and discarded within 4–6 hours of reconstitution. The recommended dose is 0.1 ml or 0.05 ml as suggested by the manufacturer of the vaccine. Dosage does not depend on the age and weight of the baby. Injection of BCG should be strictly intradermal, using a tuberculin syringe and a 26G / 27G needle. The convex aspect of the left shoulder at level of deltoid insertion is preferred for easy visualization of the BCG scar and for optimum lymphatic drainage. Other sites such as thigh should be avoided. The selected site may be swabbed clean using sterile saline and local antiseptics should be avoided.

Reaction

A wheal of 5 mm at the injection site indicates successful intradermal administration of the vaccine. Subcutaneous administration of BCG is associated with an increased incidence of BCG adenitis. The injected site usually shows no visible change for several days. Subsequently, a papule develops after 2–3 weeks, which increases to a size of 4–8 mm by the end of 5–6 weeks. This papule often heals with ulceration and results in a scar after 6–12 weeks. The ulcer at vaccination site may persist for a few weeks before formation of the final scar. No treatment is required for this condition.

Adverse Effects

Secondary infection at the vaccination site may require antimicrobials. Ipsilateral axillary / cervical lymphadenopathy may develop a few weeks / months after BCG vaccination. Antitubercular therapy is of no benefit in such situations and should not be administered. The nodes regress spontaneously after a few months. It should also be noted that if fine needle aspiration cytology of the nodes is carried out, stain for acid-fast bacilli may be positive. These are bovine vaccine bacilli and should not be misconstrued as being suggestive of tuberculous disease. In some

children, the nodes may even liquefy and result in an abscess. Surgical removal of the nodes or repeated needle aspiration is the treatment of choice; again antitubercular therapy is not recommended. Disseminated BCG infection is extremely unusual but may occur in children with cellular immunodeficiency.

BCG & HIV

BCG should be avoided in the immunocompromised, especially those with cellular immunodeficiency; it may, however, be given at birth to children born to HIV positive mothers. BCG may be given with other vaccines on the same day or at any interval with the exception of measles / measles mumps rubella (MMR) vaccine where a gap of 4 weeks between the two vaccines is recommended.

Conclusion

Although the efficacy of the BCG vaccine continues to be controversial, live attenuated BCG is still the only vaccine in use for the prevention of TB in humans. It is effective against the severe forms of TB and its use prevents a large number of deaths that would otherwise be caused by TB every year. The choice of the BCG strain to be used for vaccination remains an important issue. Currently, it is difficult to determine which strain should be used and further detailed analysis of the genomics and immunogenicity of BCG sub-strains may provide an answer to this important question. The World Health Organization and the International Union against Tuberculosis and Lung Disease could identify the BCG sub-strains that provide the best protection and recommend them for future vaccination.

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Include summary of key findings (primary outcome measures, secondary outcome measures, results as they relate to a prior hypothesis); Strengths and limitations of the study (study question, study design, data collection, analysis and interpretation); Interpretation and implications in the context of the totality of evidence (is there a systematic review to refer to, if not, could one be reasonably done here and now?, What this study adds to the available evidence, effects on patient care and health policy, possible mechanisms)? Controversies raised by this study; and Future research directions (for this particular research collaboration, underlying mechanisms, clinical research). Do not repeat in detail data or other material given in the Introduction or the Results section.

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Standard journal article

[1] Flink H, Tegelberg Å, Thörn M, Lagerlöf F. Effect of oral iron supplementation on unstimulated salivary flow rate: A randomized, double-blind, placebo-controlled trial. J Oral Pathol Med 2006; 35: 540-7.

[2] Twetman S, Axelsson S, Dahlgren H, Holm AK, Källestål C, Lagerlöf F, et al. Caries-preventive effect of fluoride toothpaste: A systematic review. Acta Odontol Scand 2003; 61: 347-55.

Article in supplement or special issue

[3] Fleischer W, Reimer K. Povidone iodine antisepsis. State of the art. Dermatology 1997; 195 Suppl 2: 3-9.

Corporate (collective) author

[4] American Academy of Periodontology. Sonic and ultrasonic scalers in periodontics. J Periodontol 2000; 71: 1792-801.

Unpublished article

[5] Garoushi S, Lassila LV, Tezvergil A, Vallittu PK. Static and fatigue compression test for particulate filler composite resin with fiber-reinforced composite substructure. Dent Mater 2006.

Personal author(s)

[6] Hosmer D, Lemeshow S. Applied logistic regression, 2nd edn. New York: Wiley-Interscience; 2000.

Chapter in book

[7] Nauntofte B, Tenovuo J, Lagerlöf F. Secretion and composition of saliva. In: Fejerskov O, Kidd EAM,

editors. Dental caries: The disease and its clinical management. Oxford: Blackwell Munksgaard; 2003. p. 7-27.

No author given

[8] World Health Organization. Oral health surveys - basic methods, 4th edn. Geneva: World Health Organization; 1997.

Reference from electronic media

[9] National Statistics Online—Trends in suicide by method in England and Wales, 1979-2001. www.statistics.gov.uk/downloads/theme_health/HSQ 20.pdf (accessed Jan 24, 2005): 7-18. Only verified references against the original documents should be cited. Authors are responsible for the accuracy and completeness of their references and for correct text citation. The number of reference should be kept limited to 20 in case of major communications and 10 for short communications.

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