International Journal of Food Science and Nutrition
ISSN: 2455-4898; Impact Factor: RJIF 5.14
Received: 20-01-2019; Accepted: 27-02-2019
www.foodsciencejournal.com
Volume 4; Issue 2; March 2019; Page No. 130-135

A review on anaemia and its management

Souvik Tewari
PhD Scholar, Department of Food science and Technology, Department of Warner College of Dairy Technology, Sam Higginbottom University of Agriculture, Technology and Sciences (SHUATS), Allahabad, Uttar Pradesh, India

Abstract
Anaemia is a common clinical disorder. According to WHO (2004) reports, one third of the global populations (over 2 billion) are anaemic due to imbalance in their nutritious food intake. All living things contain liquid inside their bodies which serves many functions like blood act as important connective tissue in transport of oxygen to all the cells similarly to excrete all the waste material from the cell to out sidethe body. Blood cells contain a red coloured pigment, which is called hemoglobin and its central portion is iron. When the level of haemoglobin falls in the body this condition is called anaemia. In this review I investigate anaemia types, causes, symptoms, diagnostic test and their management.

Keywords: hemoglobin, anaemia, iron, folic acid

Introduction
Anaemia is a condition in which there is diminished O2 capacity of the blood, as a result of reducing in total circulating hemoglobin and reducing in red cell mass.
Anemia is a global public health problem affecting both developing and developed countries at all age group. According to the World Health Organization (WHO), "Anemia is defined as hemoglobin (Hb) levels <12.0 g/dL in women and <13.0 g/dL in men". However, normal Hb distribution varies not only with sex but also with ethnicity and physiological status. New lower limits of normal Hb values have been proposed, according to ethnicity, gender, and age. Anemia is often multifactorial and is not an independent phenomenon. (Cappellini, M.D., Motta, L. 2015).

Cut of value of hemoglobin

Table 1

Children	
Birth	13.5 to 24.0 g/dl (mean 16.5 g/dl)
<1 mth	10.0 to 20.0 g/dl (mean 13.9 g/dl)
1-2 mths	10.0 to 18.0 g/dl (mean 11.2 g/dl)
2-6 mths	9.5 to 14.0 g/dl (mean 12.6 g/dl)
0.5 to 2 yrs	10.5 to 13.5 g/dl (mean 12.0 g/dl)
2 to 6 yrs	11.5 to 13.5 g/dl (mean 12.5 g/dl)
6-12 yrs	11.5 to 15.5 g/dl (mean 13.5)
Females	
Age 12-18 yrs	12.0 to 16.0 g/dl (mean 14.0 g/dl)
Age >18 rs	12.1 to 15.1 g/dl (mean 14.0 g/dl)
Males	
12-18 yr	13.0 to 16.0 g/dl (mean 14.5 g/dl)
>18 yrs:	13.6 to 17.7 g/dl (mean 15.5 g/dl)

Nutritional Anaemia
Nutritional anaemia may be defined as the condition of reduction of normal hemoglobin concentration or amount of inadequate supply of one or more nutrients. Nutritional anaemia may occurs due to deficiency of any dietary nutrient that involves in the formation of hemoglobin.

- **Types of Anaemia**

There are several types and classifications of anaemia. The occurrence of anaemia is due to the various red cell defects such as production defect (aplastic anaemia), maturation defect (megaloblastic anaemia), defects in haemoglobin synthesis (iron deficiency anaemia), genetic defects of haemoglobin maturation (thalassaemia) or due to the synthesis of abnormal haemoglobin (haemoglobinopathies, sickle cell anaemia and thalassaemia) and physical loss of red cells (haemolytic anaemias) (Mukherjee and Ghosh, 2012) [6].

1. Iron-Deficiency Anaemia
Iron is one of the most important nutrient for hemoglobin synthesis in human body. Iron deficiency anaemia is a condition in which the body has too little iron in the bloodstream. This form of anaemia is more common in adolescents and in women before menopause. Blood loss from heavy periods, internal bleeding from the gastrointestinal tract, or donating too much blood can all contribute to this disease (1).

- **Causes**

A. Inadequate iron intake
If the person does not intake adequate amount of iron rich foods like beans, lentils, tofu, baked potatoes, cashews and dark green leafy vegetables such as spinach, fortified breakfast cereals, whole-grain and enriched breads etc then the person suffer from iron deficiency anaemia.

B. Inadequate utilization of iron
This can take place secondary to chronic gastro intestinal disturbances, defective release of iron from the iron store in to the plasma and defective iron utilization in other chronic disorder.

C. Increased requirement
Anaemia can occurs during periods of accelerated demands likes in infancy, adolescence, pregnancy,and lactation.

D. Inadequate absorption of iron

Inadequate iron absorption occurs during diarrhea, in chronic renal disease, excessive amount of phytates and phosphates in the diet and excessive consumption of tea can decreases the absorption of iron.

- **Symptoms**
- Diminished work performance
- Cognitive development
- Behavior implication
- Tiredness

- **Lethargy**
- Feeling faint and becoming breathless easily
- Headaches, irregular heartbeats (palpitations)
- Altered taste, sore mouth and ringing in the ears (tinnitus).

- **Management**
- Oral administration of inorganic iron in the ferrous form, ferrous sulfate 50-200 mg/day 3 times for adults and 6mg/ kg for children.
- Ascorbic acid greatly increases iron absorption through its capacity, to maintain iron in the reduce state.
- Iron therapy should be continued for several month after re-absorption of normal hemoglobin levels.

2. Pernicious anaemia

Pernicious anaemia is the most common cause of Vitamin B12 deficiency. Vitamin B12 is essential for human health. It is needed to make new cells in the body such as the many new red blood cells which are made every day. Meat, fish, eggs, and milk these are good source of Vitamin B12. A lack of vitamin B12 leads to Pernicious anaemia and sometimes to other problems. A lack of vitamin B12 (B12 deficiency) is one cause of Pernicious anaemia. Pernicious anaemia usually develops over the age of 50 years. In this type of anaemia, women are more commonly affected than men, and it tends to run in families. It occurs more commonly in people who have other autoimmune diseases. Certain medicines used also may affect the absorption of vitamin B12. (Turner and Talbot, 2009).

Causes

A. Inadequate ingestion

Chronic alcoholism, poverty, releasing taboos and dietary fads can cause pernicious anaemia

B. Inadequate utilization

This is due to the presence of vitamin B12 antagonized.

C. Inadequate absorption

Inadequate or absence of secretion of intrinsic factos due to hereditary and endocrine disorder associated with GI damage.

- **Symptoms**

Psychological problems like depression, confusion, difficulty with memory or even dementia and Nervous problems like numbness, pins and needles, vision changes and unsteadiness.

Common symptoms are

- Abnormal discomfort
- Anorexia
- Weight loss
- General weakness
- Redness and swelling (inflammation) of the tongue (glossitis)
- Frequent diarrhoea

- **Management**
- High protein diet (100-150 gm) of protein with high calorie diet is required.
- Spices and condiments food should be avoided from the diet
- A soft diet is recommended for anorexia and irritation of GI patient.
- Ascorbic acid is given to the patient diet and high protein and high calorie should be given between 2-3 times daily.

3. Haemolytic Anaemia

Haemolytic anaemia is a condition in which red blood cells are destroyed and removed from the bloodstream before their normal lifespan is up. Haemolytic anaemia can affect people of all ages, races and sexes.

- **Causes of hemolytic anemia may include**
- Inherited conditions, such as sickle cell anemia and thalassemia
- Stressors such as infections, drugs, snake or spider venom, or certain foods
- Toxins from advanced liver or kidney disease
- Inappropriate attack by the immune system (called hemolytic disease of the newborn when it occurs in the fetus of a pregnant woman)
- Vascular grafts, prosthetic heart valves, tumors, severe burns, exposure to certain chemicals, severe hypertension, and clotting disorders
- In rare cases, an enlarged spleen can trap red blood cells and destroy them before their circulating time is up.

- **Symptoms**
- Fatigue,
- Pain
- Arrhythmias
- An enlarged heart and heart failure.

- **Treatment**

Treatments for haemolytic anaemia include blood transfusions, medicines, plasmapheresis, surgery, blood and marrow stem cell transplants and lifestyle changes.

4. Sickle cell anaemia

Anaemia in which the body makes sickle-shaped ("C"-shaped) red blood cells is called Sickle Cell anaemia. It is a rate genetic or hereditary disease. It occurs due to the presence of an abnormal hemoglobin (HbS). It differs from the normal hemoglobin (HbA) by the presence of valine. In which the body makes sickle shaped red blood cell. Sickle shaped means that the red blood cells are shaped like a crescent.

- **Symptoms**
- Severe pain
- Chest pain and difficult breathing

- Strokes
- Severe infection

- **Dietary Management**
- High calorie and high micro nutrient diet should be needed to the patient diet, who suffer from sickle cell anaemia.
- Whole green leafy vegetable, legumes and fruits should be included in the diet to prevent deficiency.
- Fruit supplement food should be given to prevent the hydration.
- One multivitamin tablet should be given to the patient diet.
- Vitamin E,B6,A and minerals like Zink should be provided.
- Omega-3 fatty acid rich food like olive oil, ground nut and fish etc should be given to the patient diet.

- **After over a decade of preclinical research and development, a new gene therapy process is reversing sickle cell anaemia symptoms in patients.**

The gene therapy process uses reduced-intensity preconditioning to enhance its global transportability, therefore easing movement of the treatment across resource-challenged parts of the world where the disease is most common and sickle cell anaemia symptoms could develop.

- **Gene therapy in clinical trials for sickle cell anemia**

Twelve clinical trials studying gene therapy to treat sickle cell anemia are now ongoing. Nine of the 12 are currently recruiting participants.

Four trials (NCT02186418, NCT03282656, NCT02247843, NCT02140554) are testing the efficacy and safety of gene therapy to replace the mutated HBB gene with a healthy HBB gene. These Phase 2 trials are recruiting both children and adults in the United States and Jamaica.

Three trials (NCT02193191, NCT02989701, NCT03226691) are investigating the use of Mozobil (plerixafor) in patients with sickle cell anemia to increase the production of stem cells to be used for gene therapy. This medication is already approved to treat certain types of cancer. All three are recruiting U.S. participants.

One trial (NCT00669305) is recruiting sickle cell anemia patients in Tennessee to donate bone marrow to be used in laboratory research to develop gene therapy techniques.

The final study (NCT00012545) is examining the best way to collect process and store umbilical cord blood from babies with and without sickle cell anemia. Cord blood contains abundant stem cells that could be used in developing gene therapy for sickle cell anemia. This trial is open to pregnant women in Maryland — both those who risk having an infant with sickle cell anemia, and those who do not.

One clinical trial (NCT02151526) conducted in France is still active but no longer recruiting participants. It is investigating the efficacy of gene therapy in seven patients. For the trial, a gene producing therapeutic hemoglobin that functions similarly to fetal hemoglobin is introduced into the patient's stem cells. A case study from one of the seven was published in March 2017; it showed that the approach was safe and could be an effective treatment option for sickle cell anemia.

- **New approaches to the management of anemia of chronic kidney disease.**

Anemia is a common and debilitating condition seenworldwide but it is specially common and severe in patientswith chronic kidney disease (CKD) (peritubular cells beingthe source of erythropoietin) and cancer (secondary to myelosuppressive treatment). Before the introduction of thefirst erythropoiesis-stimulating agent (ESA), recombinant human erythropoietin (rHuEPO), in the late 1980s, blood transfusion and anabolic steroids were the mainstay of treatment. Even though rHu EPO changed the way anemia was managed in patients with renal disease, it does have its limitations including efficacy, duration of activity, route of administration and concomitant iron deficiency, and inflammation. There are several innovative agents including hypoxia-inducible factor stabilizers and erythropoietin-mimetic peptides that are being developed to overcome some of these limitations of rHu EPO and are currently in various stages of clinical testing. Iron deficiency is also very common in patients with CKD because of impaired absorption and mobilization of iron and increased demands for iron when ESAs are administered. There are several novel agents including ferumoxytol, dialysate iron, and iron oligosaccharide that are being evaluated to manage iron deficiency in this patient population.

Epo gene therapy (Erythropoietin) Osada *et al*. reported on the results of gene therapy with the human erythropoietin gene as a method of treating anemia ofrenal origin. They studied mice with polycystic kidney disease, transfected cells with an adenovirus vector and the human EPO gene, and inserted these cells intraperitoneally. There was a significant increase in serum EPO levels and reticulocyte response. Similar results have been reported with human EPO gene therapy in primates. This mode of therapy is very promising but has some potential drawbacks including but not limited to irreversibility of the mode of treatment, possible overexpression, and oncogenic potential.

Diagnostic Evaluation of Anaemia

Anaemia is one of the major signs of disease. It is never normal and its cause(s) should always be sought. The history, physical examination, and simple laboratory testing are all useful in evaluating the anaemic patient. The workup should be directed towards answering the following questions concerning whether one or more of the major processes leading to anaemia.

5. History taking

5.1 Onset, duration and progress

Insidious onset, long duration and gradual progress of symptoms in a patient with anaemia suggests nutritional anaemia, chronic haemolytic anaemia (congenital or acquired), anaemia of chronic disease and anaemia due to chronic blood loss. Rapid onset, short duration and rapid progress of symptoms indicate acute leukaemia, acute haemolytic anaemia, hemolytic/aplastic crisis in chronic haemolytic anaemia, anaemia secondary to acute blood loss and infiltrative disorders of the bone marrow. Presence of symptoms other than those due to anaemia is a pointer to underlying disease causing anaemia and provides clues for further work up of the patient. Inquiry should be made to uncover conditions that may cause gastro-intestinal, genito-

urinary or any other blood loss. An anaemic patient who complains of angina or symptoms of cerebral hypoxia urgently needs the oxygen carrying capacity raised by red cell transfusions and inspired oxygen, whatever may be the cause of anaemia. Passage of dark red or brown urine indicates haemoglobinuria and suggests haemolysis. History of episodes of bone pain, backache, abdominal pain in past suggests the diagnosis of Sickle Cell Disease.

5.3 Age & sex

Anaemia is more common in pregnant women, females in reproductive age and children during the phase of rapid growth. A predominantly cereal based diet which is poor in green leafy vegetables and vitamin C containing foods is a common cause of iron deficiency. In a female patient, a detailed menstrual history and history of reproductive performance (number of deliveries and interval between deliveries), provide information about stress on iron balance and raises the possibility of iron deficiency anaemia. Self imposed or improperly advised dietary restrictions can contribute to nutritional anaemia.

5.4 Drug ingestion

Drug ingestion can cause anaemia in several ways. Long term ingestion of aspirin (in patients of coronary artery disease) can lead to chronic blood loss and iron deficiency anaemia. Certain drugs can cause haemolysis in individuals with G-6-PD deficiency. Rifampicin and alpha methyl dopa can cause autoimmune haemolytic anaemia. Chemotherapeutic drugs can cause marrow depression and pancytopenia. A past history of cardiac valve surgery can indicate the possibility of haemolysis.

6. Physical examination

Clinical examination can provide a wealth of diagnostic information. Although signs are not always present, they can be helpful in making a clinical diagnosis. A smooth (bald) tongue and nail changes of koilonychia (brittle, flat or concave nails, more common in toe nails than in finger nails), and bilateral, painless parotid enlargement in a patient with anaemia suggests the diagnosis of iron deficiency anaemia. Skin pigmentation in the peri-oral region and over the knuckles is suggestive of megaloblastic anaemia. The presence of mild jaundice would suggest possibility of haemolytic anaemia. A generalized greyish discoloration of skin indicates iron overload in anaemic patients who have been given blood transfusions over several years. Skin pigmentation and various skeletal abnormalities may be present in some cases of constitutional hypoplastic anaemia.

The presence of petechial haemorrhages would indicate a marrow infiltrative disease (leukaemia, lymphoma, myeloma, metastases, etc.), they may also be seen in megaloblastic anaemia. Fronto-temporal bossing, malar prominence, upper jaw and teeth projecting beyond the lower jaw, flat bridge of the nose - all giving rise to typical facial appearance are characteristic of Thalassemia syndromes. Puffiness of lower eye lids, loss of eye brown hair and thick voice would suggest myxedema as the cause of anaemia; this can be confirmed by delayed relaxation of muscle after eliciting deep reflexes. The presence of hypertension should alert the clinician to the possibility of anaemia secondary to chronic renal failure. Tenderness of calf muscles suggests megaloblastic anaemia but could be present even in Iron deficiency anaemia. Signs of sub-acute combined degeneration indicate pernicious anaemia. Lymphadenopathy suggests the possibility of leukaemia or lymphoma.

7. Laboratory evaluation

Lab tests in the diagnosis of Anaemia

M/E ratio – ratio of myeloid to erythroid precursors.

Source – Harrison's Principles of Internal Medicine, 16th edition.

7.1 Mean corpuscular volume

The normal range for MCV is from 80 to 100 femtoliters (fL). Values in excess of 115 fL are almost exclusively seen in vitamin B12 or folic acid deficiency. Even higher values can occur as an artifact when cold agglutinins are present, which causes RBCs to go through the counting aperture in doublets or triplets. Low values usually indicate a microcytic anaemia.

7.2 Mean corpuscular haemoglobin

The normal MCH ranges from 27.5 to 33.2 picograms of haemoglobin per RBC. Low values are seen in iron deficiency and thalassemia, while increased values occur in macrocytosis of any cause.

7.3 Mean corpuscular haemoglobin concentration

The mean normal value for the MCHC is 34 grams of haemoglobin per dL of RBCs. Low values occur in the same conditions that generate low values for MCV and MCH, while increased values occur almost exclusively in the presence of congenital or acquired spherocytosis or in other congenital haemolytic anaemia in which red cells are abnormally desiccated (eg, sickle cell anaemia, haemoglobin C disease, xerocytosis).

7.4 Reticulocyte count

The reticulocyte count, either as a percentage of all RBCs, the absolute reticulocyte count, the corrected absolute reticulocyte count, or as the reticulocyte production index, helps to distinguish among the different types of anaemia:

- Anaemia with a high reticulocyte count reflects an increased erythropoietic response to continued haemolysis or blood loss.
- A stable anaemia with a low reticulocyte count is strong evidence for deficient production of RBCs (ie, a reduced marrow response to the anaemia).
- Haemolysis or blood loss can be associated with a low reticulocyte count if there is a concurrent disorder that impairs RBC production (eg, infection, prior chemotherapy).
- A low reticulocyte percentage accompanied by pancytopenia is suggestive of aplastic anaemia, while a reticulocyte percentage of zero with normal white blood cell and platelet counts suggests a diagnosis of pure red cell aplasia.

7.5 White blood cell count and differential

A low total white blood cell (WBC) count (leukopenia) in a patient with anaemia should lead to consideration of bone marrow suppression or replacement, hypersplenism, or deficiencies of cobalamin(B12) or folate. In comparison, a high total WBC count (leukocytosis) may reflect the presence of infection, inflammation, or a hematologic

malignancy.

Clues to the specific abnormality present may be obtained from the WBC differential, which, in conjunction with the total WBC may show increased or decreased absolute numbers of the various cell types in the circulation. Examples include:

- An increased absolute neutrophil count in infection or steroid therapy
- An increased absolute monocyte count in myelodysplasia
- An increased absolute eosinophil count in certain infections
- A decreased absolute neutrophil count following chemotherapy
- A decreased absolute lymphocyte count in HIV infection or following treatment with corticosteroids.

8.6 Neutrophil hyper segmentation

Neutrophil hyper segmentation (NH) is defined as the presence of >5 percent of neutrophils with five or more lobes and/or the presence of one or more neutrophils with six or more lobes. This peripheral smear finding, along with macro-ovalocytic red cells, is classically associated with impaired DNA synthesis, as seen in disorders of vitamins B12 and folic acid.

8.7 Circulating nucleated red blood cells

Nucleated RBCs (NRBCs) are not normally found in the circulation. They may be present in patients with known hematologic disease (eg, sickle cell disease, thalassemia major, various haemolytic anaemia after splenectomy), or as a part of the leukoerythroblastic pattern seen in patients with bone marrow replacement. In patients without known hematologic disease, NRBCs may reflect the presence of a life-threatening disease, such as sepsis or severe heart failure.

8.8 Platelet count

Abnormalities in the platelet count often provide important diagnostic information. Thrombocytopenia occurs in a variety of disorders associated with anaemia, including hypersplenism, marrow involvement with malignancy, autoimmune platelet destruction (either idiopathic or drug-related), sepsis, or folate or cobalamin deficiency. High platelet counts, in comparison, may reflect myeloproliferative disease, chronic iron deficiency, and inflammatory, infectious, or neoplastic disorders. Changes in platelet morphology (giant platelets, degranulated platelets) also may be important, suggesting myeloproliferative or myelodysplastic disease.

8.9 Pancytopenia

The combination of anaemia, thrombocytopenia, and neutropenia is termed pancytopenia. The presence of severe pancytopenia narrows the differential diagnosis to disorders such as aplastic anaemia, folate or cobalamin deficiency, or hematologic malignancy (eg, acute myeloid leukaemia). Mild degrees of pancytopenia may be seen in patients with splenomegaly and splenic trapping of circulating cellular elements(hypersplenism).

8.10 Blood smear

Many clinicians rely on the above RBC parameters and the RDW in evaluating a patient with anaemia. However, the RDW is of limited utility, and examination of the peripheral blood smear provides information not otherwise available. As examples, the automated counter may miss the red cell fragmentation ("helmet cells", schistocytes) of microangiopathic haemolysis, microspherocytes in autoimmune haemolytic anaemia, teardrop RBCs in myeloid metaplasia, a leukoerythroblastic pattern with bone marrow replacement, the "bite cells" in oxidative haemolysis, or RBC parasites such as malaria or babesiosis.

8.11 Serial evaluation of haemoglobin and hematocrit

Measuring the rate of fall of the patient's Hb or HCT often provides helpful diagnostic information. Suppose the Hb concentration has fallen from 15 to 10 g/dL in one week. If this were due to total cessation of RBC production (ie, a reticulocyte count of zero) and if the rate of RBC destruction were normal (1 percent/day), the Hb concentration would have fallen by 7 percent over seven days, resulting a decline of 1.05 g/dL (0.07 x 15). The greater fall in Hb in this patient (5 g/dL) indicates that marrow suppression cannot be the sole cause of the anaemia and that blood loss and/or increased RBC destruction must be present.

8.12 Evaluation for iron deficiency

More complete evaluation for iron deficiency is indicated when the history (menometrorrhagia, symptoms of peptic ulcer disease) and preliminary laboratory data (low MCV, low MCH, high RDW, increased platelet count) support this diagnosis. In this setting, the plasma levels of iron, iron binding capacity (transferrin), transferrin saturation, and ferritin should be measured. This is discussed in detail below.

8.13 Evaluation for haemolysis

Haemolysis should be considered if the patient has a rapid fall in haemoglobin concentration, reticulocytosis, and/or abnormally shaped RBCs (especially spherocytes or fragmented RBCs) on the peripheral smear. The usual ancillary findings of haemolysis are an increase in the serum lactate dehydrogenase (LDH) and indirect bilirubin concentrations and a reduction in the serum haptoglobin concentration. The combination of an increased LDH and reduced haptoglobin is 90 percent specific for diagnosing haemolysis, while the combination of a normal LDH and a serum haptoglobin greater than 25 mg/dL is 92 percent sensitive for ruling out haemolysis.

8.14 Intravascular haemolysis

Serum or plasma haemoglobin and urinary hemosiderin should be measured if intravascular haemolysis is a consideration, as with paroxysmal nocturnal haemoglobinuria.

8.15 Bone marrow examination

Examination of the bone marrow generally offers little additional diagnostic information in the more common forms of anaemia. If erythropoiesis is increased in response to the anaemia, the bone marrow will show erythroid hyperplasia, a nonspecific finding. Similarly, although the absence of stainable iron in the bone marrow had previously been considered the "gold standard" for the diagnosis of iron deficiency, this diagnosis is usually established by laboratory tests alone

Indications for examination of the bone marrow in anaemic patients include pancytopenia or the presence of abnormal cells in the circulation, such as blast forms. Such patients may have aplastic anaemia, myelodysplasia, marrow replacement with malignancy, or a myeloproliferative disease. Other findings that may be seen in the marrow in anaemic patients include megaloblastic erythropoiesis (folate or cobalamin deficiency), absence of recognizable RBC precursors (pure RBC aplasia), vacuolization of RBC precursors (alcohol or drug-induced anaemia), and increased iron-laden RBC precursors (the sideroblastic anaemia).

8.16 Multiple causes of anaemia

Multiple causes are frequently present in adults, particularly the elderly. Common examples are:

- A patient with gastrointestinal bleeding secondary to colon cancer may also have the anaemia of chronic disease, leading to a blunted reticulocyte response.
- A patient with a chronic hemolytic anaemia (eg, sickle cell anaemia, hereditary spherocytosis) may develop worsening anaemia following acute infection, particularly with parvovirus B19, which may blunt or temporarily ablate erythropoiesis and the reticulocyte response.
- A patient with autoimmune hemolytic anaemia may develop worsening anaemia from gastrointestinal blood loss following treatment with corticosteroids.
- Anaemia, renal failure, and congestive failure are often found together, a condition that has been termed "cardio-renal anaemia syndrome." Treatment of the anaemia may improve both the renal failure and heart failure.

Conclusion

Anaemia is a frequent clinical finding, often leading to significant ill health and always requires prompt investigation and selective treatment. By following a simple escalation pathway from history, examination and targeted investigations, a diagnosis can usually be made and effective treatment applied.

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