



# Pandu an review article: ancient ayurveda and modern aspect

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## ABSTRACT:-

In *Ayurveda*, *Pandu roga* is a considered as a specific disease with its own Specific *Nidana*, *Purvarupa*, *Rupa*, *Samporapati* and *Chikitsa*. Thus an attempt has been made to study the disease *Pandu roga* according to Ayurvedic text.

*Pandu*, is a disease characterized by pallor of body which strikingly resembles with 'Anaemia' of modern science, disease to reduction in number of RBCs per cu mm of Blood and quantity of Hb% resulting in pallor like other symptoms.

The need for the discussion of *Pandu roga* becomes important due to the gravity of the problem. It is the commonest nutritional disorder all over the world and forms a major problem of mankind especially in a country like India due to low socio economic status, illiteracy and malnutrition in a major part of the population. It affects the physical and mental health of children, affects school performance and increases susceptibility to various infections. Further infants born to anaemic mothers have low Iron reserves and fall prey to Anaemia later in life with its adverse consequences.

The word Anaemia in Greek means a lack of blood. Actually it is a Deficiency of haemoglobin and there are various grades of this deficiency. Anaemia is the most common and widespread nutritional disorder in the world. As well as affecting a large number of children and women in developing countries. It is the only nutrient deficiency which is also significantly prevalent in industrialized countries.

The number are staggering 2 billion people - over 30% of the world's population are Anaemic may due to iron - deficiency and in resource poor areas.

Anaemia can result from a large number of causes, including nutritional Deficiencies, acute or slow loss of blood due to trauma or diseases, destruction of red blood cells due to various metabolic and immunological abnormalities or toxins, disease of the bone marrow, general systemic diseases like infections and various varieties of cancers, kidney failure because kidney produces an important hormone, erythropoietin,

which is required to stimulate the bone marrow to produce red blood cells and finally there may be genetic abnormalities in the formation of the haemoglobin molecules itself. The best known examples of these abnormalities are Thalassemia and sickle cell disease. In this write-up we will mainly concern ourselves with the nutritional aspect of Anaemia because that is where public awareness can help in the prevention and treatment of this disease.

A survey in Asia by World Health Organization showed that approximately 10% of men, more than 20% women (more than 40% during pregnancy); more than 50% of children of all ages and 92% of children below the age of two years suffer from Anaemia. In India 20-40% of the population, equally of all ages and either sexes have been suffering from Iron deficiency and nutritional anaemia. For its diffuse global spread IDA is recognized as a world health problem.

## HISTORICAL REVIEW

The study can be divided into:

- 1) *Vedic – Samhitas, Brahimanas, Upanishad, Kalpasutra* (2000-1000BC)
- 2) *Samhita* (1000BC – 500 AD).
- 3) *Sangraha Kala* (500 AD – 1700 AD).
- 4) *Adhunika Kala* (1700 AD onwards).

### **Vedic:**

*Ayurveda* is intimately connected with the *Vedas*. This is evident from the fact that the former is regarded as *Upaveda* of *Rigveda* (*Kashyapa*). Some scholars consider *Ayurveda* as *Upaveda* of *Atharvaveda* (1-50; 1-22-4) because of its similarity. *Hariman* and *Harita* are the diseases mentioned in *Rigveda* and *Atharvaveda*. *Hariman* is interpreted by *Sayana* as *pallor* and yellowishness of the body caused by the disease. Similar explanation is available in *Rigveda* (Rig 1-50-11-13).

Prof. P. V. Sharma opines, “Initially it is *Harita* denoting pallor of skin (*Pandu*) developing further into *Hariman* (*Kamala*, jaundice). In post-vedic texts, the same description about *Pandu* and *Kamala* is observed. It is interesting to note that they have retained the Vedic term *Hariman* in a slightly modified form as *Halimaka* and have described it as a type of *Kamala*.”

*RigVeda* prescribes its treatment with morning sun rays. In *Atharvaveda*, red cow’s milk and the drug *Anjana* has been said as *Harita Bhesaja*. *Kaushika Sutra* prescribes intake of cooked rice mixed with *Haridra* and anointing the same over the body for this disease.

In *Jaiminiya Brahmana*, there is reference regarding *Hariman*.

In the *Mahabharata*, King *Pandu*, who was the father of *Pandavas*, was suffering from *Pandutwa*.

This period is considered to be golden era in the *Ayurvedic* history. *Ayurveda* attained its summit both in conceptual and treatment aspects. In this period, it received patronage from both king as well as society. Like other diseases, *Pandu roga* is also elaborately discussed including its *Nidana*, *Samprapti*, *Lakshanas*, *Bheda*, *Upadrava* and *Chikitsa*. It is extensively dealt by Acharyas like *Charaka*, *Sushruta* and *Vagbhata*.

**Charaka:**

This *Samhita* plays main role in wide explanation of *pandu roga*, in *charaka samhitchikitsa sthana* 16th chapter *pandu roganidanas*, *bhedas*, *lakshanas* and *chikitsa* mentioned very clearly. In *pandu bhedas mrutbhakshana janya pandu* mainly explained very particularly.

**Sushruta:**

*Sushruta* explained *pandu roga* in *uttara tantra* 44th chapter in detailed along with its *nidana*, *purva roopa*, *Roopa*, *sadhya asadhya* and treatment. *Kamala* and other diseases are maintained as a stage of the *pandu roga*. *Sushruta* explained synonyms for the term *pandu* as *kamala*, *panaki*, *panduva*, *kumba kamala*, *laghavaka* and *alasangam*.

**Vagbhata:**

*Astanga hridayam nidana sthana* 13th chapter *pandu roga nidana*, *bhedas*, *lakshanas* and *upadravas* mentioned very clearly. Over all the *brihatrayee* the detail description of *pandu roga* is available and which is mostly followed by *laghuthrayees* in its treatment and other aspects.

**Madhava nidana & Bhava prakasha:**

In *madhava nidana* 8th chapter explained *pandu roga nidana*, *bhedas*, *lakshanas* and *upadravas* and also *sadhya asadhya*.

In *Bhava prakasha* 46th chapter explains the disease of *pandu roga nidana*, *bhedas*, *lakshanas* and *chikitsa* very clearly.

The description about *pandu roga* is also seen in *ksayapa samhitha*, *belasamhitha*.

**Sangraha (500-1700 AD):**

*Chakrapani*, *Dalhana*, *Indu*, *Vijayarakshita*, *Srikantadatta*, *Adamalla*, *Amarasimha*, *Madhavakara*, *Sharangadhara*, *Bhavamishra*, *Yogaratanakara*, *Arunadatta* and *Hemadri* have dealt *Pandu Roga* in detail. They have commented and discussed on the important previous works. In *Garuda Purana*, in *Nidana*, many diseases are described including *Pandu* and treatment being *Loha Churna* administered with *Takra*.

**Adhunika Kala (1700 AD onwards):**

Kaviraja Sri Rama Raksha Pathak has devoted a complete book to *Pandu Roga* giving elaborate discussion regarding different aspects of *Pandu Roga*. *Rasa Tarangini* by Sri Sadananda Sharma and *Bhaishajya Ratnavali* by Sri Govind Das have also contributed descriptions regarding *Pandu roga* during this period.

**VYUTPATTI & PARIBHASHA:**

The word *Pandu* is formed from the root *dhatu* "PADI GATOU" means *Gati* i.e. *parinama* or transformation. This signifies the transformation of various *dhatu*s from *ahara rasa*.

The disease *Pandu* is named after the *Varna*, as it is a mixture of *shweta* and *peeta Varna*<sup>2</sup>. *Pandu Varna* is the combination of *shweta* and *peeta Varna* in equal proportion, similar to pollen grains of *Ketaki* (*Harita samhitha*).

In *Shabdakalpdruma* *Pandu Varna* is mentioned as combination of *shweta* and *peetha*. In *Raja nighantu* *Pandu Varna* is the combination of *shukla* and *peetha Varna*.

In Ayurvedic classics, different definitions has been given, stating -

The disease in which *Pandu bhava* is more predominant is called *Pandu roga*<sup>3</sup>.

The disease in which *Panduthwa* is predominant<sup>4</sup>.

Predominant features in all *Pandu bhedha* are *Pandu Varna*<sup>5</sup>. Disease named after *Panduthwa*<sup>6</sup>. It is evident that the colour is being used as main criteria for diagnosing and differentiating the vyadhi Pandu from other diseases.

### **Paryaya**

Various *paryaya* are mentioned for *Pandu roga* which are mainly based on the colour<sup>7</sup>. They include - *Pandu, Kamala, Panaki, Kumbhava, Lagharaka, Alasakshya, Haridra, and Haritha*.

Though *Kamala*<sup>8</sup>, *Kumbha kamala* has been mentioned as *paryaya* even then separate *nidana, lakshana* and *chikitsa* are mentioned. *Dalhana* clarifies that *Susrhutha* has considered it as synonyms only based on *Kamala* being one of the *avasta vishesha* of *Pandu*.

### **NIDANA**

क्षाराम्ललवणात्युषणविरुद्धासात्म्यभोजनात्|

निष्पन्माषपिण्याकतिलतेलनिषेवणात् ||

विदग्धेऽत्रेदिवस्वप्नादव्यायामान्धेथुंनान्तथा|

प्रतिकर्मतुर्वेषम्याद्वेगानांचविधारणात् ||

कामचिंताभयक्रोधशोकोपहतचेतसः|(charak.chi 7-9/16)

Nidana in ayurvedic classics have two different meanings

- 1) *Vyadhi bhodaka hetu*
- 2) *Vyadhi janaka hetu*.

That which points out those factors, which help us to know clearly about the disease, is *vyadhi bhodaka hetu* and that which points out those factors that produces the disease is called as *vyadhi janaka hetu*. Here *nidana* is used, as *vyadhi janaka hetu* and the knowledge of these are very important for the proper understanding of *samprapti, sadyaasadyata, upadrava* and *chikitsa*. The *nidana parivarjana* forms the first line of treatment. The factor, which supports the formation of the disease, is summerised under four major headings so that it could be studied elaborately.

- 1) *Aharaja nidana*
- 2) *Viharaja nidana*
- 3) *Chikitsa apacharaja nidana*
- 4) *Nidanaarthakara roga*

### **AHARAJA NIDANA**

Improper diet and dietic practices are the prime factors responsible for the disease manifestation particularly when taken in excess. Such etiological factors related to food are<sup>9</sup>,

- 1) *Rasa – Amla, lavana, kshara*.
- 2) *Guna – Ruksha, ushna, tikshna*.

3) *Veerya – Ushna*.

4) *Dravya – Vidagdha anna, nishpava, pinnyaka, matsya, amisha, pista, paya, tila taila, masha atisevana, Madhya*.

### IMPROPER DIETIC PRACTICES

1) Concept of *virudha ahara*.

2) Improper practice of *ahara sevana vidhi* like *adhyasana, vishama ashana*.

### VIHARATAHA

1. *Ratri jagarana* 2. *Ati nidra* 3. *Divaswapna* 4. *Vegadharana*<sup>10</sup>

5. *Ativyayama* 6. *Avyayama* 7. *Ativyavaya* 8. *Ritu vaishamya*

### MANSIKA KARANAS

*Acharya Charaka* has given some of the *manasika karanas* which could pre-dispose a person towards the disease *Pandu*. They include<sup>11</sup>,

1. *Kama* 2. *Krodha* 3. *Chinta* 4. *Bhaya* 5. *Shoka*

### CHIKITSA APACHARAJA

1. *Panchakarma vaishamya*<sup>12</sup>

2. *Vamana virechana vyapat*<sup>13</sup>.

3. *Snehapana vibrama can cause Pandu*<sup>14</sup>.

4. *Grahi oushadi prayoga in ama atisara*<sup>15</sup>.

5. *Akala sneha prayoga*<sup>16</sup>.

6. *Chardi nigrahana*<sup>17</sup>.

7. *Ati yoga of yapana vasti*<sup>18</sup>.

### NIDANA ARTAKA ROGA

They can be classified into two depending up on the *karanas* for the *vyadhi*.

***Nija karanas for the roga***

***Angantu karanas for rogas***

*Rakta arbudha*<sup>20</sup>

*Katika taruna marma vedha*<sup>19</sup> *Antarlohita*<sup>21</sup> *Raktavahi*

*dhamani vedha*<sup>45</sup> *Raktapita upadrava*<sup>22</sup> *Revati graham*<sup>46</sup>

*Rakta pradra*<sup>23</sup>

*Mamsa marma abhighata*<sup>47</sup>

*Rakta kshaya*<sup>24</sup>

*Rajiman sarpa damsha*<sup>48</sup>

*Raktha srava*<sup>25</sup>

*Udarastha visha*<sup>49</sup>

*Punaravartaka jwara*<sup>26</sup>

*Mushaka damshttra*<sup>50</sup>

*Grahani*<sup>27</sup>

*Antarmruta shishu*<sup>51</sup>

*Arsha poorva roopa*<sup>28</sup>

*Pureeshaja krimi*<sup>29</sup>

*Rakta pitta*<sup>30</sup>

Asrugdhara<sup>31</sup>Kaphaja yoni vyapath<sup>32</sup>Pleehodara<sup>33</sup>Yakrudalyudra<sup>34</sup>Pittaja pratishaya<sup>35</sup>Vyavaya shosha<sup>36</sup>Pittaja kasa<sup>37</sup>Shukra kshaya<sup>38</sup>Beejopaghatha klaibya<sup>39</sup>**POORVA ROOPA**

These features develop before the actual onset of disease they give clues about the forth-coming disease and are called Prodromal symptoms or *Poorvaroopa*.

These are produced during the stage of *Sthana samshraya* of vitiated *doshas*. *Poorvaroopa* will give us the information regarding the forthcoming disease;

If treated at this stage, progress or severity of disease can be checked.

*Poorvaroopa* may continue to exist in the actual state of disease as *Roopa*, some may disappear. Some times *poorvaroopas* contradictory to *roopas* may also appear, like *vibandha* before *atisara*.

**Table no .1 POORVA RUPA OF PANDU**

Sl. no	Lakshana	C. S.	S. S.	A.H.	A.S.	M.N.	B.P.	H.S.	Y.R.	G.N.
1	Hrudaya Spandadhikya	+	-	+	+	-	-	-	-	-
2	Roukshya	+	-	+	+	-	-	-	-	-
3	Swedabhava	+	-	+	+	-	-	-	-	-
4	Shrama	+	-	+	+	-	-	-	-	-
5	Twak Sphotana	-	+	-	-	+	+	+	+	+
6	Shteevana	-	+	-	-	+	+	+	+	+
7	Gatra Sada	-	+	-	-	+	+	+	+	+
8	Mrudbhakshana Iccha	-	+	-	-	+	+	+	+	+
9	Prekshana Koota Shotha	-	+	-	-	+	+	+	+	+
10	Avipaka	-	+	-	-	+	+	+	+	
11	Vitpeetata	-	+	-	-	+	+	+	+	
12.	Mootra Peetata	-	+	+	+	+	+	+	+	+

13.	Aruchi	-	-	+	+	-	-	-	-	-
14.	Alpa Vahni	-	-	+	+	-	-	-	-	-
15	Sada	-	-	+	+	-	-	-	-	-
16	Pipasa	-	-	-	-	-	-	-	-	-
17	Hrullasa	-	-	-	-	-	-	-	-	-
18	Urodaha	-	-	-	-	-	-	-	-	-
19	Anga gourava	-	-	-	-	-	-	-	-	-
20	Rakta lochana	-	-	-	-	-	-	-	-	-
21	Shareera Pandutwa	-	-	-	-	-	-	+	-	

### **SAMPRAPTHI**

The manner of *dosha* vitiation right from contact with the *nidanas* and the course they follow culminating in the development of specific clinical manifestation is known by the name *samprapthi*. Every factor connected with the process of disease at various stages is considered in detail in *samprapthi*. It gives a clear idea of the disease process helping management of the condition

*Charaka* considers *Pandu* to be a *santharpanaja rasa vaha srothovikara* and *susrutha* says it to be *rakthavaha srotovikara* and clearly projects *pitta dosha* as the major factor behind the whole pathogenesis in *Pandu*. Due to the involvement of *rasa, raktha* and *ojas* the main presenting symptom is *Panduta, indriya-bala- varnahani* and the *nidanas* for *Pandu* is *pittaja nidanas*.

*Samprapthi* of *Pandu* can be explained and understood based on *shadkriyakala-sanchaya, prakopa, prasara, sthanasamsraya, vyaktha* and *bheda*.

#### **Sanchaya**

This is the initial stage of the disease, where in the *doshas* is accumulated and stagnated in its own *sthanas*. *Chayavastha* is characterised by vague and

Ill-defined symptomatology though some symptoms may indicate the underlying *doshic* involvement such as dullness and fullness by *vata*, laziness and heaviness of limbs by *kapha*. There may be aversion towards contraries. Here since there is accumulation of *pitta dosha* the person may feel an aversion towards those factors, which are similar to that of *pitta*. If it is neglected or due to negligence in treatment it may enter in to *prakopa*.

#### **Prakopa**

The liquifaction of the accumulated *dosha* in previous stage as well, as if the person is continuously indulging in the *dosha* aggravating factors, it leads to *prakopaavastha*. In the present, contest the *pittakara nidanas* like *teekshna, amla, kshara* etc leads to further aggravation and excitation of *pitta dosha* providing perfect base for the manifestation of *Panduroga*. This stage may give rise to symptoms characteristic of *doshas* like abdominal pain, thirst, burning sensation, disinclination for food, nausea etc. Neglecting this stage result in the next stage, i.e. *praasara*.

**Prasaravastha**

The term *prasara* means to spread. In this stage the increased and excited *dosha* spread over to other parts, which is the *sthana* of other *dosha*. It is to be noted that *Vayu* which possess the power of locomotion is responsible for this stage and the *prakupitadoshas* especially *pitta* produces symptoms like burning

Sensation in various parts of the body, painful sucking sensation impaired digestion may be seen.

The accumulated *pitta dosha* from *hridaya* spread to various parts of body through *dasadamanis* in this stage. In the above three stage of *kriyakala* a vague manifestation of symptom will be seen which is not sufficient for diagnosis of *Pandu*. If it is detected in the stage, the progression of the disease may be arrested by timely intervention.

**Sthanasamsrayavastha**

The vitiated *dosha* relocate them in the site of other *doshas* vitiating the *dushyas* present there and mark the beginning of specific diseases pertaining to the site. Obviously, this stage represents the prodromal phase or the stage of *poorvaroop*a and disease is yet to manifest fully.

The *Doshas* thus spread through the *dasha dhamanies* relocate themselves in the *srothas* of *rasa*, *raktha* and *medovaha* producing *kha vaigunya* and get *asraya* in *twak mamsa* and vitiate *doshas* and *dushyas* like *asruk*, *twak*, *mamsa* resulting in various type of discolouration in the body and manifestation of prodromal symptoms like *hridayaspandanadhikya*, *roukshya*, *sweda abhava*, *srama* are seen.

**Vyarthavastha**

This stage in which the disease manifest completely with its symptoms in a fully developed form. This stage is marked by presence of cardinal features like *Panduta*, *hridrava*, *aruchi arohana-ayasa*, *shishira dweshi*.

**Bhedavastha**

The disease when neglected in *vyarthavasta* turns into *bhedavasta*. This stage can make the condition worse by manifestation of *kamala* and enters into deep *dhatu*s. The disease proceeds into more severe form due to extensive *dhathukshaya* and finally attains the *asadhyaavastha*.

**Samprapthi of Panduroga<sup>52</sup>:**

A disease has been deliberated due to the result of *dosha dushyasammurchana*. Due to the *nidana*, *sevana* in the form of *mithya ahara* and *vihara*, there will be *pitta pradhana tridosha prakopa* and *Vagbhata* says that there is an increase in *snigdhattha* and the *dusthi* of *pitta* is in the form of *dravyatha vrudhi*, *gunata* and *karmata kshaya*. This vitiated *dosha* is carried to *hridhaya*, *vyana vayu* throws this through the *dasha dhamani* to the *sarva shareera*, and get *stana samsraya* between *twak* and *mamsa* resulting in discolouration of the skin such as paleness and yellowish discolouration of which *Panduta* being the predominant colour. There will be *indriya* and *ojohani*, which will produce *Varna bala kshaya* and *indriya saithilya* and produce five types of *Pandu* with specific character.



**Vyadhi ghatakas**

Vyadhi ghatakas have been described with relation to the disease process and *nidana* is said to cause *vyadhi* by deranging the *samprpti ghatakas*. Here an effort is made in narrating the role of these in producing the *vyadhi Pandu*.

- 1) *Dosha*<sup>53</sup>- *Pitta pradhana tridosha*
- 2) *Dooshya*<sup>54</sup>- *Rasa, Rakta, Medha*
- 3) *Agni- Agni dusthi (mandagni)*
- 4) *Ama- Amaja vyadhi*
- 5) *Srotas- Rasa vaha srotas, Raktavaha srotas, Medovaha srotas*
- 6) *Udbhava sthana – Amashaya*
- 7) *Vyakta sthana – Sarva shareera*
- 8) *Sanchara sthana- Dashadhamanis and Sarvashareera*
- 9) *Ashraya- Twak and Mamsa*

**PITTA**

*Pitta* is the prime *dosha* involved for the manifestation of disease *Pandu* and the Normal physiological understanding of *pitta bhedas* gives its importance in the *vyadhi*.

(i) ***Pachaka pitta***- The main *karma* of *pachaka pitta* is to digest the *ahara* and *Sara kitta vibhajana*.

This *bheda* of *pitta* gets vitiated by *nidana sevana* and cause *agnimandya* and leads to formation of improper *adyadhatu, ama* and *dhatushaithilya*.

(ii) ***Ranjaka pitta***- Its *sthana* is *yakrit* and *pleeha* and is responsible for the *rasa ranjana*. *Sushruta* says that *amashaya* is the *ranjaka pitta sthana* and when there is a vitiation of *Ranjaka pitta*, there is improper conversion of *rasa* into *rakta*. *Sarangadhara* says that *hridaya* is the *sthana* of *ranjaka pitta*.

(iii) ***Alochaka pitta***- It is present in the eyes and is responsible for *roopalochana*. In *Pandu* due to the *indriyasthityata* proper *drishti* does not occur.

(iv) ***Sadaka pitta***- It is present in the *hridaya*, helps for *dhee, dhriti, dhairya, abhiprekshata sadhana* and these functions are hindered, and *manas* gets involved.

v) ***Bhrajaka pitta***- The seat of this variety of *pitta* is *twak*, it gives color to the skin, and vitiation of the *bhrajaka pitta* produces alteration of the normal color and brings about *Panduta* of *twak*.

**KAPHA**

*Kapha* also has an important role to play in the pathogenesis of *Pandu roga*. *Avalambaka kapha* is responsible for *uru palana*, in case of *Pandu*, due to the *kaphavridhi* the *sthana* is vitiated and that leads into *hriddrava, arohana ayasa*.

*Bhodakakapha* is responsible for *ruchi grahanam*, which is destroyed in *Pandu*. *Shleshaka kapha*, which is responsible for *sandhi samsleshana*, gets vitiated and leads to *parvashoola*. *Kapha* situated in the *twak* produces *shwetaavabhasata*.

**VATA**

The role of *vata* is very important in the manifestation of the disease because the vitiated *dosha* enters the *hridaya* and it is carried from the *hridaya* to the *sarvashareera* through the *dasha dhamanis* by *vyana vayu* and gets displaced between *twak* and *mamsa*.

**DOOSHYA**

(i) *Rasa dhatu*- *Acharya Charaka* mentions it as a *rasa pradoshaja vikara* and *Chakrapani* comments that the aggravated *pitta dosha* does the *kshapana* of *rakta poshaka rasa* and its *anutpadana* due to the impairment of the *Agni* resulting in *dhatu shaithilya*.

(ii) *Rakta dhatu*- *Raktalpata* is mentioned by *Charaka* as *pradhana lakshana* and *pitta* being the *pradhana dosha* there will be the involvement of the *rakta dhatu*.

iii) *Medhas*- *ALPA medhata* is due to the improper *uttarothara dhatu* formation.

**AGNI**

There is a gross vitiation of *jataragni*, *bhoothagni* and *dhatwagni*.

The *nidana sevana* will cause increase in the *Drava guna* of *pitta* resulting in *jataragni mandhya*. This *jataragni* governs the state of functioning of all the agnis. This leads to the production of *Sama ahara rasa* and impaired conversion to subsequent *dhatu*s. So *poshana* of *dhatu*s and *upadhatu*s does not occur properly. This leads to *ojokshaya*.

**AMA**

*Agnimandhya* being one of the key factors for the *Pandu*, the role of *ama* cannot be ruled out.

**SROTAS**

Important *srotas* affected in *Pandu* is *rasa* and *raktavaha*. I) *Pandu* is a *rasa pradoshaja vikara*. In *Pandu* due to *agnimandya* *rasa dhatu* is first affected and *prakupita pitta* having *sthana samsraya* in *hridaya* which is the *moola sthana* for *rasavaha srotas* and many *rasa kshaya lakshana* and *dushti lakshanas* are seen in *Pandu*.

II) *Pitta dosha* and *rakta dhatu* are responsible for *Varna prasadana* i.e. *Agni gunabhuyishta*. There is also *alparaktata* and some symptoms of *raktavaha sroto dushti* like *bhrama*, *trishna*, *Panduta* are seen.

**SAMPRAPTI BHEDA OF PANDU****1) Sankhya samprapti:**

5 types of *Pandu* (*Charaka* and *Vaghbata*)

4 types of *Pandu* (*Sushruta*)

8 types of *Pandu* (*Harita*)

**2) Vidhi samprapti:**

The method in which the *dosha* is *kupita* leading into *dosha dushya sammurchana* and *vyadhi sanghatana* is caused as *vidhi samprapti*, which is mentioned above.

The *swatantra Pandu* is *sadhya* and *paratantra Pandu* is *kashta sadhya*.

**3) Vikalpa samprapti:**

The *amshamsha kalpana* of the *samprapti* of *Pandu* is separately dealt under the heading of *samprapti ghatakas* of *Pandu*.

**4) Pradhanya samprapti:**

*Pradhana dosha – pitta pradhana tridosha*

*Swatantra Pandu is pradhana*

*Nidanarthakara roga janita Pandu is apradhana*

*Anubandhya Pandu is apradhana*

**5) Bala samprapti:**

The *bala* or strength of *Pandu* depends on whether the *nidana*, *poorvarooopa* and *roopa* are manifested partially or completely.

**6) Kala samprapti:** It is the *samprapti* that confirms the role of a particular *dosha* in a disease i.e. the *bala* that produce the disease or increase its intensity with change in time like *Dina*, *ratri* etc. or in accordance with the stage of digestion.

**Vishesha samprapti:**

1) **Vataja Pandu-** Etiological factors, which mainly increase *vata* along with *pittadi doshas*, lead to the production of *Pandu roga* with *vata anubandha* producing *vataja Pandu*.

2) **Pittaja Pandu-** Etiological factors, which mainly increase *pitta* along with other *doshas*, leads to the production of *pittaja Pandu*.

3) **Kaphaja Pandu-** Etiological factors, which mainly increase *kapha* along with *pittadi dosha*, leads to production of *Pandu roga* with *kapha anubandha* thus producing *kaphaja Pandu*.

4) **Tridoshaja Pandu-** Etiological factors, which mainly increase all the *tridosha* simultaneously, lead to production of *tridoshaja Pandu*.

5) **Mrit bhakshanajanya Pandu**<sup>55</sup>- Habitual intelligence in eating *mrit* aggravates one of the *tridoshas*. If the *mrit* is of *kashaya rasa*, then it aggravates *vayu*. If it is *ushara*, then *pitta* gets aggravated, if it is *madhura kapha*. Because of its *ruksha*, *guna* the *mrit* causes *rukshata* in the *rasa* then the undigested *mrit* produces *savarodha* of *srotas* and causes *indriya bala hani*, *agnimandya* and thus producing *Pandu*.

**Classification of pandu:**

- *Charaka* has classified *Pandu roga* into five<sup>56</sup>, i.e *Vataja*, *Pittaja*, *Kaphaja*, *Sannipataja* and *Mruhbakshanaja*.
- *Sushrutha* mentions only four types of *Pandu* and says- *Mruhbakshana* is one of the causes for *Pandu roga*.
- *Vijayaraksita* the commentator of *Madhavanidana* supports the opinion of *Charaka* and says that signs and symptoms as well as treatment of *mruhbakshanajanya Pandu* are entirely different than the general course of *Panduroga* hence it should be considered as separate one.
- *Harita* have included *Kamala*, *Kumbhakamala* and *Haleemaka* as *Pandu bhedhas* and described the disease *kamala* as later stage of *Pandu roga*.

Table no. 2 TYPES OF PANDU

Sl. No.	Types	C. S.	S. S.	A. H.	A. S.	M. N.	B. P.	Y. R.	Sh. S.	G. N.	K. K.	Ba sa.	H. S.
1.	<i>Vataja</i>	+	+	+	+	+	+	+	+	+	+	+	+
2.	<i>Pittaja</i>	+	+	+	+	+	+	+	+	+	+	+	+
3.	<i>Kaphaja</i>	+	+	+	+	+	+	+	+	+	+	+	+
4.	<i>Sannipataja</i>	+	+	+	+	+	+	+	+	+	+	+	+
5.	<i>Mrudbhakshanaja</i>	+	+	+	+	+	+	+	+	+	+	+	+
6.	<i>Kamala</i>												+
7.	<i>Kumba Kamala</i>												+
8.	<i>Haleemaka</i>												+

**ROOPA**

The term *roopa* implies to both signs and symptoms through which a disease is identified. In addition to the cardinal signs and symptoms, a number of constitutional symptoms also manifest in *Pandu roga*. A few of the symptom aid in distinguishing the type based on *dosha anubandhata*. Accordingly that can be classified into:

- *Pratyatma lakshanas* (cardinal signs & symptoms)
- *Samanya lakshana* (general signs & symptoms).
- *Vishista lakshana* (distinguishing features of dosa anubandhata)

**Pratyatma lakshanas:** The *vyadhi Pandu* is distinguished by the unique paleness of skin. *Panduta* of *twak* can be considered as the *pratyatma lakshna* of *Pandu roga*. This impairment of *skin* is due to *rasa* and *rakta kshaya* and the colour is almost like the pollen grains of *Ketaki flower*.

*Charaka* says that *arohanaayasa* is specifically seen in all the variety of *Pandu roga* due to the *rakta kshaya* and *vyana vata vridhi*.

“*Visheshat arohanaayasam*” specific colour of the skin depends on the specificity of *doshic* predominance.

**Samanya lakshana:** A number of constitutional symptoms manifest in varying degree, which are considered as general symptoms.

They are as follows; *alpa rakthata*, *dourbalya*, *hriddrava*, *swasa*, *bhrama*, *kati-uru -parshva ruk*, *shotha*, *shoonakshi koota*, *gourava*, *sadana*, *mandagni*, *karna ksheweda*, *hata prabha*, *shweta akshitwa*, *satwa hani*, *shweta nakha*.

Table no .3 SAMANYA LAKSHANA

Sl. No.	SAMANYA LAKSHANA	C.S.	S.S.	A.H.	A.S.	K.S.
1.	<i>Karna Kshweda</i>	+	-	+	+	-
2.	<i>Hatanala</i>	+	-	+	+	+
3.	<i>Dourbalya</i>	+	-	+	+	-
4.	<i>Sadana</i>	+	-	+	+	-
5.	<i>Bhrama</i>	+	-	+	+	-
6.	<i>Annadwasha</i>	+	-	+	+	-
7.	<i>Shrama</i>	+	-	+	+	-
8.	<i>Gatrashoola</i>	+	-	-	-	-
9.	<i>Jwara</i>	+	-	+	+	-
10.	<i>Shwasa</i>	+	-	+	+	-
11.	<i>Gaurava</i>	+	-	+	+	-
12.	<i>Aruchi</i>	+	-	-	-	-
13.	<i>Gatramarda</i>	+	-	-	-	-
14.	<i>Gatrapeeda</i>	+	-	-	-	-
15.	<i>Gatronmathana</i>	+	-	+	+	-
16.	<i>Shunakshikoota</i>	+	-	+	+	+
17.	<i>Hareeta Varnata</i>	+	-	-	-	-
18.	<i>Sheerna lomata</i>	+	-	+	+	-
19.	<i>Hataprabha</i>	+	-	-	-	-
20.	<i>Kopa</i>	+	-	+	+	-
21.	<i>Shishiradwasha</i>	+	-	+	+	-
22.	<i>Nidralu</i>	+	-	-	-	-
23.	<i>Shtivana</i>	+	-	+	+	-
24.	<i>Alpavak</i>	+	-	+	+	-
25.	<i>Pindikodweshtanam</i>	+	-	-	-	-
26.	<i>Katiruk</i>	+	-	-	-	-
27.	<i>Kati sada</i>	+	-	-	-	-
28.	<i>Padaruk</i>	+	-	-	-	-
29.	<i>Pada sada</i>	+	-	-	-	-
30.	<i>Uru ruk</i>	+	-	-	-	-
31.	<i>Uru sada</i>	+	-	-	-	-
32.	<i>Dhatushaithilya</i>	+	-	+	+	-
33.	<i>Ojo gunakshaya</i>	+	-	+	+	-
34.	<i>Alparaktata</i>	+	-	+	+	-

35	<i>Alpamedaskata</i>	+	-	+	+	-
36	<i>Nissarata</i>	+	-	+	+	-
37	<i>Hridrava</i>	+	-	+	+	-
38	<i>Shithilendriya</i>	+	-	+	+	-
39	<i>Shareera Vaivarnya</i>	-	-	-	-	+
40	<i>Twachi Panduta</i>	+	+	-	-	-
41	<i>Nabhi Shotha</i>	-	-	-	-	+
42	<i>Shwetha Akshi</i>	-	-	-	-	+
43	<i>Shwetha Nakha</i>	-	-	-	-	+
44	<i>Shwetha Vakrata</i>	-	-	-	-	+
45	<i>Shotha</i>	-	-	-	-	+
46	<i>Karshya</i>	-	-	-	-	+
47	<i>Satwahani</i>	-	-	-	-	+

**Vishista roopa:** The signs and symptoms specified to virulence of *dosa* are an important part of our study for early diagnosis and purpose of treatment.

1. *Vataja Pandu*<sup>57</sup>: *vata vridhi* produces various *vataja* manifestations in the presentation of *Pandu roga* like *krushnata of nakha, angamarda, ruja, toda, shiroruja, varchashosha* etc.

2. *Pittaja Pandu*<sup>58</sup>: *pitta vridhi* produces various *pittja* presentations like *peetaavabhasa, jwara, trishna, pipasa, murcha*.

3. *Kaphaja Pandu*<sup>59</sup>: *kapha vridhi* produces various *kaphaja* manifestations like *shukala varnata, gourava, shwayathu, aruchi, praseka*.

4. *Tridosaja Pandu*<sup>60</sup>: vitiation of all the *dosa* causes severe degree of *dhatu shaithilya* and *dhatu gourava* from which deterioration of *dhatu*s and *ojas* occur rapidly and are considered *ashadhyas*. *Harita* has clearly explained the *sannipata Pandu lakshana; thandra, alasya, vit bheda, hrullasa, kasa, shotha, jwara, moha, trishna*.

5. *Mrit bhakshana janya Pandu*<sup>61</sup>: it causes *Agni mandhya* and *roukshata* to *shareera, shotha, dhatu dourbhalya, indriya-teja-bala-virya kshaya* and also may produce *krimi*.

*Madhavakara* has considered it as a *vyadhi hetu*. *Susruta* has considered it upper *tridosaja Pandu* as in take of *mrit* causes *tridosaja prakopa*.

### SADHYA- ASADHYATA<sup>62</sup>

The prognosis of a disease can be established only after consideration of the *sadhyaasadhyata*. The signs and symptoms indicating the incurability of the disease are as follows,

- When the disease is *chirakari*.
- When there is *Varna kshaya* due to *rukshata*.
- Due to the chronicity of *vyadhi* when *shotha* has appeared.
- *Baddhata* or *alpata* of *vit pravruithi*.
- When patient view every thing as yellow.
- When there is *harita* and *sakapha mala pravarti*.

- When patient is affected with *chardi*, *murcha* and *trishna*.
- When there is *Panduta shwetabhasa* due to *asrik kshaya*.
- When *shotha* is seen in extremities or in trunk and emaciation of body parts.
- *Tama praveshya*.
- *Pandu* associated with *jwara* and *atisara*.
- *Shotha* in *guda pradesa*, *shepha* and *mushka*.
- *Panduta* of *danta*, *nakha*, *netra* and *Pandu darshi*.
- *Tridosaja Pandu* is *asadhya* for *chikitsa*.

### UPADRAVA

*Upadravas* are those ailments that are *rogashrita* and are manifested after the manifestation of main disease. These are nothing but the supervening symptoms that occur along with the disease or as a sequel. These generally subside when the main disease subsides or some times indicate the fatality of the disease. Some times *samanya lakshana* may itself may be converted into *upadrava* by increasing their severity. *Susruta* has explained the *upadrava*.

**TABLE NO. 4: UPADRAVA OF PANDU**

Sl. no.	Lakshana	S.S	A.H.	Bas
1	<i>Aruchi</i>	+	-	+
2	<i>Pipasa</i>	+	-	-
3	<i>Chardi</i>	+	-	+
4	<i>Jwara</i>	+	-	-
5	<i>Shiroruja</i>	+	-	-
6	<i>Agnisada</i>	+	-	-
7	<i>Shopha</i>	+	+	+
8	<i>Kanthagata abalatwam</i>	+	-	-
9	<i>Moorcha</i>	+	-	-
10	<i>Klama</i>	+	-	-
11	<i>Hridayavapedanam</i>	+	-	-
12	<i>Shwasa</i>	+	-	-
13	<i>Atisara</i>	+	-	+
14	<i>Kasa</i>	+	-	-
15	<i>Daha</i>	+	-	-
16	<i>Avipaka</i>	+	-	-
17	<i>Swarabheda</i>	+	-	-
18	<i>Sada</i>	+	-	-
19	<i>Adhmana</i>	-	-	+

20	<i>Tandra</i>	-	-	+
21	<i>Pandu danta</i>	-	-	+
22	<i>Pandu nakha</i>	-	-	+
23	<i>Pandu netra</i>	-	-	+
24	<i>Pandu sangatha darshi</i>	-	-	+

### **ARISTA LAKSHANA**<sup>63</sup>

The signs and symptoms indicating the occurrence of death in the near future are referred to as *arista lakshanas*. There is no death with out the evolution of the *aristalakshana*. The *aristas* have been classified into *sthayi* and *asthayi* by Acharya Vagbhata and have stated that *sthayi arista* definitely kill the patient. Some of the *arista lakshanas* pertaining to *Pandu* are;

- Excess *Pandu varnata*.
- *Ati krisha*.
- Excessive *trishna*

### **Pathyaapathaya**<sup>64</sup>

*Pathya:*

#### **A. Ahara:**

1. *Suka dhanya varga - Purana Shali, Purana Yava, Godhuma*
2. *Shami dhanya varga - Mudga*
3. *Mamsa varga - Jangala Mamsa, Matsya.*
4. *Shaka varga - Patola, Kushmanda, Jeevanti, Bimbi, Punarnava, Nagakesara, Guduchi, Dronapushpi.*
5. *Phala varga - Kadali phala, Abhaya, Dhatri.*
6. *Ikshu varga - Ikshu Rasa*
7. *Gorasa varga - Takra, Ghrita, Navaneeta.*
8. *Mootra varga - Gomutra*
9. *Madya varga- Souviraka, Tushodaka.*
10. *Kritanna Varga- Yusha.*
11. *Anyā dravya- Haridra, Chandana, Yavakshara, Loha bhasma.*

#### **B.Karma:**

- i. *Vamana.*
- ii. *Virechana.*
- iii. *Abhyanga.*

#### **Apathya:**

##### **A. Ahara:**

1. *Rasa- Kshara, Amla, Katu, Lavana.*
2. *Anna- Viruddha bhojana, Asatmya bhojana.*
3. *Jala- Adhikambupana, Dushita jalapana.*
4. *Kritanna varga- Pinyaka.*



5. *Shamidhanya varga- Masha, Tila, Kulatha, Nishpava.*

6. *Sneha varga - Tila taila.*

7. *Gorasa varga- Dadhi masthu.*

8. *Madya varga- Saktu.*

9. *Ahara varga- Hingu, Tambula, Teekshnapadartha like Maricha, Vidahi padartha, Atyushna padartha.*

10. *Anyā dravya- Mruttika.*

#### **B. Vihara:**

*Agni, Atapa atisevana,*

*Adhika vyayama.*

*Adhika vyavaya.*

*Krodha.*

*Adhika marga gamana.*

#### **C.Karma:**

1. *Rakta Sruti.*

2. *Dhoomapana.*

3. *Swedana.*

4. *Vamana Vega dharana.*

#### **Food rich in iron:**

The inadequate diet results in iron deficiency and the best source of iron is red meat because haem can be absorbed as such.

- Vegetable have variable amount of absorbable iron, soybean is rich source.
- Iron rich foods include red meat, liver, green leafy vegetables, fruits like apple, apricot, spinach, egg yolk and fishes.
- Milk particularly cow milk is well known for its iron deficiency.
- Non-green vegetables are deficient in iron content
- Rice and bread are rich in phytates and prevent iron absorption.

**TREATMENT OF PANDU ROGA**

Treatment given in *Pandu* can be divided in to part<sup>65</sup>:

1) *Shodhana* 2) *Shamana*

**1) Shodhana:** Shodhana is done by *Snigdha*, *Teekshna Vamana* and *Virechana*. Prior to *Shodhana*,

a) *Snehana* is done as

- Body's *Sneha* quality is greatly reduced in *Pandu*.
- *ALPA raktata*, *ALPA medaskata* and *Ojo kshaya* cause predominance of *Rukshata* in the body.
- To bring back the *Shakhashrita dosha* to *Koshta* Eg. *Kalyanaka Ghrita*, *Panchagavyaghrita*.

b) *Swedana* has been contradicted in *Pandu*<sup>66</sup>. However, *Mridu swedana* can be performed.

c) *Shodhana* is done for:

1) *Koshta shuddhi*

2) To combat the *Dosha bahulyata*.

*Brihatrayees* accept both *Urdhwa* and *Adho shodhana* keeping in accordance with the condition.

*Shodhana* is followed by *Shamana oushadha* and *Pathya*.

**2) Shamana:** In *Shamana*, various single and compound preparations are told which include herbal, mineral and herbomineral preparations. Illustrating a few,

1) *Vyoshadya Ghrita*

2) *Shuddha Kanta Loha Bhasma*<sup>67</sup>

3) *Vidangadi Loha*<sup>68</sup>

A point of interest to be noted here is that most mineral preparations contain *Loha*.

**Mrudbhakshanajanya Pandu Chikitsa**<sup>69</sup>: At the outset, the *Balabala* of the patient has to be assessed.

**1) Shodhana:** *Teekshna shodhana* in order to remove the ingested *Mruttika*.

**2) Shamana:** 1) Medicated *Ghrita* ie *Sarpi* for *baladana*. Eg. *Kalyanaka Ghrita*

2) Treatment according to the *Prakupita Dosha*.

3) *Krimihara Chikitsa* in *Udara Krimi*.

**3) Nidana Parivarjana:** *Mruttika*, given *bhavana* with *Vidanga*, *Ela*, *Ativisha*, *Nimbapatra*, *Pata*, *Varthaka*, *Katurohini*, *Murva* and *Kutaja*. These Will produce aversion towards *Mrudbhakshana* i.e. *Dweshartha*. *Mrudbhakshanajanya dosha nashaka*.

**ANAEMIA****History:**

The term Anaemia is an ancient one and can be found in the *Carpus Hippocraticus*. It literally means without blood and is derived from Greek 'a' or without and 'haima' or blood.

James Combe, an Edinburgh Physician (1824), first used the term Anaemia in English. Gebrial Andral, a French physician, laid much of the foundation on modern concepts of Anaemia in 1843.

The therapeutic use of Iron was mentioned in Greek mythology in the story of Iphicius. Vanandeus applied the term chlorosis and was described by Jahonnes Lange in 1554. In 1830, Hoefer detected

hypochromia in blood. In 1832, Pierre described the response of chlorosis to his famous pills containing ferrous sulphate and potassium carbonate.

### Classification of Anaemia:

Classification of Anemia's by pathophysiology

#### I. Blood Loss

- a) Acute Haemorrhage
- b) Chronic Haemorrhage

#### II. Decreased Production of Red Blood Cells

1. Hameoglobin synthesis-

- a) Iron deficiency anaemia
- b) Thalassemia (hereditary)

c) Anaemia of chronic disease

2. D.N.A synthesis - Megaloblastic Anaemia
3. Stem cell - Aplastic Anaemia
4. Bone Marrow infiltration – a) Carcinoma b) Lymphoma
5. Pure Red cells Aplasia

#### III. Increased destruction (Classification of Haemolytic Anaemia)

##### a) Haemolysis (Intrinsic)

- 1) Membrane - i) Hereditary Spherocytosis ii) Elliptocytosis
- 2) Haemoglobin – i) Sickle cell ii) Unstable Haemoglobin
- 3) Glycolysis - Pyruvate Kinase
- 4) Oxidation - G6PD deficiency

##### b) Haemolysis (Extrinsic)

- Immune - i) Auto immune  
 ii) Drug toxicity  
 iii) Lympho Proliferative Disease

### IRON DEFICIENCY ANAEMIA

Historical Review:

- In 1554, Han Lounji had described the disease by the name of "Colorosis"
- In 17th cen. A.D. application of the name of anaemia was started.
- In 1829, it was defined clearly with specific definition.

### Definition:

Iron deficiency anaemia has been defined as iron store depletion refers to an imbalance between normal physiologic demands such as body growth, menstrual blood loss and pregnancy and the level of dietary iron intake.

**Etiology:**

- Defective Intake: Children, psychiatric patients, patients having anorexia.
- Defective Absorption: Gastrectomy, gastrojejunostomy, sprue syndrome.
- Excessive Demand: Growing children, female during reproductive years, thyrotoxicosis.
- Excessive Loss: Hookworm anaemia, bleeding piles, hiatus hernia, iron sequestration, pulmonary haemosiderosis, menorrhagia, recurrent haematemesis and melaena, recurrent blood donation, acute and chronic haemoglobin urea.

**Iron Balance:**

An adult male on a balanced diet will invest approximately 15 to 20 mg of iron/day, while the adult female will ingest 10 to 15 mg/day. In the male only 1 to 2 mg needs to be absorbed to replace the iron lost from desquamation of skin and mucosal cells. The adult pre-menopausal female needs to absorb more from the diet to make up for menstrual blood loss.

The same is true for the frequent blood donor. Infants, children and adolescents may be unable to maintain normal iron balance because of the increased demands of body growth and much lower dietary intakes of iron. This is also true for pregnant women. During the last two trimesters of pregnancy, the daily iron requirement increases to 3 to 5 mg, a level that can not be supplemented unless the diet is rich in heme iron or the women receives an iron supplement.

**Clinical Features:**

- General: Weakness, fatigue, lassitude, oedema, pallor, dry skin, lustreless hair, white sclera.
- Cardiovascular: Palpitation, anginal pain, sinus tachycardia, collapsing pulse, dancing carotids, and engorged neck veins, haemic murmur, congestive cardiac failure.
- Respiratory: Breathlessness
- G. I. System: Anorexia, acidity, heart burn, palpable spleen and liver.
- Neurological: Dizziness, tingling, numbness, insomnia, dimness of vision, forgetfulness, lack of concentration.
- Reproductive: Amenorrhoea, menorrhagia, abortion.

Table no.5 “CLINICAL FEATURE OF ANEAMIA

SL. No.	Symptoms	Davidson's P.P. of Medicine	Harrison's Internal Medicine	Robin's Pathologic Basis of Diseases
1	Lassitude	+	+	+
2	Fatigue	+	+	+
3	Breathlessness on exertion	+	+	+
4	Headache	+	+	+
5	Palpitation	+	+	-
6	Dizziness	+	+	-
7	Angina	+	-	+
8	Angular Stomatitis	+	+	-
9	Glossitis	+	+	-
10	Pika	+	+	-
11	Tinnitus	+	+	-
12	Dimness of vision	+	-	+
13	Insomnia	+	-	-
14	Paraesthesia in fingers & toes	-	-	-
15	Hypersensitivity to cold	-	+	-
16	Anorexia	-	+	-
17	Nausea	-	+	-
18	Bowel irregularity	-	+	-
19	Abnormal menstruation	-	-	-
20	Amenorrhoea and menorrhoea	-	-	+
21	Loss of Libido	+	-	-
22	Dysphagia	-	-	-
23	Low grade fever	-	-	+
24	Indigestion	-	+	-

**Laboratory investigation:**

Blood examination: Hb%: Below 11.7 g / dL

RBC count: Usually follows Hb%

MCV: 50-80 fL

MCH: 15-26 Pg

MCHC: 24-30 g/dL

Peripheral blood film shows hypochromia, anisocytosis, and poikilocytosis.

Blood biochemistry: Serum iron: Below 50 g/dL

Iron binding capacity: More than 360 g/dL

Percent saturation of transferrin: Below 20%

Serum ferritin: Below 15 g/L

### Stages of Iron Deficiency:

1. Iron store depletion: This is identified using the serum ferritin level and Marrow iron stain.

Ferritin level: Less than 20 g/L

Visible iron stores: 0 to 1+

2. Iron-deficient erythropoiesis:

Serum Ferritin level: Below 15 g/L

Serum iron level: Below 60 g/dL

TIBC: Increases

Percent saturation of transferrin: Less than 20%

Haemoglobin level: 10 to 12 g/dL

3. Iron deficiency anaemia: Serum iron: Below 30 g/dL

TIBC: More than 400 g/dL

Percent saturation of transferrin: Below 10%

Serum ferritin level: Below 15 g/L

Haemoglobin level: Below 10g/dL

### General line of treatment:

- The cause of anaemia should be treated as far as practicable.
- When the haemoglobin level is below 40%, blood transfusion is to be given.
- When the haemoglobin level is more than 40% then iron supplement should be given.

### Causes of Iron Deficiency Anaemia (API Medicine)

1. Nutritional - 50%
2. Malabsorption - 20%
3. Parasitic Infestation - 20%
4. Chronic blood loss - 5 %
5. Others - 5%

#### 1) Nutritional (commonest cause)

- a) Poor dietary Intake
- b) Poor bioavailability of Iron from cereal based diet
- c) Increased requirement of Iron

#### 2) Malabsorption

- a) Any malabsorptive disorder leads to poor iron absorption
- b) Geophagia interferes with Iron absorption and aggravates IDD.

### 3) Parasitic Infestation

Ankylostomiasis

### 4) Chronic blood loss

- a) GIT bleeding Eg: Peptic Ulcer Disease, Bleeding gum, Ulcerative colitis, Crohns disease, Oesophageal varices, MeloryWeiss syndrome.
- b) Anorectal Disease Eg: Bleeding Piles
- c) Genito urinary bleeding Eg: Menorrhagia, Metrorrhagia, Recurrent Haematuria, Haemosideriuria, antipartal post partal, haemorrhage etc.

### 5) Others

Regular blood donation in India where majority of the population has a precarious iron balance. In many cases IDA is multifactor in origin, with dietary deficiency, poor bioavailability and blood loss all contributing to a variant extent.

#### Major etiological factors in iron deficiency:

##### 1) Females in the reproductive period of life

- Menstruation
- Pregnancy
- Lactation
- Pathological blood loss
- Deficient diet

##### 2) Adult males and post-menopausal females

- Pathological blood loss
- Deficient diet

##### 3) Infant and children

- Deficient diet
- Diminished iron stores at birth.

##### 4) Chronic gastrointestinal blood loss due to

- Peptic ulcer
- Haemorrhoids
- Hiatus hernia
- Carcinoma of the stomach
- Carcinoma of the colon
- Chronic aspirin ingestion
- Oesophageal varices
- Ulcerative colitis
- hook worm infestation

## **PATHOGENESIS OF IRON DEFICIENCY ANAEMIA**

Three pathogenic factors are implicated in the Anaemia of iron deficiency. They are as follows.

### **1) Impaired Haemoglobin Synthesis**

When transferrin saturation falls below 16% the supply of iron to the marrow is inadequate to meet the basic requirement for haemoglobin production. Each cell produced contains less haemoglobin resulting in hypochromia. The number of cell divisions and the ultimate erythrocyte size are related to the rate of haemoglobin synthesis. Haemoglobin enters the nucleus and reacts with nucleohistones, thereby causing nuclear inactivation. In iron deficiency, it takes long to reach the critical haemoglobin. Concentration and the generation time is unaffected, hence more cell divisions occur before nuclear inactivation and the resulting cell is microcytic or small in size.

### **2) Generalized defect in Cellular Proliferation**

In iron deficiency, the cellular proliferation is decreased as evidenced by a reduced red blood cell count, reticulocyte count and haemoglobin percentage. The degree of erythroid hyperplasia is low in relation to the degree of anemia.

There is a significant component of ineffective erythropoiesis. A portion of cells in iron deficient subjects are so defective that they are rapidly destroyed.

### **3) Reduced Erythrocyte Survival**

This is the least important factor involved in the pathogenesis of iron deficiency anaemia and is found only when the anaemia is severe. Cross transfusion studies have demonstrated that the shortened survival results from an intra corpuscular defect. There is a significant co-relation between the proportion of morphologically abnormal cells on blood smear and the degree of reduction in red cell survival. The reduced erythrocyte viability is associated with decreased membrane deformability. This abnormality appears to resist it from productive damage to the membrane which in turn may be a consequence of reduced glutathione peroxidase activity.

Several iron proteins are reduced in iron deficiency and some of these proteins may be responsible for certain clinical and pathological manifestations of the disease. Iron is a component of haem proteins like cytochromes, myoglobin, catalase and peroxidase, iron sulphur proteins and metalloflavo proteins are important in oxidation reduction reactions, especially those that take place in mitochondria. Iron is a co factor for certain enzymes and a reduction in tissue enzymes is presumed to be related to the occurrence of epithelial changes in iron deficiency. Impaired resistance to infection in iron deficiency is multifactorial, one important cause being myeloperoxidase deficiency.

### **Stages of Iron Deficiency Anaemia**

Iron deficiency is usually the end result of a long period of negative iron balance and develops in sequential stages. These stages include

#### **1) Stage of Iron Depletion (Pre Latent Iron Deficiency)**

During this stage, the Iron stores in the hepatocytes and the macrophages of the liver, spleen and bone marrow are exhausted, serum ferritin values are reduced. This is because iron stores are mobilized for erythropoiesis.



Iron absorption in the gut is usually increased in an attempt to compensate for the negative iron flow. The RDW (reticulocyte distribution width) is frequently elevated and may be the first indication of a developing iron deficiency in a non anaemic patient.

## 2) Stage of Iron Deficient Erythropoiesis (Latent Iron Deficiency)

In this 2nd stage certain biochemical abnormalities in iron metabolism are usually detected. Serum iron is decreased. TIBC is increased and transferrin saturation is decreased. FEP (free erythrocyte protoporphyrin) levels measured as ZPP (zinc protoporphyrin) are increased. Measurement of ZPP is a sensitive index of this stage of iron deficiency. Other observations include sub normal urinary iron excretion after desferroxamine injection, decreased tissue cytochrome oxidase levels and absence of bone marrow sideroblasts and marrow iron is markedly reduced.

Few microcytes may be detected on the peripheral smear. But MCV (mean corpuscular volume) remains within normal limits. However Hb level is still normal. A large portion of Indian population falls under this group.

## 3) Iron Deficiency Anaemia

In the last stage, the blood haemoglobin falls below the lower limit of normal. The most significant findings is the classic microcytic hypochromic anaemia. Other iron containing enzymes, such as the cytochromes, also reach abnormal levels during this period. Epithelial manifestations of iron deficiency usually represent a very late phase of iron depletion.

As the negative iron balance continues, serum iron level falls further, TIBC increases and transferrin saturation falls below 16%. Serum ferritin is reduced. Iron deficient erythropoiesis ensures with the appearance of erythroid precursors with ragged cytoplasmic margins in the marrow. MCV, MCH, MCHC falls down.

## DIFFERENTIAL DIAGNOSIS OF IDA

In a patient with hypochromic microcytic anaemia, the major diagnostic possibilities are

1. Iron deficiency Anaemia
2. Thalassemia
3. Anaemia of chronic Inflammation
4. Lead poisoning

**Table.No 6**

THE DIFFERENTIAL DIAGNOSIS OF IRON DEFICIENCY ANAEMIA

SL. NO.	Test	Iron Deficiency	Chronic Disorders	Thalassaemia	Siderblastic Anaemia
1)	MCV, MCH, MCHC	Reduced	Low normal to reduced	Very low	Very low (except MCV raised in acquired type)
2)	Serum Iron	Reduced	Reduced	Normal	Raised
3)	TIBC	Raised	Reduced	Normal	Normal
4)	Serum Ferritin	Reduced	Raised	Normal	Raised (complete saturation)
5)	Marrow Iron stores	Absent	Present	Present	Present
6)	Iron in normoblasts	Absent	Absent	Present	Ring sideroblasts
7)	Hb electrophoresis	Normal	Normal	Abnormal	Normal

**Table no. 8****Table. Showing the Similarities between Aneamia and Pandu**

<b>Aneamia general symptoms</b>	<b>Pandu roga Samanya Lakshana</b>
a) Pallor of skin mucous membrane conjunctiva, nails	Pandutha of twak, nakha, ekshana, Anana, Vivarnatha
b) Exhertional dyspnoea	Hataprabha
c) Lassitude, Fatigue Exhaustion	Arohana, Adhwa ayasa Anga sada, Nissaratha,
d) Weakness	Dourbalya, Bala Hani
e) Palpitations, Tachycardia	Hridrava
f) Anorexia, Indigestion	Annavit, Aruchi
g) Tinnitus	Karnakshweda
h) Brittle nails, Koilonychia, nail Cracking	Nakha rukshatha
i) Hypersensitive to cold	Shishira dweshi
j) Headache	Shiro ruk,
k) Nausea	Hrillasa, Praseka
l) Bowel irregularity	Vit bandha (vit shosha)
m) Insomnia	Anidra
n) Low grade fever	Jwara
o) Aches and pains in Various parts of the Body	Gatra mardhana peedanadi
p) Oedema	pindikodwestana, kati, uru, padaruk,gatrashoola,Shoona akshi,koota Shopha

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2. A.K. Khanda, 5th varga
3. S.S. Ut. 44/4
4. A.H. Ni 13/3-4
5. Dl on S.S. Ut 44/3
6. M.N Madhu 8

**Paryaya**

7. S.S. Ut. 44/6
8. Dl on S.S. Ut 44/6

**Nidana**

9. C.S. Ch 16/3
10. C.S. Su 7/14
11. C.S. Ch 16/18
12. C.S. Ch 16/7-8
13. C.S. Si 6/69
14. C.S. Su 3/75
15. C.S. Ch 9/16
16. C.S. Su 13/21
17. C.S. Su 7/14
18. C.S. Si 12/30
19. S.S.Sa 6/27
20. S.S. Ni 11/17
21. S.S Ch 2/51
22. C.S. Ch 2/27
23. S.S. Sh 2/20
24. H.S. 3/9/19
25. Sh.S Ut 40/177
26. C.S. Ch 3/337
27. S.S. Ut 40/177
28. C.S. Ch 14
29. A.H. Ni 15/56
30. C.S. Ni 2/7
31. S.S. Sh 2/19
32. C.S Ch 30/13
33. A.H. Ni 12/26
34. S.S Ni 7
35. S.S Ut 24/8
36. S.S Ut 41
37. S.S. Ut 12/9
38. C.S Su 17/69
39. C.S Ch 30/161
40. C.S. Ch 30
41. S.S Ni 6
42. A.H Ni 13
43. C.S Ch 23
44. C.S Su 28/9
45. S.S.Sa 9

46. K.S. Ch Balagraha

47. S.S. Su 25

48. A.S. Ut 41

49. K.S. Kila 9

50. S.S Ka 7/11

51. S.S Ni 8/14

**Samprapthi**

52. C.S Ch 16/4

53. C.S. Ch 16/10

54. C.S. Ch 16

55. M.N 8

**Pandu bheda**

56. C.S. Ch 16/33

**Roopa**

57. A.H. Ni 13/9

58. A.H. Ni 13/10

59. A.H. Ni 13/11

60. A.H. Ni 13/12

61. A.H. Ni 13/13

**Sadhya Asadhyatha**

62. C.S. Ch 16/31

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