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 B₁₂, 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 on level 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 B₁₂ and folic acid deficiency which known as Megaloblastic 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

(B) **Morphological**:-
- 1. Normocytic normochromic anaemia
- 2. Normocytic hypochromic anaemia
- 3. Macrocytic anaemia
- 4. Microcytic hypochromic anaemia¹
Initial classification of anaemia:

The functional classification of anaemia has three major categories.
1. Bone marrow production defect (hypoproliferative)
2. Red cell maturation defect (ineffective erythropoiesis)
3. Decreased red cell survival (haemolysis)

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 haemorrhage. However, the replacement of RBC’s doesn’t occur quickly and it taken at least 4 to 6 weeks.

Decreased RBC’s count causes hypoxia, 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 haemoglobin 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:**
- Brittle nails
- Spoon-shaped nails
- Brittle hair
- Topley of papilla in tongue
- Dysphagia (difficulty in swallowing)

**Thalassemia:**

The thalassaemia’s are a diverse group of hereditary disorder in which there is reduced synthesis of one or 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.

**Types:**
1. α- Thalassaemia- α- chain is absent or abnormal
2. β- Thalassaemia- β- chain is absent or abnormal

**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 them 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.

**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 disorders occur in the following 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.

**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 granulo-poasis and mega-karyo-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.

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.

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.

Classification of haemolytic anaemia:-
I. Acquired:-
a) Autoimmune haemolytic anaemia (AIHA):
   a. Warm antibody of AIHA
   b. Cold antibody of AIHA
   b) Drug-induced immune-haemolytic anaemia
   c) Isoimmune 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 (haemoglobinopathies):
   i. Structurally abnormal haemoglobins:- Sickle syndrome
   ii. Reduced globin chain synthesis:- Thalassemia

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) \(^1\).

**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 (\(4^0\)c) 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 \(\alpha\)-chain are normal and \(\beta\)-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 syndrome occur 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 \(\beta\)-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 to decreased 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):

<table>
<thead>
<tr>
<th>Sr. no</th>
<th>Test</th>
<th>Test details</th>
<th>Normal value</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>RBC</td>
<td>Total RBC</td>
<td>M-4.3-5.7 million cells/mcl</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>F-3.9-5.0 million cells/mcl</td>
</tr>
<tr>
<td>2</td>
<td>MCH</td>
<td>Mean corpuscular haemoglobin</td>
<td>27-31 picro-gram/cells</td>
</tr>
<tr>
<td>3</td>
<td>MCHC</td>
<td>Mean corpuscular haemoglobin concentration</td>
<td>32-36 grams/deciliter</td>
</tr>
<tr>
<td>4</td>
<td>MCV</td>
<td>Mean corpuscular volume</td>
<td>80-100 femto-liter</td>
</tr>
<tr>
<td>5</td>
<td>Hb</td>
<td>Haemoglobin</td>
<td>M-13.5-17.5 g/dl</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>F-12.0-14.5 g/dl</td>
</tr>
<tr>
<td>6</td>
<td>PCV</td>
<td>Haematocrit</td>
<td>M-38.8-50 %</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>F-34.9-44.5 %</td>
</tr>
</tbody>
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- 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. HbA₂: Absent 2%
4. HbF : 98%
5. Hb: Reduced
6. Haematocrits: Reduced
7. MCH : < 22pg
8. MCV : < 50-70fl

**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 AMAR-KOSA).

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**.

**ROLE OF PITTA IN PANDUTA**: There is a significant role of **Pitta** in varnautaptti, and **Acharay Charaka** has quoted that **Pittadosha** is very imp (substance) in Natural colour of body, and if **pitta** gets vitiated the 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. **Pitta** Pratishayay, **Pitta Kasa**, **Pittadushta** Stanya, **PittajaPrameha**, **PittaArsha** etc. He also observed that pandut-vam is main observing symptom in these Roga.

**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 a 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 assimilative process. If **Kshara** and **Amal** are used in excess it might be that, they may injure the gastric mcosa 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 vataprapoka...
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.


RUPA:
PratityatmaLing or Cardinal symptom of this disease is Panduta or Pandubhava, which is invariable feature. Various types of discoloration 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 Dhatu 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 Doshha & Dhatu.

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


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. Mridbhakshajany 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 in 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 raktah 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 Dhatshaithilaya 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, Vaivar-nata 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,�at rashula, 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 Tanda 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 Aṣṭhidhatu. The loss of lusture and debility are suggestive of depletion of Oja.

CONCLUSION

Pandu roga is Pitta pradhana vyadhī, Pitta is responsible for the normal colour of the body but when it get vitiatiated, the Rakta as it happens in Pandu roga and thus loss of complexion or Panduta occurs. Pandu roga is also considered as Santarpajanayavyadhi, which broadly means Anabolism, brings about an increase in kapha which in term may cause the disease by generating Ama and Mandagnī(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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