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AN AYURVEDIC PERSPECTIVE OF PANDUROGA W.S.R TO IRON DEFICIENCY ANAEMIA IN CHILDREN -A REVIEW

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ABSTRACT

Pandu roga was well known to Indian people since the Vedic period. In *pandu roga*, the colour of the body changes like pallor of skin, sclera, nail, and tongue due to *Rakta, alpata* means Haemoglobin level decreases than the normal level. It is related to two crucial *dhatu* known as *Rasa Dhatu* and *Rakta Dhatu*. We can correlate this disease to Iron Deficiency Anemia in modern science. Iron Deficiency anaemia is a global public health problem among young children and pregnant women. Iron Deficiency anaemia is often caused by blood loss or malabsorption. Iron Deficiency anaemia and *pandu roga* share very similar clinical features. The vitiated Doshas in Pandu Roga cause a disruption in tissue metabolism, which results in *Dhatu Shaithilya*. This article presents the Ayurvedic concept of *pandu roga* with correlation to IDA.

Keywords: Pandu, Anaemia, Iron deficiency anaemia (IDA)

INTRODUCTION

In Ayurvedic texts, *pandu roga* is described as an independent disease and a sign in various other diseases. Ayurveda describes *pandu roga* as a *pitta pradhana* disease associated with *rakta* and *rasa dhatu*. *Pandu roga* is characterised by a change in the colour of the skin to *shwetabha* (pale, whitish), *peetabh*a(yellowish), *harita* (greenish)[1]. The other general signs and symptoms of the disease are vaguely similar to iron deficiency anemia of modern science.

Iron deficiency is the most widespread nutritional disorder in the world. It is estimated that 30-50% of the global population has iron-deficiency anaemia, and most live in developing countries. The prevalence of anaemia among six groups as per the National Family Health Survey 5 (2019-21) is 25.0 percent in men (15-49 years) and 57.0 percent in women (15-49 years).31.1 percent in adolescent girls,52.2 percent in pregnant women (15-49 years) and 67.1 percent in children (6-59 months). According to the National Family Health Survey (NFHS) III data, the incidence of anemia in urban children is 71%, rural is 84%, and overall is 79%[2]

MATERIALS AND METHODS

We conducted a systematic search of all the Ayurvedic scriptures and various articles citing pandu roga, anaemia, iron deficiency anaemia, and an Ayurvedic review on pandu roga. We also searched for other published books and journals with related subjects.

REVIEW OF LITERATURE

The term '*pandu'* means paleness. This disease is a *varnoplakshita vyadhi* (characterized by the change in colour). Acharya Sushruta used the terms Kamala, Panki, apnaki, Laghrak, Alas, and Kumbhahwa as synonyms of *Pandu*[3]. Acharya Harita has used the term *kamala*, *kumbha kamala*, and *Halimaka* as types of *pandu*[4]. Acharya Harita have included two types of *kamala* in as types of *pandu*. However, some Acharyas have mentioned *kamala* and *halimaka* are secondary to *pandu roga*. Hence, these should be included as the types of *pandu*.

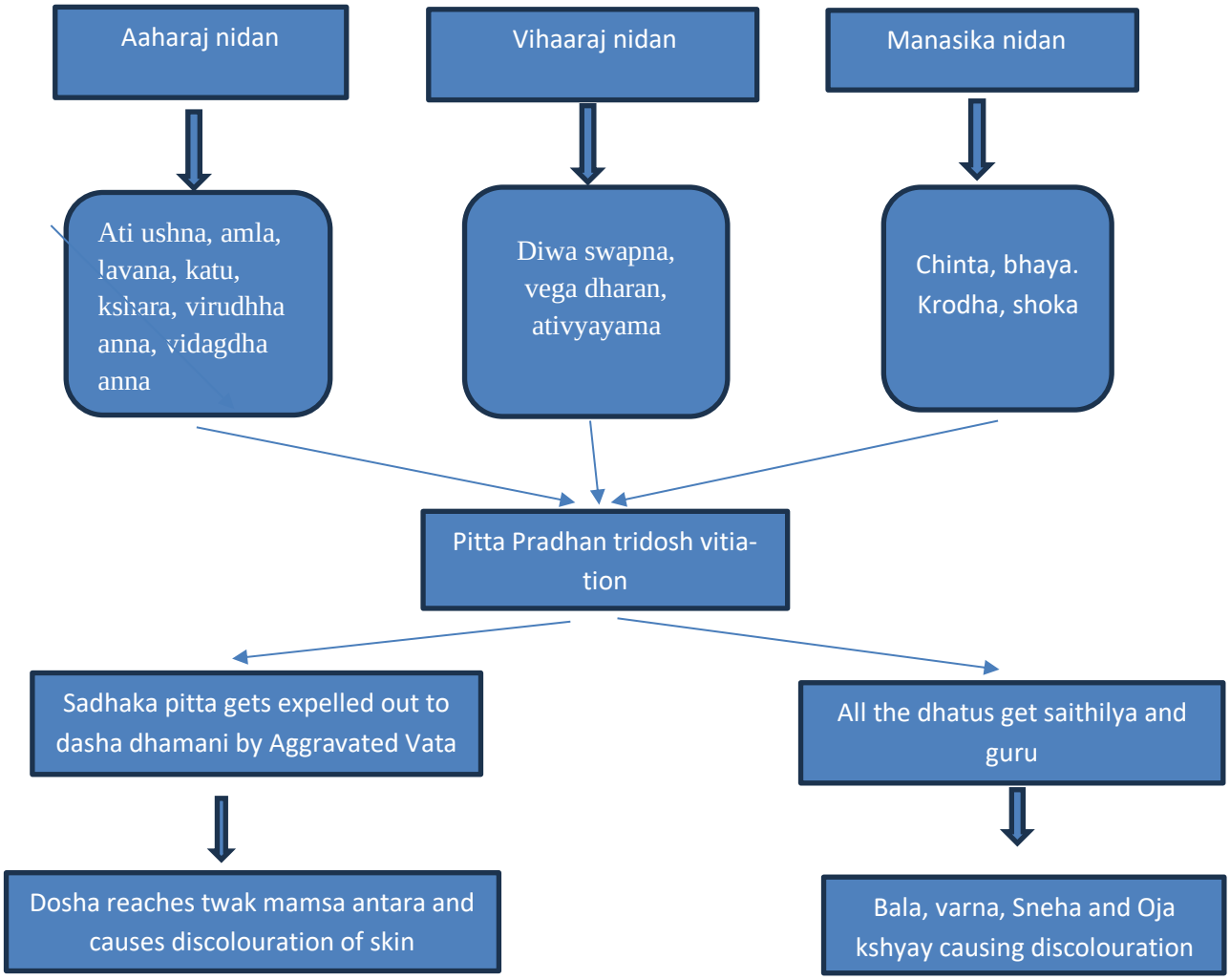
Types of *Pandu*

Types of pandu	Charaka Samhita[5]	Sushruta Samhita[6]	Ashtanga Hridaya[7]	Harita Samhita[8]	Madhava nidana[9]
Vataja pandu	+	+	+	+	+
Pittaja pandu	+	+	+	+	+
Kaphaja pandu	+	+	+	+	+
Sannipataja pandu	+	+	+	+	+
Mritbhakshyana janya pandu	+		+	+	+
Kamala koshthashrita	-			+	
Kamala shakhashrita	-			+	
Halimaka	-			+	

PATHOPHYSIOLOGY ACCORDING TO AYURVEDA

In Ayurvedic literature, *pandu* is described as a disease of *rasavaha strotas* and a *lakshana* in *raktavaha strotas viddha*. *Pandutwa* (pallor) is the cardinal sign of this disease, the change in *the prakrita varna of the* skin. The complexion of skin is due to *bhrajaka pitta*. Foods like ati ushna, amla, katu, lavana, kshara, virudhha anna, vidagdha anna, regimens like diwa swapna, vega dharana, and ativyayama, and psychological factors like chinta, bhaya, krodha, and shoka cause the vitiation of *pitta pradhana tridosha*. *Sadhaka pitta*, situated in the heart, gets aggravated by these nidana. By powerful Vata, it gets expelled to dasha dhamani (vessels attached to the heart) and then circulates all over the body. This aggravated *Pitta* reaches *twakmamsaantara* (the space between

skin and muscle tissue) and causes vitiation in *Kapha, Vata, Asrik, Twak and Mamsa*. This leads to the discolouration of the skin into *pandu*, *haridra*, and *harita*.[10] Acharya Charaka has used '*vaivar-na*' for this phenomenon.

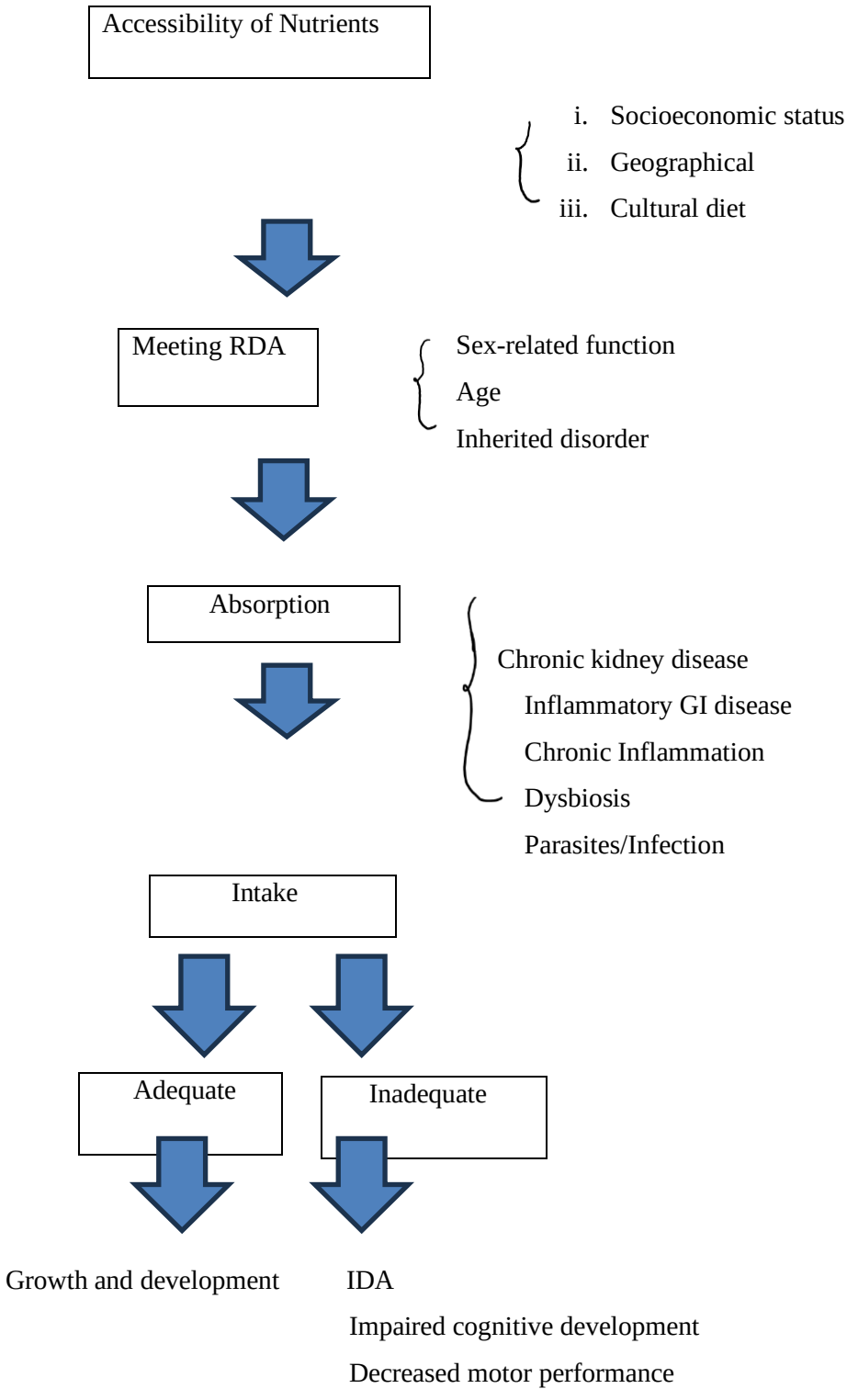


Samprapti Ghataka[11]

Dosha	Pitta Pradhana tridosha
Dushya	Twak, rasa, rakta, mamsa, meda
Strotas	Rasa vaha, rakta vaha
Stroto dushti	Sanga and vimarga gamana
Agni	Jatharagni, dhatwagni
Agni dushti	Mandagni
Udbhava sthana	Amashaya
Adhistana	Twak mamsa abhyantara
Vyakti sthana	Twak
Swabhava	Chirakari
Sadhyasadhyata	Sadhya in early stage, Asadhya in later stage

PATHOGENESIS OF IDA ACC.TO MODERN[12]

FACTORS



The mechanism behind the pathogenesis of iron deficiency anemia (IDA) is influenced by both the availability of nutrients and their absorption. Socioeconomic and cultural factors shape the quantity and quality of the diet. Dietary choices and other environmental influences affect both the availability and absorption of nutrients, leading to an increased recommended dietary allowance (RDA) and a heightened risk of insufficient intake. Inflammation in the gastrointestinal (GI) tract and an imbalance in gut microbiota caused by metabolic and genetic disorders, parasites, and infections result in inadequate iron and other essential nutrient intake, contributing to IDA. The mild to moderate symptoms associated with IDA include fatigue, weakness, koilonychia, pica, and paleness, ultimately causing significant impairments in growth, motor skills, and cognitive functioning.

METABOLISM AND ABSORPTION13

Iron is essential to virtually all living cells, specifically human cells. Non heam iron is released as Fe^{3+} state, Fe3+ converted in to Fe2+(ferrous) in the stomach by gastric acid, divalent metal transporter 1 (DMT 1) will take iron (Fe2+) from the lumen of intestine, from the cell ferroprotein again take it from cell to the blood, ultimately iron again reach in the blood from (Fe3+), but ferroportin again oxidizes the iron (from Fe2+ to Fe3+), now in the blood iron can't move alone it requires transporters (transporters of iron is knows as transferrin) transferrin 2-2 iron molecule in set and carry iron in the everywhere of the body thought of the body, iron is required in the bone marrow so it will be the carry to the bone marrow via transferrin, it will be taken by the Normoblast present inside the bone marrow, they will take it and they will synthesis hemoglobin out of the iron. After the extra iron go to the liver and the extra iron will be stored in the liver in the form of ferritin (is the storage of backup from iron), sometimes we eat too much iron in the diet, all iron will go and maximum iron will be accumulated in the liver in the form of ferritine , but liver do not required ferritin. Once the excess of iron in the liver, liver synthesis the protein k/s Hepcidin (that protein synthesis by the liver), they will go to the via blood that will go to the ferroprotein and inhibit and degrade the ferroprotein.

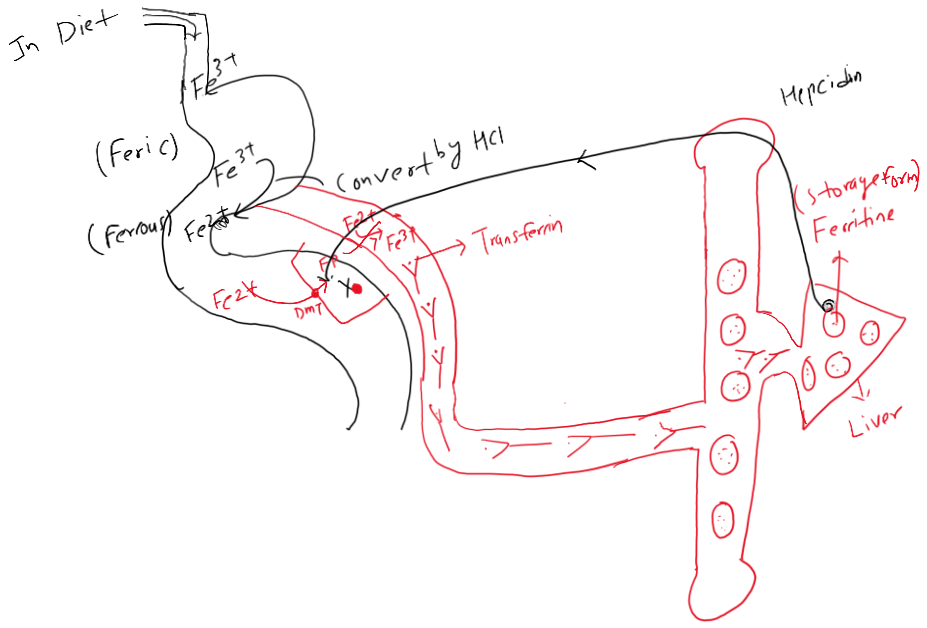
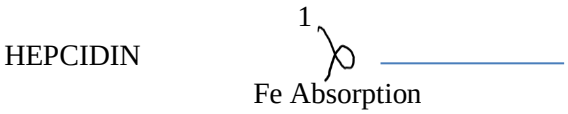


Figure: Iron on absorption from the upper small intestine circulates in plasma and is bound to transferrin. It is then transported to the bone marrow for utilisation in hemoglobin synthesis.



1. High hepcidin levels inhibit iron absorption into the blood when the body is replete with iron.
2. When body iron stores is low – hecidine synthesis fall – facilitates iron absorption into the blood.

Factors increasing iron absorption	Factors decreasing iron absorption
Ferrous form (Fe2+)	Ferric form (Fe+)
Acid (HCL) in the stomach	Achlorrydria(Absence of HCL secretion)
Ascorbic acid	Alkaline food (pancreatic secretion)
Amino acid and sugars in the food	Phytates, tannates and phosphates in diet
Iron deficiency	Iron overload
Physiological condition (pregnancy and hypoxia)	Tetracyclines and EDTA
	Inflammatory disorders

(1) ABSORPTION OF HAEM IRON

Heme iron, which originates from the breakdown of hemoglobin and myoglobin found in meat and fish, has a higher rate of absorption[14]. The term bioavailability of iron refers to the portion that the body can absorb and utilise for its physiological functions; this rate of absorption can vary based on multiple factors, including bio accessibility, which is the amount that can be liberated from the food matrix during digestion and enter the soluble fraction, making it available for uptake by the body through the epithelial cells of the gastrointestinal lining[15]. The initial step in rendering a nutrient bioavailable is to release it from the food matrix and convert it into a chemical form that can interact with and permeate the spaces between intestinal cells. Chewing and enzymatic digestion of food enhance the bio accessibility of nutrients. The primary site for nutrient absorption is the small intestine. While the terms bioavailability and bio accessibility are frequently exchanged, it's crucial to recognize that bioavailability encompasses bioaccessibility[16].

(2) TRANSPORT AND DISTRIBUTION OF IRON IN THE BODY

Within the body, iron is carried between sites of absorption and utilization through the plasma glycoprotein known as transferrin (Tf) and is stored as well[17]. The specific Tf receptors (TfR) found on cell membranes are essential for cells to acquire iron as they recognize transferrin in the plasma. These receptors attach to the iron-transferrin complex on the cell surface and facilitate its internalization, allowing iron to be released. The Fe3+ liberated from the Tf-TfR complex undergoes reduction within the endosomes. This step is crucial as it enhances iron uptake by red blood cell precursors, while excess iron is directed to functional compartments or stored as ferritin[18].

LABORATORY EVALUATION OF IRON STATUS: -

Hemoglobin Concentration (Hb):- Hemoglobin and hematocrit (Hct) are similarly valuable tests and are evaluated similarly. Typically, hematocrit levels are approximately three times the concentration of hemoglobin. A decrease in Hb or Hct indicates anemia but does not provide insight into the underlying cause. The hemoglobin concentration alone cannot differentiate between iron deficiency anemia and anemia resulting from other factors[19].

RBC:-

Microcytic: -Micro-Small, Cystic-Size

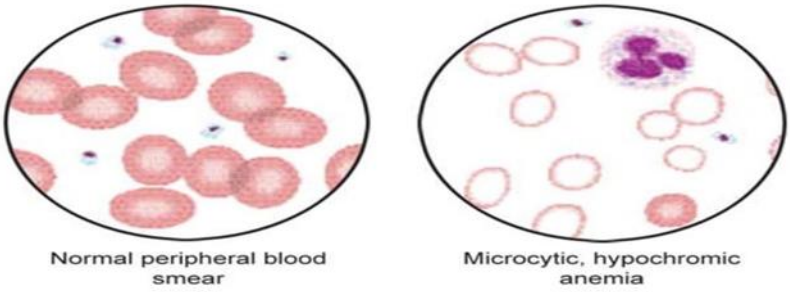
Microcytic describes red blood cell (RBC) size smaller than the normal range.

Hypochromic:-Hypo-Less, chromic-Color

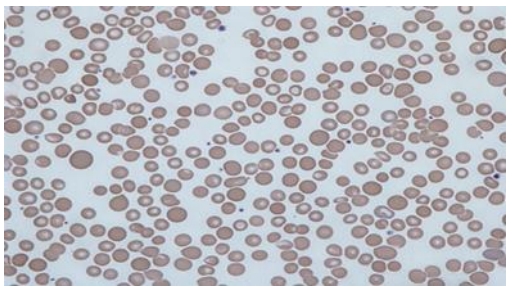
Hypochromic means that the red blood cells have less colour than normal when examined under a microscope.

In iron deficiency, Microcytic hypochromic anaemia is caused by disruption of the iron supply in the diet due to decreased iron content, pathology of the small intestines like sprue and chronic diarrhea, gastrectomy, and deficiency of vitamin C in the diet. It may be due to acute or chronic blood loss and also suddenly increased demands of pregnancy or major surgery.

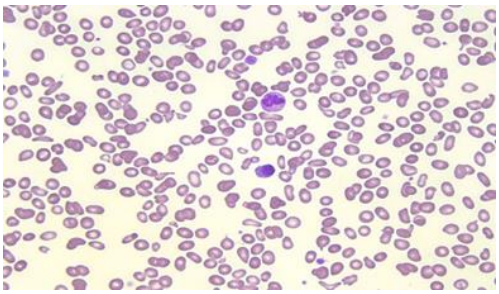
Reduced hemoglobin in the RBCs decreases the amount of oxygen delivered to the peripheral tissues, leading to tissue hypoxia[20]



Anisocytosis: Anisocytosis is the medical term for having red blood cells (RBCs) that are unequal in size. Iron deficiency anaemia occurs when the body doesn't have enough iron, either due to blood loss or a dietary deficiency. It usually results in microcytic anisocytosis[21]



Poikilocytosis: In poikilocytosis, RBCs are irregularly shaped and may not carry enough oxygen. Not all RBCs will take on an abnormal shape. Patients with poikilocytosis have some normally shaped cells RBCs mixed with abnormally shaped poikilocytes. Sometimes, many different types of poikilocytes are present in one patient's blood smear in conditions like Iron deficiency anaemia, megaloblastic anaemia, etc. 22

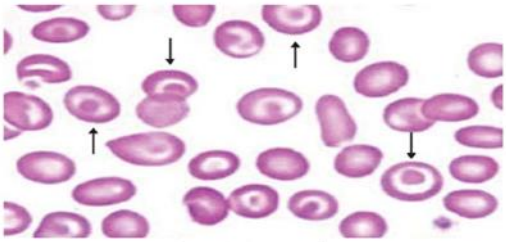


Target cell:-Target cell adopt a 'Bullseye" Morphology where hemoglobin is concentrated in the center part and periphery with a colourless zone between the areas.

The target cell membrane is thinner than the normal cell membrane.

Target cells have an increased surface area to volume ratio and decreased osmotic fragility.

This may result from a decrease in hemoglobin, as in iron deficiency anaemia, or an increase in the cell membrane[23]



RETICULOCYTE COUNT: Reticulocyte hemoglobin content decrease is an early sensitive indicator of iron deficiency erythropoiesis. It is also reliable in the assessment of iron therapy response[24]

RED CELL INDICES: -

MCV: In children, the mean corpuscular volume (MCV) is lower than that of adults, and for those aged 2 to 10 years, the minimum MCV is around 70 fl. The maximum MCV can be calculated by adding 0.6 fl for each year beyond the first year of life, starting from 84 fl, until it reaches the adult upper limit of 96 fl[25]

The MCV (mean corpuscular volume) is elevated at birth and declines quickly during the first six months of life. A reduced MCV may indicate low iron levels, iron deficiency, thalassemia, infections, and chronic diseases. A significantly low MCV and a normal hemoglobin level may point to thalassemia minor. An increased MCV is seen in cases of megaloblastic anemia and liver disorders 26.

MENTZER INDEX- The ratio of MCV to RBC count in million, Value <13% is in thalassemia minor in 85%, while >13% in 85% chances in iron deficiency. In contrast, MCH and MCHC do not add much significant clinical information[27].

MCH: Mch defines the amount of hemoglobin per red blood cell28.The normal values for Mch are 27 to 31 picograms(pg) per cell. MCH is below normal in hypochromic anaemia, often due to a low iron level.

MCHC: MCHC defines the amount of hemoglobin per unit volume. In contrast to MCH, MCHC correlates the hemoglobin content with the cell's volume. It is expressed as gm/dl of red blood cells or as a percentage value[29]. The normal values for MCHC are 32 to 36 gm/dl.

RDW: The RBC distribution width (RDW) quantifies the variability in erythrocyte size distribution and is calculated as the coefficient of variation of erythrocyte volume distribution expressed as a percentage. In simpler terms, it indicates the level of anisocytosis in the blood.[30] An elevated red cell distribution width (RDW) of more than 14.5% strongly suggests iron deficiency. In thalassemia trait and anaemia of chronic disease, the RDW is normal.[31]

Serum ferritin: The serum ferritin level is a sensitive indicator of iron status. It is estimated that each ng/ml of ferritin corresponds to approximately 8-10 mg of stored iron. A serum ferritin level of less than 12 ng/ml is highly specific for iron deficiency, though it does not provide insights into the severity of the deficiency. Another significant drawback of serum ferritin is that its concentration can rise in chronic conditions, such as chronic infections, which may lead to the oversight of concurrent iron deficiency anemia.[32]

Serum Iron: The typical serum iron level can fluctuate significantly due to its diurnal pattern, often reaching its highest point in the morning and dropping in the evening. Chronic infections, cancer, and chemotherapy can also influence serum iron concentrations. A serum iron level below 40 micrograms/dl (below 12 mcg/dl in young children) indicates iron

deficiency that is not complicated by infections or other conditions impacting iron metabolism.[32]

TIBC (Total Iron Binding Capacity): TIBC indicates the amount of transferrin in the bloodstream. Typically, there is sufficient transferrin in 100 ml of serum to bind approximately 250-450 micrograms of iron. Since the normal serum concentration is 100 micrograms/dl, transferrin is usually about one-third saturated with iron. In cases of iron deficiency, TIBC levels rise, and transferrin saturation drops below 16% (less than 14% for children). A TIBC of less than 200 micrograms/dl is commonly associated with inflammatory diseases.[32]

Transferrin Saturation[32]: -Transferrin saturation = Serum iron/TIBC ×100

TREATMENT

The treatment modalities according to Ayurveda depend upon the involvement of *doshas* and the *Bala* of the disease and patient. Pandu roga's treatment principle is *Shodhana Chikitsa* (purification procedure). The patient should be given proper Snehan therapy, followed by appropriate *Tikshna Sodhana, i.e Vamana and Virechana,* to eliminate the vitiated doshas from the body. For *Vataj pandu*, medicine should be *Sneha Pradhan*; for Pittaja pandu, tikta rasa and sheeta virya drugs should be used. For *kaphaja pandu*, drugs of *katu, tikta rasa* and *ushna virya* should be used. For *tridoshaja pandu*, a mixed therapy should be used. For *Mritbhakshyana janya pandu, Shodhana* should be done, followed by medicines to promote *agni* and *bala.* The treatment in Ayurveda is multidisciplinary. The physician should prescribe the medicines according to their *yukti*, based on the predominance of *dosa* and *bala* of *roga* and *rogi*[33]

The key principles in managing iron deficiency anaemia involve establishing the diagnosis, exploring the underlying causes of the iron deficiency and addressing those causes, replenishing the iron levels, enhancing nutritional intake, and educating patients and their families. Iron deficiency is typically treated with oral iron supplements, commonly available ferrous sulfate, which is affordable and generally well absorbed. Dosing is based on elemental iron: children typically receive 3 to 6 mg/kg daily, whereas adolescents are given 60 mg per dose[34]. The reaction to iron treatment is usually quick when the iron deficiency is due to nutritional reasons 35. The approach to treating anaemia is determined by its severity and any associated complications. Patients with a hemoglobin level below 5 g/dl may need to be hospitalized, as some might already be experiencing congestive heart failure. Blood transfusions are only indicated in the most critical situations where hemoglobin falls below 3 g/dl. Young children might require transfusions if their hemoglobin levels drop below 4-5 g/dl due to a greater risk of congestive heart failure. Rapidly correcting anaemia through transfusions can be risky because it may lead to hypervolemia and cardiac dilation. It is recommended that packed or sedimented red blood cells be administered slowly, ideally 2-3 ml/kg at a time[36].

1. **MEDICINAL IRON THERAPY**

For infants and children, the advised therapeutic dosage is 3 mg of iron for each kilogram of body weight per day. For women aged 15 years and older who have severe anemia (Hb < 7 gm/dl), the National Nutritional Anemia Control Program (NNACP) suggests taking three iron-folate tablets each day (with each tablet consisting of 100 mg of elemental iron and a specific amount of folic acid) for at least 100 days. While the targeted hemoglobin level is typically achieved within two months, iron treatment should continue for an additional two months to replenish iron stores to 250-300 mg or to elevate the serum ferritin level to 30 micrograms/liter.[36]

Supplementation of iron folic acid: The Intensified National Iron Plus Initiative			
Age	**Frequency of administration of iron + folic acid**	**Preparation**	**Albendazole (tablet: 400 mg), twice a year**
6 mo. To 5yr	Twice a week (fixed days)	1 ml IFA =20 mg elemental iron +100 micro gm folic acid	1-2 years:1/2 tablet >2 years:1 tablet
5-10 yr	Once a week	45 mg elemental iron +400 micro gm folic acid	
10-19 yr	Once a week	100 mg elemental iron +500 micro gm folic acid	

2. PARENTERAL IRON THERAPY

The parenteral route is generally best avoided and is reserved for situations where there are severe side effects from oral therapy, issues with compliance, or gastrointestinal bleeding that worsens with oral iron treatment. A complete iron requirement can be delivered in one infusion, known as total dose infusion. However, this infusion should only be administered in a hospital setting due to the risk of anaphylactic reactions. Iron dextran complex is the most widely used formulation. The necessary level of hemoglobin can be accurately computed. Considering that 50% or more of the iron is needed to replenish body stores, the iron requirement can be calculated using the following equation: [37]

Iron(mg) = Weight (kg) × Hb deficit (g/dl) × 80 / Weight (kg) ×Hb deficit (g/dl) × 4

PREVENTION OF IRON DEFICIENCY

The American Academy of Pediatrics, the World Health Organization, and other prominent pediatric organizations have suggested numerous guidelines for preventing iron, the most prevalent nutritional deficiency globally. These guidelines encompass fortifying foods with iron, providing iron-rich formulas when breast milk is inadequate, avoiding cow's milk during the first year of life, screening infants for iron deficiency between 9 to 12 months, and administering iron prophylaxis to infants 38.

DISCUSSION

Pandu roga is a common disease which is seen in growing children because they always face a GIT problem. In these conditions, children cannot achieve the original nutrients and ions from the food. In Charaka Samhita, Sushruta Samhita, Ashtanga hridaya and other classics are given many verities of treatment for Panduroga, which is very effective in the management of pandu roga.

CONCLUSION

A detailed history and clinical examination (panduta, Aruchi, Daurbalya, Gatra Shula, Pindikodveshtana, Sadana, Bhrama, Hridspanadana) are the most common steps to make the diagnosis. During an investigation, one should try to interpret simple investigations like complete blood count to check haemoglobin levels and if the Hb level is below the normal level along with MCV. MCH and MCHC below the normal level so we calculate iron deficiency anaemia according to Mentzer index if Mentzer index >13 suggest that the patients have iron deficiency anaemia and Peripheral blood smear Show Microcytoic Hypochromic Anaemia, well these two tests are enough but if we need to confirm the exact result so we can go for iron profile. During the management

of iron deficiency anaemia, the cause should be managed first along with the replacement of the deficiency of iron or vitamins.

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