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A CRITICAL REVIEW ON ANAEMIA

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ABSTRACT

Anaemia is a condition that develops when your blood lacks enough healthy red blood cells or hemoglobin. Hemoglobin is a main part of red blood cells and binds oxygen which refers to a decreased number of circulating red blood cells and is the most common blood disorder. Symptoms can include headaches, chest pains, and paleness of skin. Anemia currently affects more than 3 million Americans and an estimated 1.62 billion people, globally. There are three main types of anemia: that due to blood loss, that due to decreased red blood cell production and that due to increased red blood cell breakdown. Causes of blood loss include trauma and gastrointestinal bleeding, among others. Causes of decreased production include iron deficiency, a lack of vitamin B_{12}, thalassemia, and a number of neoplasms of the bone marrow. Causes of increased breakdown include a number of genetic conditions such as sickle cell anemia, infections like malaria, and certain autoimmune diseases. It can also be classified based on the size of red blood cells and amount of hemoglobin in each cell. If the cells are small, it is Microcytic Anemia. If they are large, it is Macrocytic Anemia while if they are normal sized, it is Normocytic Anemia. Diagnosis in men is based onlevel of hemoglobin, if less than 130 g/L (13 g/dL), while in women; it must be less than 115 g/L (11.5 g/dL). Further testing is then required to determine the cause. Anemia is not strictly a disease, but a disorder. It is often a by-product of other diseases that either interfere with the body's ability to produce healthy red blood cells or abnormally increase red blood cell breakdown or loss.

Key words: Anemia, PCV, MCV, MCHC, CRPA, PNH, AIHA

INTRODUCTION

Anaemia is defined as reduced haemoglobin concentration in blood below the lower limit of the normal range for the age and sex of the individual. In adults, the lower extreme of the normal haemoglobin is taken as 13.0 g/dl for males, 11.5 g/dl for females and 15.0 g/dl for infants. Although haemoglobin value is employed as the major parameters for determining whether or not Anaemia is present, the Red cells count, haematocrit (PCV), absolute values (MCV- Mean corpuscular volume)and (MCH-mean cell haemoglobin or mean corpuscular haemoglobin) and (MCHC-Mean corpuscular Haemoglobin concentration) provide alternate means of assessing Anaemia.

Symptoms:-

1) Tiredness
2) Easy fatigability
3) Generalised muscular weakness
4) Lethargy and headache
5) Cardiac failure, angina.

Signs:-

1) **Pallor:-**Pallor is the most common and characteristic sign which may be seen in the mucous membrane, conjunctiva and skin.
2) **Cardiovascular system:-**Tachycardia, collapsing pulse, cardiomegaly, dyspnoea and congestive heart failure.
3) **Central nervous system:-**Attack of faintness, giddiness, headache, tinnitus, drowsiness, numbness and tingling sensation of the hands and feet.
4) **Gastrointestinal system:-**Anorexia, nausea, constipation and weight loss may occur.
5) **Reproductive system:-**Menstrual disturbance such as amenorrhea, menorrhagia and loss of libido.
6) **Renal system:-**Mild proteinuria and impaired concentrating capacity of the kidney may occur in severe anaemia.
7) **Ocular manifestation:-**Retinal haemorrhage may occur if there is associated vascular disease.

Classification of Anaemias:-

(A) Pathophysiologic:-

❖ **Anaemia due to increased blood loss:-**

1. Acute post haemorrhagic Anaemia
2. Chronic blood loss

❖ **Anaemia due to impaired red cell production:-**

1. Cytoplasmic maturation defect-

a) Deficient haem synthesis which known as Iron deficiency anaemia
b) Deficient globin synthesis which known as Thalassemia syndrome

2. Nuclear maturation defect-

Vitamin B_{12} and folic acid deficiency which known asMegaloblastic anaemia

3. Defect in stem cell proliferation and differentiation-

a) Aplastic anaemia
b) Pure red cell aplasia

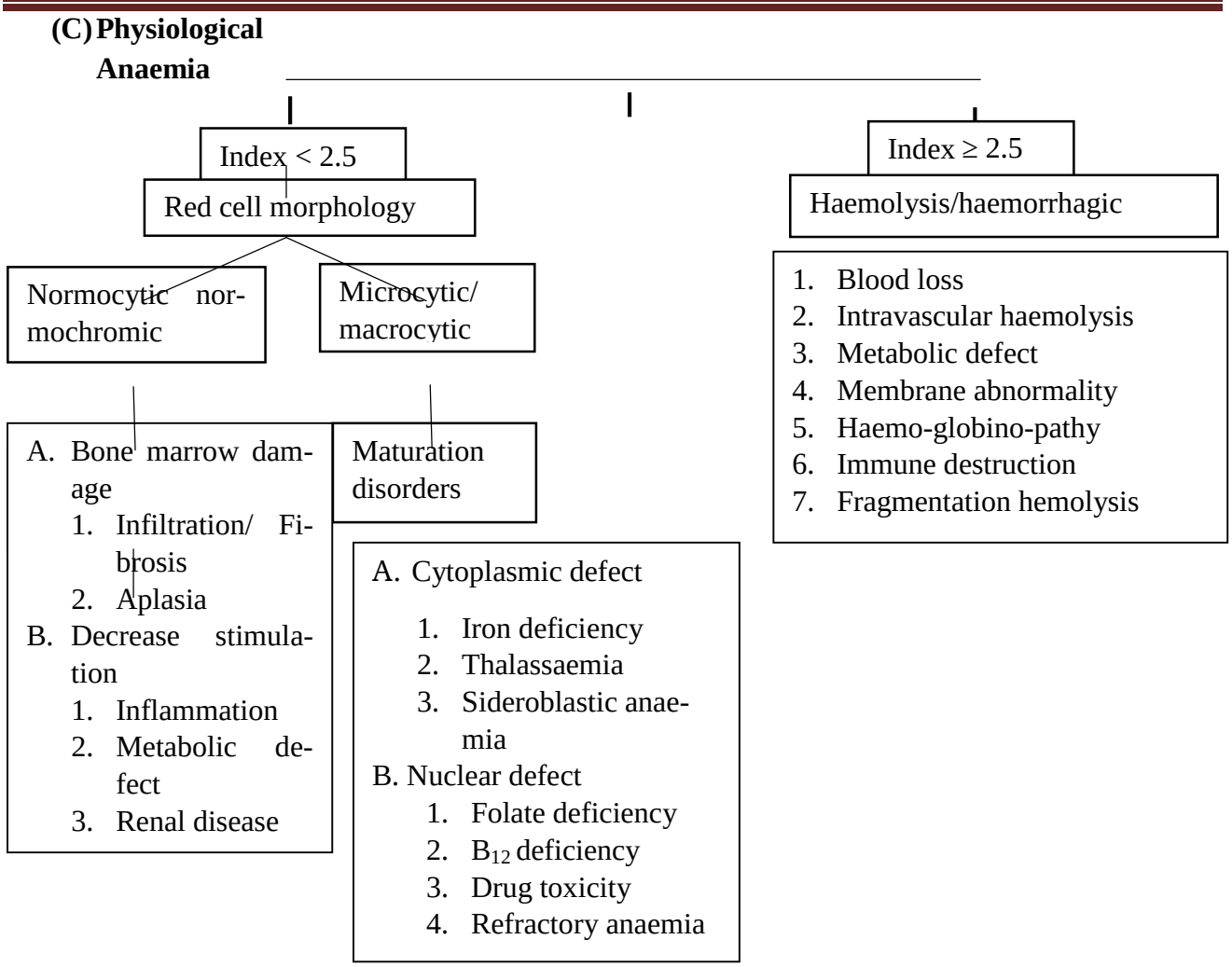
4. Anaemia of chronic disorder
5. Bone marrow infiltration
6. Congenital anaemia

❖ **Anaemia due to increased red cell destruction (Haemolytic anaemia)**

1. Extrinsic (extra corpuscular) red cell abnormalities
2. Intrinsic (intra corpuscular) red cell abnormalities

(B) Morphological:-

1. Normocytic normochromic anaemia
2. Normocytic hypochromic anaemia
3. Macrocytic anaemia
4. Microcytic hypochromic anaemia[1]



Initial classification of anaemia:-

The functional classification of anaemia has three major categories.

1. Bone marrow production defect (hypo-proliferative)
2. Red cell maturation defect (ineffective erythropoiesis)
3. Decreased red cell survival (haemolysis)[2]

Acute haemorrhage:-

Acute haemorrhage refers to sudden loss of a large quantity of blood as in the case of accident. The plasma protein of blood is replaced within 24 hours after the haemor-rhage. However, the replacement of RBC's doesn't occur quickly and it taken at least 4 to 6 weeks.

Decreased RBC's count causes hy-poxia, which stimulate the bone marrow to produce more number of RBC's.

Chronic haemorrhage:-

It refers to internally or externally blood loss, over a long period of time. Conditions like-

1. Peptic ulcer
2. Purpura
3. Haemophilia
4. Menorrhagia

Iron deficiency anaemia:-

Iron deficiency anaemia is the most common type of anaemia. It develops due to inadequate availability of iron for haemoglo-bin synthesis. RBC's are **Microcytic.**

Causes:-

a) Loss of blood
b) Decreased intake of iron

a) Increased demand for iron from intestine
b) Increased demand for iron in condition like growth and pregnancy

Signs:-

a) Brittle nails
b) Spoon-shaped nails
c) Brittle hair
a) Topley of papilla in tongue
b) Dysphagia (difficulty in swallowing)[3]

Thalassemia:-

The thalassaemia's are a diverse group of hereditary disorder in which there is reduced synthesis of one are more of the globin polypeptide chain, the word "Thalassa" in Greek means "The Sea" since the condition was found commonly in region surrounding the Mediterranean Sea. It is also known as Mediterranean anaemia which is more common in Thailand[4].

Types-

1. α- Thalassaemia- α- chain is absent or abnormal
2. β- Thalassaemia- β- chain is absent or abnormal[5]

Megaloblastic anaemia:-

The megaloblastic anaemia are disorders caused by impaired DNA synthesis and are characterised by a distinctive abnormality in the haematopoietic precursors in the bone marrow which the maturation of the nucleus is delayed relative to that of the cytoplasm. Since cell division is low but cytoplasmic development progressive normally, the nucleated red cell precursors tend to be layer which "Ehlich" in 1880 termed megaloblasts. Megaloblasts are both morphologically and functionally abnormal with the result that the mature red cell formed from then and released into the peripheral blood are also abnormal in shape and size, the most prominent abnormality being macrocytosis.

The underlying defect for the asynchronous maturation of the nucleus is defective DNA synthesis due to deficiency of vitamin B-12 and / or folic acid[6].

Aplastic anaemia:-

Aplastic anaemia defined as pancytopenia (i.e. simultaneous presence of anaemia and thrombocytopenia) resulting from aplasia of bone marrow.

Aplastic anaemia is due to the disorder of red-bone marrow. Red bone marrow in reduced and replaced by fatty tissues. Bone marrow disorderoccur in the fallowing condition:-

1. Repeated exposure to X-ray or gamma ray radiation.
2. Presence of bacterial toxin, quinine, and radium etc.
3. Tuberculosis
4. Viral infection like hepatitis and HIV infection.

Clinical features:-

1. Anaemia and its symptoms like mild progressive weakness and fatigue.
2. Haemorrhage from various sites due to thrombocytopenia such as from the skin, nose, gums, vagina and bowel and occasionally in CNS and retina.
3. Infection of the mouth and throat are commonly present.
4. The lymph nodes, liver and spleen are generally not enlarged[7].

Pure red cell Aplasia:-

Pure red cell aplasia (PRCA) is a rare syndrome involving a selective failure in the production of erythroid-elements in the bone marrow with normal grenulo-poasis and megakaryo-cyto-poasis.

PRCA exists in the following forms-

1. Transient self – limited PRCA – it is due to temporary marrow failure in aplastic-oasis in haemolytic anaemia.
2. Acquired PRCA –
3. It is seen in middle aged adults in association with some other diseases, most com-

monly are rheumatoid arthritis, lymphoid malignancies and solid tumour.

4. Chronic B-19 parvovirus infection- PRCA may from chronic B19 parvovirus infection in children and in common.
5. Congenital PRCA (Blackman- diamond syndrome) – it occurs due to mutation in a ribosomal RNA processing gene termed as RPS19[8].

Anaemia of chronic diseases:-

Anaemia of chronic disease is the second common type of anaemia (next to iron deficiency anaemia). It is characterised by short life span of RBC's, caused by disturbance in iron metabolism or resistance to erythropoietin action. Anaemia develops after few months of sustained disease.

Common causes:-

1. Non-infection inflammatory diseases such as rheumatoid arthritis.
2. Chronic infection like tuberculosis and abscess in lump.
3. Chronic renal failure, in which the erythropoietin secretion decreases.
4. Neoplasia disorders such as Hodgkin's disease (malignancy involving lymphocytes) and cancer of lung and breast[9].

Bone marrow infiltrations:-

Disease that infiltrates bone marrow may cause anaemia by disrupting erythropoiesis in multiple myeloma (MM). However, most patients have relatively uniform marrow infiltration by myeloma. Myelofibrosis, chronic myeloid leukaemia, and lymphoid leukaemia may uniformly infiltrate marrow, but splenomegaly disorders from extramedullary haematopoiesis may confirm assessment of erythropoiesis[10].

Classification of haemolytic anaemia:-

I. Acquired:-

1. Antibody:-

a) Autoimmune haemolytic anaemia (AIHA):-

a. Warm antibody of AIHA
b. Cold antibody of AIHA

b) Drug-induced immune-haemolytic anaemia

c) Isoimmunise haemolytic anaemia

2. Mechanical trauma:-Micro-angiopathic haemolytic anaemia
3. Direct toxic effects:-Malaria, ,infections and other agents
4. Acquired red cell membrane abnormalities:-Proximal nocturnal haemoglobinuria(PNH)

II. Hereditary:-

1. Abnormalities of red cell membrane:-

a) Hereditary spherocytosis

b) Hereditary ellipto-cytosis

c) Hereditary stomato-cytosis

2. Disorders of red cell interior:-

a) Red cell enzyme defects (Enzymopathies):-

i. Defect in the hexose monophosphate shunt
ii. Defects in the glycolytic pathway:- Pyruvate kinase deficiency

b) Disorder of haemoglobin (haemoglobinpathies):-

i. Structurally abnormal haemoglobins:- **Sickle syndrome**
ii. Reduced globin chain synthesis:- **Thalassaemia**

Haemolytic anaemia:-

Haemolytic anaemia is defined as anaemia resulting from an increase in the rate of cell destruction. Normally, red cells undergo lysis at the end of their lifespan of 120± 30 days within the cells of reticuloendothelial (RE) system in the spleen and elsewhere (extra vascular haemolysis), and haemoglobin is not liberated into the plasma in appreciable amounts. The red cell lifespan in shortened in haemolytic anaemia i.e. there is accelerated haemolysis.

The premature destruction of red cells in haemolytic anaemia may occur at either of the following 2 sites:-

1. Firstly, the red cells undergo lysis in the circulation and release their contents into plasma (intravascular haemolysis),
2. The red cells are taken up by cells of the RE system where they are destroyed and digested (extra vascular haemolysis) [11].

Autoimmune haemolytic anaemia:-

"WARM" antibody **AIHA**

"WARM" antibodies reactive at body temperature and coating the red cells are generally IgG clam antibodies and occasionally they are IgA.

Clinical features:-

1. Splenomegaly
2. Occasionally hyperbilirubinemia

"COLD" antibody **AIHA**

Antibodies which are reactive in the cold (4^0c) may induce haemolysis[12].

Sickle cell anaemia:-

Sickle-cell-anaemia is an inherited blood disorder, characterised by sickle-shaped red blood cells. It is also called haemoglobin SS disease or sickle cell disease.

Sickle cell anaemia is due to the abnormal haemoglobin called haemoglobin S (Sickle cell haemoglobin). In which α-chain are normal and β-chain are abnormal. The molecules of haemoglobin S polymerised into long chain and precipitate inside the cells. Because of this, RBC's attain sickle (crescent) shape and become more fragile leading to haemolysis. Sickle cell anaemia occurs when a person inherits two abnormal genes (one from each parent). In children, haemolysed sickle cells aggregate and block the blood vessels, leading to infection[13].

The most important and widely prevalent type of haemoglobin-pathy is due to the presence of sickle haemoglobin (HbS) in the red blood cells. The red cells with HbS develop "sickling" when they are exposed low oxygen tension. Patients with HbS are relatively protected against falciparum malaria.

Sickle syndromeoccur in 3 different forms:-

1. A heterozygous state for HbS: sickle cell trial (AS)
2. As homozygous state for HbS:- sickle cell anaemia (SS)
3. A double heterozygous state e.g., sickle β-thalassaemia, sickle – C disease (SC), sickle – D disease (SD)

Clinical features:-

1. Micro-infarcts affecting particularly the abdomen, chest, back and joint.

2. Micro-infarcts involving most commonly the spleen, bone marrow, bones, lump, kidney, CNS, retina and skin result in anatomic and functional damage of these organ[14].

Pernicious anaemia or Addison's anaemia:-

Pernicious anaemia is the anaemia due to deficiency of vitamin $B_{12.}$ It is due to atrophy of the gastric mucosa because of autoimmune destruction of partial cells, results todecreased production of intrinsic factor and absorption of vitamin $B_{12,}$ which is the maturation factor for RBC's[15].

Clinical features:-

1. Lemon yellow colour of skin
2. Red sore tongue
3. Neurological disorder[16]

Laboratory diagnosis:-[17]

1. CBC (complete blood count):-

Sr. no	Test	Test details	Normal value
1	RBC	Total RBC	M-4.3-5.7 million cells/mcl F-3.9-5.0 million cells/mcl
2	MCH	Mean corpuscular haemoglobin	27-31 picro-gram/cells
3	MCHC	Mean corpuscular haemoglobin concentration	32-36 grams/deciliter
4	MCV	Mean corpuscular volume	80-100 femto-liter
5	Hb	Haemoglobin	M-13.5-17.5 g/dl F- 12.0-14.5 g/dl
6	PCV	Haematocrit	M- 38.8-50 % F- 34.9- 44.5 %

❖ **In iron deficiency anaemia:-**

1. Hb estimation:-< 10 mg/dl
2. Erythrocyte count: it varies with severity of anaemia
3. MCH=15-16 Pg
4. MCV=16-18 gl
5. MCHC= 20-30 %
6. RDW= More than 17.5

❖ **In Haemolytic anaemia:-**

1. Reduction in Hb%α RBC count
2. PCV α RBC count
3. RDW = increased
4. Reticulocytes= increased usually -5-20% (0.2-2%)

❖ **In Aplastic anaemia:-**

1. Hb% -<3g/dl
2. Erythrocyte count and PCV: Reduced
3. MCV: >94fl
4. TLC: <200 ccb/cumm
5. Thrombocytes: <10,000/ cumm
6. ESR: Increased
7. BT: Prolonged

❖ **In Megaloblastic anaemia:-**

1. Hb%: Decreased
2. Erythrocyte count and PCV: Reduced
3. MCV: >94fl
4. MCH: >32pg
5. MCHC: >32%
6. RDW: Increased

❖ **In Thalassaemia:-**

1. Electrophoretic pattern of Hb is α or β thalassemia
2. HbA :Absent
3. HbA_2 : Absent 2%
4. HbF : 98%
5. Hb: Reduced
6. Haematocrits : Reduced
7. MCH : < 22pg
8. MCV : < 50-70fl[18]

Ayurvedic view

Pandu

The word "*Pandu*" is derived from root "*PADI NASANE*" with suffix "*KU*" and elaboration through "*NI*". (*Shabdakalpadruma* – Part – 3)

The word "*Pandu*" is described as white, yellowish white etc. According to various dictionaries, *Pandu* means a white colour mixed with yellowish tinge (according to AMARKOSA)

According to *Shabdastoma Mahanidhi*, *Pandu* is disease which can be diagnosed by observing the patient and not by interrogating. *Pandu* has been kept under the disease group which is classified and named according to the change colour, therefore '*Nashana*' will be of the *varna* are colour which is further approved by *Acharya Charaka* by the word "*VIVARNA*".*Pandu* is disease in which there is '*Vivarna*' or change in normal colour of Body.

After considering all these descriptions, one may find it difficult to decide about actual colour by '*Pandu varna*' but if we give a due consideration to *samprapti* of *Pandu* by *Acharya Charaka* who has mentioned that in

this disease there is *Kshaya* or Loss of *Varna* or general Complexion. It is a fact that the natural complexion and redness of skin is maintained by proper blood flow through the skin and when there is diminution in quantity and quality of blood, Pallor in the skin follows. So, in this disease, that is *Alpa Rakta* (Lack of Blood), which causes the pale colouration.

Panduroga literally means disease condition marked with pallor or paleness or yellowish white colouration or the body. (Chakr. on Cha. Chi. 16/1) There may be various modification of colour such as yellow, green, *harita*, *haridra* in the disease condition described under the *Panduroga*, but as the disease condition is characterized by pallor as the predominant sign, the disease is termed as *PANDU ROGA*.

Pandu or Pallor of skin is first observed on the most superficial portion of body and that is skin. But pallor should also be examined in other parts of body described by *Acharyas*, as per thepaleness should be also observe in Eyes, Palate, Tongue, Nose, Lips, Palms, Soles, Nails, Faeces and also in urine. (Cha.Chi. 16). These are the important sites to be well examined, complete clinically observations or examine the patients for *Panduroga*.

ROLE OF *PITTA* IN *PANDUTA*: There is a significant role of *Pitta* in *varnauttaptti*, and *Acharay Charaka* has quoted that *Pittadosha* is very imp (substance) in Natural colour of body, and if *pitta* gets vitiated rhe normal color of body and other sites of body turns into Pandu, Harita and Haritadi *varnas*. *Acharaya Sushruta* has described *Panduta* as a *Rupa* in some disease i.e. *Pittaja Pratishayay*, *Pittaja Kasa*, *Pittadushta* Stanya, *PittajaPrameha*, *PittjaArsha* etc. He also observed that *pandutvam* is main observing symptom in these *Rogas*.

***NIDANA PANCHAKA* OF *PANDU ROGA*:**

Nidana Panchaka is the combination of parameters, which are used in the diagnosis of the disease. They are –

1) Nidana
2) Purvarupa
3) Rupa
4) Upashaya-Anupashaya
5) Samprapti

***NIDANA* OF *PANDU ROGA*:**

The general etiology or *Samanya Nidana* of *Pandu roga* is described in *Charak Samhita*, Which all factors mainly related to *Aharaja*, *Viharaja* and *Nidanarthkar roga*.

CAUSES RELATED TO *AHARAJA HETU*: Food or diet plays the crucial role in the normal development and maintenance of the different *Dhatus* of body. *Panduroga* may be caused due to indulgence of food containing more *Amla, Kshara, Lavana, Ushna, Ruksha* More intake of carbohydrates; only milk and clay produce Anaemia. In food derived from garins, Iron after forms a stable complex with phytates and only small amount of such iron can be converted to absorbable form. Protein in dairy products generally have little effect on Iron absorption. Asatmaya Bhojana and *Viruddha Bhojana* may inhibit normal process by producing *Aam* or Anti-substance and lead to disturbance of the digestive are assimilative process. If *Kshara* and *Amal* are used in excess it might be that, they may injure the gastric mucosa first and then mixed with *rasa* may lead to haemolysis, and disturbed metabolism of various tissues following developing of Anaemia, and if person may use very hot substances in his diet daily, it may injure the gastric mucosa and by this may give rise to *Pandu roga* due to improper digestion.

CAUSES RELATED TO *VIHARAJA HETU*: This factors deals with both mental and physical activities of individual. In physical activities *Ratrijagrana* causes *vataprakopa*

and *Diwasvapana* causes *kapha prakopa* due to *Ativyayam*, *Ativyavaya*, *Adhikasharma* caloric output, out balancing of calories in the food takes place, while is the cause of *Panduroga*, *Vegas* are Natural regulators of body functions. Habitual suppression of optimal positive health was always taken into consideration of all factors however minor they may appear. Climatic changes or disturbance or abnormal season upset the normal function of the body. In mental activities, *Chinta Shoka* is main cause of *Panduroga*. As the majority of the persons fall in poor or lower middle income groups, Hence *Chinta* can be considered one of the constant factor in all types of *Pandurogas*.

CAUSES RELATED TO *NIDANARTHAKARA ROGA*: *Ayurvedic* literature has indicated a correlation of various diseases with *Panduroga* either as symptoms or as *Updravas*. So, all these can be causes of *nidanarthkara Rogas* of *Panduroga*. Some of which are *Raktatipravatan, Raktarsha, Raktarbuda, Asrigdara, Arsha* or *Kaphajarsha, Swasha* etc. which directly or indirectly vitiate *Vata, Pitta* and *Kapha* singly or combination. Though *Pitta* plays a predominant role in the manifestation of *Panduroga*, *Vata* and *Kapha* are also involved in the process.

PURVARUPA:

Classical texts have mentioned the following symptoms and the heading of *Purvarupa* of *Panduroga*.

Avipaka, AkshikutaShotha, Aruchi, Alpavahnita, Angasada, Gatrasada, Hridspandaman, Mutra Pitata, Mridbhakshanaechcha, Panduta, Rukshata, Swedabhava, Shrama, Sthivanadhikya, Twakasphutana, Varchapittatvam.

RUPA:

PratiyatmaLing or Cardinal symptom of this disease is *Panduta* or *Pandubhava*, which is invariable feature. Various types of discolouration have mentioned by almost all *Acharyas*. They have also described *Rupa* or Clinical features in different types of *Panduroga* and the symptoms of *doshikaPandu* have also mentioned by all *Achayas*. As per them this disease is not only due to lack of blood but along with it other *Dhatus* and *Dosha* are also involved to certain extent. Therefore, along with *Rakta* other *Dhatu* and *Doshas* also show specific symptoms of their deficiency or in other words the general symptoms described here, got relation with *Dushti* of specific *Dosha* & *Dhatus*.

Classical texts have mentioned the following symptoms and the heading of *Rupa* of *Panduroga*.

Akshikutashotha, Aruchi, Arohaneayasa, Alpawaka, Annadwesha, Balakshaya, Bhrama, Durbalya, Dhatugaurava, Dhatushithilya, Gatramarda, Gaurava, Hatanala, Hatprabhatva, Jwara, Kopana, Karnashweda, Karnashweda, Katiurupadaruka, Medalpata, Nidraluta, Nisharata, Ojagunakshaya, Pindikodweshtanam, Panduta, Raktalpata, Shishiradwesha, Swasha, Shirnalomata, Sadana, Shrama, Shthivanadhikya, Shithilendriya, Sannasakthi.

CLASSIFICATION OF *PANDU ROGA:*

Sushruta classified *Pandu Roga* 4 varieties:-

1. Vataja Pandu Roga
2. Pittaja Pandu Roga
3. Kaphaja Pandu Roga
4. Tridoshaja Pandu Roga

Charaka mentioned one additional variety of *Pandu roga* that is *Mrid Bhakshanjanya Pandu*. Hence, In *Charaka's* classification of *Pandu* five different varieties are seen. The classification given in *Madhava Nidana* is identical to that of *Charaka*. If viewed logically, the classification given by *Charaka* seems to be more rational and acceptable.

1. Vataja Pandu Roga
2. Pittaja Pandu Roga
3. Kaphaja Pandu Roga

4. Sannipataja pandu Roga
5. Mridbhakashajanya pandu Roga

***SAMPRAPATI* OF *PANDU ROGA*:**

Samprapati corresponds in general to the development of disease including the sequences of process or events from inception to the characteristic development of disease or in short, we can say *samprapati* is the *vyadhi vyapara parampara.*

When the *Pitta* located in its normal abode of heart, become expelled by the vitiated *vayu*, it gets entry into the *dashadhamani* and is mobilized throughout the body. It gets localized in between the *Twacha* and *Mamsa* and vitiates the *Kapha, Vata, Asrika, Twacka and Mamsa* subsequently causing a variety of colour in the skin such as *Pandu, Haridra, Harita* etc. This condition is known as *Panduroga*. (Cha.Chi.16/8-11).

Sushruta has mentioned that *Pandu bhava* caused by vitiation of *twaka* through the vitiated *rakta* in one who indulgence in *ahita ahara vihara*. (Su.Utt.44/7).

Vagbhatta has mentioned the *samprapati* given by *Charaka*. The pathology of *Panduroga* is mainly concerned with vitiation of *pitta* which in turn *vitiates* the *Rakta*, leading to condition of *pandubhava*.Thus, *Pitta* being the *Pradhan dosha* or main factor in the causation of *panduroga*. All the fivefold functions of it are affected more or less, but as the main seat of the disorganization is the *Rakta*, the *Ranjana* function of *Pitta* is to bear the brunt. In keeping with the basic doctrines relating to causation of disease, *Ayurveda* considers that due to *nidanaa sevana* the process of *panduroga* is commenced with the prapoka of all three *Doshas* in the *Dhatus.* The Pitta Dosha takes leading part in the production of *Dhatushaithilaya* and *Dhatugaurava*. Then, occurs *balakshaya*, *varnakshaya* and *ojakshaya* arising out of the *Dosha Dushya pradushana*. Thus, the *Panduroga* is stated to be afflicted with *Raktalpta, Medalpata, Nisarata, Vaivarnata and Shithilendriya.*According to subject the role of *Dosha Dushya* in manifestation of *Panduroga* is described as below –

ROLE OF *VATA DOSHA*: Though *Pitta* is *pradhanadosha* in *Pandu roga, vata dosha* also plays an important role in manifestation of *pandu roga*. Out of five kinds of *vata* mainly *Vyana vayu* has a relation with the *samprapti* of *pandu roga*. *Vitiated vata* is responsible for *kampa, angasada*, *gatrashula, raushya, twaka parushya, kati-urupada ruka* etc.

ROLE OF *PITTA DOSHA*: *Pitta* is responsible for the normal colour of body but when it vitiates the *Rakta*, as it happens in *Pandu roga* the loss of complexion or *Panduta* occurs.

ROLE OF *KAPHA DOSHA*: *Kapha* seems to play a vital role in the development of *Panduta*. According to *Charaka*, any person in whom there is a depletion of *vata*, develops the *panduta* due to the combined action of *Pitta* and *Kapha*.

It has also been stated that *santarpan* which broadly means anabolism, brings about an increase in *Kapha* which in term may cause the disease by generating *Ama* and causing *mandagni*. (Ch.Su.23/5). Thus, any diet which may increase *kapha* or any disease associated with increase in *kapha* can cause a change in complexion or *Panduta*. Vitation of *Kapha* is responsible for *Gaurava, Nidraluta, Mandagni, Alasya, Alpawaka* etc.

DUSHYAS* OF *PANDU ROGA: *Charaka* and *Vagabhatta* implicate *Twacha, Rakta* and *Mamsa* as the dominant *dushyas* vitiated in *Panduroga*. A fair approximation of *dhatus* involved can also be understood by a detailed study of symptoms.

The symptoms such as *Aruchi, Jwara, Panduta, Gaurava* and *Tandra* are indicative of *Rasadhatudushti*. *Angamarda* indicates the involvement of both *Rasa* and *Raktadhatu*.

Karshya is indicative of *mamsadhatu dushti*. *Atisweda* and *Swedabhava* are suggestive of involvement of *Twacha*, *Mamsadhatu* and *Medodhatu*. *Shiranalomata* is an important indicative of *Asthidhatu*. The loss of lusture and debility are suggestive of depletion of *Oja*.

CONCLUSION

Pandu roga is Pitta pradhana vyadhi, Pitta is responsible for the normal colour of the body but when it get vitiated, the Rakta as it happens in Pandu roga and thus loss of complexion or Panduta occurs.

Pandu roga is also considered as *Santarpan-janyavyadhi*, which broadly means Anabolism, brings about an increase in *kapha* which in term may cause the disease by generating *Ama* and *Mandagni*(Cha. Su.23/5).Thus, any diet which may increase *kapha*,any disease associated with increase in *kapha* can cause a change in complexion or *Pandu roga*.Though *Pitta* is *pradhanadosha* in *Panduroga*, *Vatadosha* also plays crucial role in manifestation of *Pandu roga*, mainly *Vyana vayu* has a relation with *Samprapti* of *Pandu roga*.

In modern era , there is different lab diagnostic test mention for anaemia with considerable result but no significant diagnostic tools is there for chronic anaemia which occurs due to metabolic defects. It is obvious that Anaemia is most common among females due to menstruation, poor general health, improper and inadequate diet which leads to malnutrition leading to Anaemia. Main cause of Iron deficiency is improper iron absorption in the GIT. *Pandu roga* can be effectively compared with Anemia on the grounds of its similar signs and symptoms. So *Ayurveda* can provide better diagnosis with the help of above information. We can easily diagnose the types of anaemia and confirm it with different lab diagnostic test for further management.

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