Contents

| 1. | Iron-deficiency Anemia in Pregnancy |
|----|--|
| 2. | Megaloblastic Anemia in Pregnancy |
| 3. | Sickle Cell Disease in Pregnancy15Sumitra Yadav•Types of Sickle Cell Disease15••Pathophysiology15•Management of Sickle Cell Crisis During Antenatal Period18••Blood Transfusion18•Management During Labor18•Contraceptives18•Future Treatment19 |
| 4. | Thalassemia in Pregnancy |
| 5. | Cardiac Diseases in Pregnancy |
| 6. | Hypertensive Disorders in Pregnancy |
| 7. | Gestational Diabetes52Pralhad Kushtagi• Classification of Hyperglycemia in Pregnancy 52• Definition 52• Prevalence 52• Risk Factors 53• Pathophysiology 53• Effect of Gestational Diabetes 54• Screening and Diagnosis 56• Management 57• Postpartum Care 60• Studies of Importance 60 |

xviii Contents

| 8. | Thyroid Disorders in Pregnancy. Srinivas Krishna Jois History 63 • Physiology 63 • Immunomodulation in Pregnancy 63 Incidence 64 • Screening 64 • Thyroid Disorders 65 Neonatal Issues 67 • Medical Termination of Pregnancy 68 Thyroid Nodules/Cancer 68 • Postpartum Thyroiditis (PPT) 68 Obstetric Management 69 | 63 |
|-----|--|-----|
| 9. | Renal Disease in Pregnancy | 71 |
| 10. | Epilepsy in Pregnancy | 76 |
| 11. | Acquired Coagulation Disorders in Pregnancy Pankaj Desai, N Palaniappan Disseminated Intravascular Coagulation 84 • Thrombocytopenia 86 Acquired Hemophilia 89 • Drugs Producing Thrombocytopenia 89 | 84 |
| 12. | Antiphospholipid Antibody Syndrome S Habeebullah Prevalence 91 • Classification of Antiphospholipid Syndrome 91 Pathogenesis 91 • Pregnancy Loss 92 • Clinical Manifestations 92 Diagnosis 92 • Poor Prognostic Factors 92 Pregnancy and Labor Management of Antiphospholipid Syndrome 94 | 91 |
| 13. | Human Immunodeficiency Virus in Pregnancy Ambarisha Bhandiwad, Achanta Vivekanand Etiology and Pathogenesis 95 • HIV and Pregnancy 96 | 95 |
| 14. | Liver Diseases in Pregnancy Shikha Seth, Ritu Sharma Liver in Pregnancy 100 • Viral Hepatitis 100 Cirrhosis and Portal Hypertension 104 • Symptoms 104 Gall Stones/Cholelithiasis and Pregnancy 105 • Liver Transplant 105 Acute Fatty Liver of Pregnancy 106 • Hepatic Adenoma and Hemangioma 106 Alcohol and Pregnancy 106 • Hyperemesis Gravidarum 106 Hellp Syndrome 107 • Common Liver Disease Presentations 107 | 100 |
| 15. | Febrile Illness in Pregnancy Deepa Lokwani Masand Malaria 110 • Dengue 115 • H1N1 (Swine Flu) 119 Varicella/Chickenpox 125 • Typhoid 128 | 110 |

| C | |
|-----------|--|
| (ontents | |
| COnternes | |

| 16. | Skin Diseases in Pregnancy | 134 |
|-----|---|-------|
| | Physiological Skin Changes During Pregnancy 134 Pemphigoid Gestationis (Herpes Gestationis/Gestational Pemphigoid Dermatitis Multiformis Gestationis) 136 Intrahepatic Cholestasis of Pregnancy 138 • Polymorphous Eruption of Pregnancy 138 Atopic Eruption of Pregnancy 139 | |
| 17. | TORCH in Pregnancy BS Jodha Incidence 141 • Fetal Implications of TORCH 141 • Screening 142 Prenatal Diagnosis 142 • Management Protocols of TORCH 143 Toxoplasma Infection in Pregnancy 143 • Rubella (German Measles) in Pregnancy 145 Cytomegalovirus in Pregnancy 146 • Genital Herpes in Pregnancy 147 Recent Advances in TORCH Management 148 | 141 |
| 18. | Preterm Labor | . 150 |
| 19. | Prelabor Rupture of the Membranes RP Rawat Pathophysiology of Fetal Membranes 158 • Etiology 158 Diagnosis of PROM 159 • Management of PROM 160 Fetal Complications of PROM 161 • PROM Following Amniocentesis 162 Preterm PROM and Future Pregnancies 162 | 158 |
| 20. | Cervical Insufficiency Hemant Deshpande, Manjula S Patil Incidence 164 • Definition 164 • Etiopathogenesis 164 Diagnosis 166 • Management 168 | 164 |
| 21. | Post-Term Pregnancy (Prolonged Pregnancy)Hemant Deshpande, Sonali Deshpande• Etiology and Associated Risk Factors175• Diagnosis176• Complications177• Recurrence Risk177• Management177 | 175 |
| 22. | Placenta Previa Ashok Kumar Incidence 183 • Classification 183 • Etiology 183 • Risk Factors 184 • Clinical Features 184 • Management 184 • Complications of Placenta Previa 186 • Prognosis 187 | 183 |
| 23. | Abruptio Placentae | 189 |

| хх | Contents |
|----|----------|

| 24. | Rh Alloimmunization Nitin S Kshirsagar Terminology 197 • Rhesus Blood Group System 197 Pathogenesis of Maternal Alloimmunization 198 • Transplacental Hemorrhage 198 Maternal Response 198 • Sensitization Phenomenon 198 • Fetal Response 198 Degrees of Alloimmunization 198 • Management 199 Middle Cerebral Artery Peak Systolic Flow Velocity 203 • Fetal Blood Sampling 203 Management Protocol for Rh Alloimmunized Patient 204 | 197 |
|-----|--|-----|
| 25. | Intrauterine Growth Restriction | 206 |
| 26. | Intrauterine Fetal Demise. SManikyarao Definition 215 • Global Burden 215 • Incidence 215 Etiology 216 • Clinical Features 216 • Investigations 216 Dead-Born/Stillborn Examination 219 • Management 219 Recommendations for Intrapartum Antimicrobial Therapy 221 Options for Suppression of Lactation 221 Psychological and Social Aspects of Intrauterine Fetal Demise 222 Complications 222 • Legal Aspects 222 • Strategies for Prevention of Stillbirth 222 Future Research 223 | 215 |
| 27. | Multifetal Gestation Vidya Thobbi Assisted Reproduction Technology 225 • Maternal Age, Race, Parity, and Ethnicity 225 Classification 225 • Etiology of Multiple Births 227 Amnionicity and its Clinical Importance 228 • Maternal Adaptation 229 Complications 229 • Fetal Complications Unique in Multiple Gestations 230 Monoamniotic Twins 235 • Congenital Abnormality 235 • Conjoined Twins 235 Diagnosis of Multiple Gestations 236 • Antepartum Management 237 Genetic Counseling 239 • Antenatal Care 239 Preterm Premature Rupture of Membranes 240 • Intrapartum Management of Twins 241 Routes of Delivery 242 • Interlocking Twins 243 Management of Gestation with High Fetal Number 243 Key Pre- and Postnatal Events to be Offered in Pregnancy 244 Preventive Interventions for Multiple Pregnancies 245 • Multifetal Pregnancy Reduction 245 Selective Termination 245 | 225 |
| 28. | Induction of Labor | 248 |

| Contents | xxi |
|----------|-----|
| | |

| 29. | Pregnancy after Previous Cesarean Section |
|-----|---|
| | Benefits of Vaginal Delivery (VBAC) Over Repeat Cesarean Section 257 Fetal Benefits of Vaginal Birth after Cesarean 258 Antenatal Preparation for Trial of Labor after Cesarean 258 Criteria for Selection for Vaginal Birth after Cesarean 258 Contraindications to Vaginal Birth after Cesarean 258 Maternal Risks of Vaginal Birth after Cesarean 259 • Fetal Risks 260 Predictors of Vaginal Birth after Cesarean Success 260 • Management 262 Assessment of Progress in Labor and Management of Failure to Progress 263 Important 264 • Special Points 265 |
| 30. | Ectopic Pregnancy |
| 31. | Hyperemesis Gravidarum |
| | Shubharanjan Smantarai Epidemiology 276 • Etiology 276 • Risk Factors 277 Clinical Presentation and Diagnosis 277 • Differential Diagnosis 278 Maternal Effects of Nausea and Vomiting of Pregnancy 278 Fetal Effects of Nausea and Vomiting of Pregnancy 279 Management 280 • Need of Hospitalization 284 Termination of Pregnancy 284 |
| 32. | Gestational Trophoblastic Disease |
| | Priya Ballal Molar Pregnancy 287 Nonmolar Lesions 289 Incidence of Gestational Trophoblastic Disease 290 Risk Factors 290 Management of Gestational Trophoblastic Disease 291 Complications 292 Prophylactic Chemotherapy 293 Postmolar Surveillance 293 Staging and Evaluation of Gestational Trophoblastic Disease 294 Evaluation 294 Classification and Staging of Malignant Gestational Trophoblastic Disease 296 Chemotherapy for Low-risk Gestational Trophoblastic Disease 296 High-risk Gestational Trophoblastic Disease 296 Management of Metastatic Sites 299 |
| 33. | Cesarean Section |
| | History 302 Clinical Anatomy 302 Indications for Cesarean Sections 303 Preoperative Preparation for Cesarean Section 304 Surgical Technique 304 Anesthesia for Cesarean Section 306 Complications 307 Strategies to Decrease Lower Segment Cesarean Section Rate 307 VBAC Versus ERCS 307 |
| 34. | Rupture of the Uterus |
| | Asnok Kumar Benera Incidence 310 • Classification 310 • Risk Factors and Etiology 310 Mechanism of Uterine Rupture 311 • Morbid Anatomy 311 Clinical Features and Diagnosis 312 • Treatment 312 • Outcome 313 |

_ _

| xxii | Contents |
|------|----------|

| 35. | Contracted Pelvis and Cephalopelvic Disproportion | | |
|-----|---|-----|--|
| 36. | Nonimmune Hydrops FetalisShipra Kunwar• Causes of Nonimmune Hydrops Fetalis323• Ultrasound Diagnostic Criteria324• Management324• Investigations326• Prognosis326• Risk of Recurrence326 | 323 | |
| 37. | Amniotic Fluid Embolism Deepa Lokwani Masand Etiopathogenesis 328 • Clinical Features 329 • Differential Diagnosis 331 Management 331 • Cardiopulmonary Resuscitation in Pregnancy 335 • Prognosis 335 | 328 | |
| 38. | Immunization in PregnancyChandrakant Madkar, Madhukar Shinde• Vaccine 336 • Immunization Recommendations 337• Vaccination During Pregnancy 337 • CDC Guidelines 338• Government of India Programs 338 • Role of WHO in Immunization Program 338 | 336 | |
| 39. | Obstetric Analgesia and Anesthesia in High-risk Pregnancies | 340 | |
| 40. | Cord Around NeckPriyanka A Dahiya• Classification 350• Etiopathogenesis 350• Management 351 | 350 | |
| 41. | Trisomy 21 JB Sharma • Rationale for Screening 354 | 354 | |
| 42. | Amniotic Fluid Abnormalities | 359 | |
| 43. | Obesity and PregnancySrinivas Krishna Jois• History 370• Definition and Classification 370• Epidemiology of Obesity in Women and in Pregnancy 370• Factors 372• Diagnosis 372• Care During Pregnancy 372• Contraception 375• Bariatric Surgery 375• Observations at Vani Vilas Hospital, Bangalore (Unpublished) 375 | 370 | |
| 44. | Fetal Distress in Pregnancy | 378 | |
| 45. | Corticosteroids for Fetal Lung Maturity Janki Pandya, Munjal Pandya Corticosteroids for Fetal Lung Maturity 383 • Mechanism 383 Other Fetal Effects 383 • Maternal Effects 383 • Timing of Administration 383 Premature Rupture of Membranes 384 • Gestational Diabetes Mellitus 384 Late Preterm Birth 384 • Multifetal Gestation 384 • Elective Cesarean Section 384 | 383 | |

Contents **xxiii**

| | Hypertensive Disorders of Brognancy 205 a Fetal Crowth Pastriction 205 | |
|-------|--|-----|
| | Hypertensive Disorders of Pregnancy 385 • Fetal Growth Restriction 385 Patient Living with HIV/AIDS 385 • Pregnancy with Hepatitis B Infection 385 Dose 385 • Duration of Benefit 385 • Timing of Effects 385 Repeat Courses 386 • Betamethasone Versus Dexamethasone 386 Other Routes for Antenatal Corticosteroid Administration 386 • Potential Complications 387 | |
| 46. | Progesterone in Pregnancy Nilesh Balkawde Functions of Progesterone in Obstetrics 389 Progesterone and Pregnancy: Physiology 389 Progesterone in Recurrent Pregnancy Loss 390 Progesterones Used in Luteal Phase Defect and Preterm Birth 392 Mechanism of Action of Progesterone 392 Guidelines for use of Progesterone in Threatened Abortion 393 | 389 |
| 47. | Varicose Veins in Pregnancy Rajendra P Shitole Incidence 395 • Etiopathology 395 • Clinical Evaluation 395 • Diagnosis 396 Treatment 396 • Development of Varicose Vein Postpregnancy 397 Varicose Vein Clinical Spectrum 397 | 395 |
| 48. | Pregnancy after Organ Transplants | 399 |
| 49. | Human Milk Banking—Need of the Time Shailaja Mane Evidence in Literature 404 • Human Milk Banking 404 | 404 |
| 50. | Oncofertility | 410 |
| 51. | Partogram | 416 |
| 52. | Nonstress TestPrakash Mehta• History 420 • Instrument 420 • Physiology 421 • Result 424• Nonstress Test in Special Situations425 • Nonstress Test Versus Other Surveillance Tests426• Litigation and Nonstress Test427 | 420 |
| 53. | Sildenafil Citrate in Obstetrics and Gynecology. Suyajna Joshi D, Sanjana Kumar History 431 • Mechanism of Action 431 • Indications 431 Well-Established Uses 431 • Indications Under Trial 431 • Common Adverse Effects 431 Contraindications 431 • Sildenafil in Obstetrics 431 • Sildenafil Citrate in Gynecology 437 | 431 |
| Index | | 441 |

Iron-deficiency Anemia in Pregnancy

Suyajna Joshi D

INTRODUCTION

CHAPTER

Anemia is the most common hematological disorder in pregnancy which has significant maternal as well as perinatal morbidity and mortality. Anemia among pregnant women is a serious global health concern. According to the World Health Organization (WHO) report, about million pregnant women suffer from anemia worldwide, of which 0.8 million women are severely anemic. An estimate by the WHO attributes about 591,000 maternal deaths globally to iron-deficiency anemia (IDA), directly or indirectly. Majority of the cases of anemia in pregnancy are due to nutritional deficiency of which >50% cases are attributable to IDA followed by folate and vitamin B₁₂ deficiency. Other causes such as hemoglobinopathies, autoimmune hemolytic anemia, aplastic anemia, chronic infections, rheumatoid arthritis, and chronic renal disease, though less commonly found, should arouse suspicion in the clinician based on symptomatology for the better management of the case.

DEFINITION

Anemia is defined as a decrease in the oxygen-carrying capacity of the blood due to a decrease in the hemoglobin concentration or due to a reduced number of red blood cells (RBCs).

CLASSIFICATION OF ANEMIA

Anemia is classified based on etiology (**Box 1**), severity (**Table 1**), and trimester (**Table 2**).

IRON METABOLISM

Iron is an essential component of every cell in the body. Although best known for its critical role in the transport and storage of oxygen (in hemoglobin and myoglobin, respectively), within a large variety of enzymes, iron also acts as a carrier for electrons, a catalyst for oxygenation and hydroxylation, and is necessary for cellular growth and proliferation. Iron supplements are widely administered to treat IDA, particularly in chronic diseases such as kidney disease, heart failure, or inflammatory bowel disease. Without a sufficient supply of iron, hemoglobin cannot be synthesized and the number of erythrocytes in the blood cannot be maintained at an adequate level.

Iron usually exists in the ferrous (Fe^{2+}) or ferric (Fe^{3+}) state, but since Fe^{2+} is readily oxidized to Fe^{3+} , which in

BOX 1: Classification of anemia based on etiology.

- Anemia with nutritional deficiency
 - Iron-deficiency
 - Folic acid deficiency
 - Vitamin B₁₂ deficiency
 - Combined deficiency
- Anemia associated with decreased production of RBC
 - Bone marrow disorders
 - Hypothyroidism
 - Chronic renal pathology
 - Bone marrow suppression
- Anemia associated with increased RBCs' destruction
 - Hemolytic anemias
 - Inherited and acquired
 - Sickle cell anemiaThalassemia
 - Hereditary spherocytosis
 - Autoimmune hemolytic anemia
 - Hemolytic uremic syndrome
 - Thrombotic thrombocytopenic purpura
 - Malaria
- Anemia due to blood loss
 - Abnormal uterine bleeding
 - GIT bleeding
- Obstetric hemorrhage

| TABLE 1: Classification of anemia according to severity. | | | |
|---|------------------|------------------|--|
| Category of anemia | WHO (Hb in g/dL) | ICMR (Hb in g/d) | |
| Mild | 9–10.9 | 10–11 | |
| Moderate | 7–8.9 | 7–10 | |
| Severe | 4–6.9 | 4–7 | |
| Very severe | <4 | <4 | |

(Hb: hemoglobin; ICMR: Indian Council of Medical Research; WHO: World Health Organization)

| TABLE 2: Classification of anemia according to trimester. | | | | |
|--|-------------------|--|--|--|
| Pregnancy state | Hemoglobin (g/dL) | | | |
| First trimester | <11 | | | |
| Second trimester | <10.5 | | | |
| Third trimester | <11 | | | |

neutral aqueous solutions rapidly hydrolyzes to insoluble iron(III)—hydroxides, iron is transported and stored bound to proteins. Effective binding of iron is essential not only to ensure that it is available where and when required, but also because Fe^{2+} can catalyze the formation of reactive oxygen species, which cause oxidative stress, damaging cellular constituents.

Total body Fe in man is 4–5 g. Daily losses, e.g., in epithelial desquamation from the gastrointestinal tract or skin, are small. Excretion in urine, bile, and sweat is negligible. The normal daily Fe requirement is thus only 1 mg, increasing with physiological need, as in growth, pregnancy, and blood loss. An additional 1,000 mg Fe is required in pregnancy and 0.5 mg Fe/mL in case of blood loss.

Three key proteins regulate the transport and storage of iron (Fig. 1). Transferrin transports iron in the plasma and the extracellular fluid. The transferrin receptor, expressed by cells that require iron and present in their membranes, binds the transferrin di-iron complex and internalizes it into the cell. Ferritin is an iron-storage protein that sequesters iron keeping it in a readily available form. About 60% of iron is found in the erythrocytes within hemoglobin, the oxygen transport protein. The remainder is found in myoglobin in the muscles, in a variety of different enzymes ("heme" and "non-heme"), and in storage form. Most stored iron is in the form of ferritin, found in the liver, bone marrow, spleen, and muscles. Serum iron (i.e., iron bound to transferrin) represents only a very small proportion of the total body iron (<0.2%). Moreover, the relationship between physiological iron compartments is highly dynamic: Erythrocytes are broken down in the liver and the spleen and new red blood

cells are produced in the bone marrow. The total serum iron pool is approximately 4 mg, but the normal daily turnover is not >30 mg, such that minor changes in the serum level due to exogenous iron administration are clinically meaningless. In this setting, conventional measurements of serum iron concentration provide no relevant information about the availability of functional iron for physiological processes, and other evaluation strategies must be pursued. A schematic representation of iron metabolism is shown in **Figure 1**.

IRON-DEFICIENCY ANEMIA

Iron is essential for normal hemoglobin (Hb) synthesis to maintain oxygen transport as well as necessary for metabolism and synthesis of DNA and enzymatic processes. Iron stores may be measured using several indices, although serum ferritin and transferrin saturation are the most common.

Iron-deficiency anemia is defined as a low Hb concentration in combination with iron-deficiency and is characterized by a defect in Hb synthesis, resulting in abnormally small (microcytic) red blood cells with a decreased Hb content (hypochromic), resulting in reduced capacity of the blood to deliver oxygen. The prevalence of Fe deficiency is much higher. Without adequate Fe supplementation, ferritin falls to subnormal levels toward the end of pregnancy, even in the industrialized nations.

Iron-deficiency anemia evolves through three distinct stages **(Table 3)**. Depletion of storage iron occurs in the first phase (stage I), where total body iron is decreased but red cell indices and hemoglobin (Hb) synthesis remain unchanged. Both these indices change when the supply of



Fig. 1: Transport and storage of iron.

| TABLE 3: Stages in the development of iron-deficiency. | | | | | |
|--|-----------------|-----------------|-------------------------------|--------------------------|--|
| Parameters | Normal | Iron depletion | Iron-deficient erythropoiesis | IDA | |
| Hemoglobin | 150 g/L (15 g%) | 130 g/L (13 g%) | 100 g/L (10 g%) | 50 g/L (5 gm%) | |
| MCV | Ν | \downarrow | \downarrow | $\downarrow\downarrow$ | |
| MCHC | Ν | Ν | \downarrow | $\downarrow\downarrow$ | |
| Iron stores | Present | Reduced | Absent | Absent | |
| Serum Fe/TIBC (µg/L) | 1,000/3,000 | 75/3,000 | 500/4,500 | 250/6,000 | |
| Serum ferritin (µg/L) | 100 | 20 | 10 | <10 | |
| RBCs | Normal | Normal | Normal | Hypochromic microcytosis | |

(IDA: iron-deficiency anemia; MCHC: mean corpuscular hemoglobin concentration; MCV: mean corpuscular volume; RBC: red blood cells; TIBC: total iron-binding capacity)

iron to bone marrow is reduced (stage II or iron-deficient erythropoiesis). Stage III, eventually iron-deficiency anemia develops due to insufficient supply of iron to sustain a normal Hb concentration.

Signs and Symptoms of Iron-deficiency Anemia

Although the Hb test is recommended at the first antenatal visit, examination for signs of pallor of the palpebral conjunctiva, tongue, nail beds, and palm should be regularly done. Some iron-deficient patients, with or without clinical signs of anemia, may have alopecia, atrophy of lingual papillae, or dry mouth due to reduced salivation.

The symptoms specific to iron-deficiency anemia include the syndrome of Plummer–Vinson or Paterson– Kelly (dysphagia with esophageal membrane and atrophic glossitis), gastric atrophy, stomatitis due to rapidly turning over of epithelial cells, spoon-shaped nails (koilonychias), and pallor. These changes were caused by the reduction of iron-containing enzymes in epithelial and gastrointestinal (GI) tracts. The restless leg syndrome might be striking neurological squeal prevalent in pregnancy. Pica, the eating disorder in which there is an appealing desire to lick or eat nonfood items such as gypsum, chalk, soil, ice (pagophagia), or paper, is prevalent in pregnant women. Pagophagia (intense desire to eat ice) is quite specific to Iron-deficiency and responds quickly to treatment.

Diagnosis of Iron-deficiency Anemia

There are four groups of tests which are available for detection of IDA:

- 1. Hb, mean corpuscular volume (MCV), red cell distribution width (RDW), reticulocyte Hb content, % hypochromic cells, red cell size factor, and low Hb density
- 2. Direct measurements of iron stores through assessment of serum iron, total iron-binding capacity (TIBC), % saturation, serum ferritin, bone marrow biopsy iron
- 3. Assessment of iron heme form through assessment of free erythrocyte protoporphyrin (EPP)

4. Assessment of iron uptake by measuring the soluble serum transferrin receptor (sTfR), soluble transferrin receptor-log[ferritin] (sTfR-F) index, and zinc protoporphyrin (ZPP)

A primary step in the diagnosis of IDA is to consider the complete blood count, including Hb, MCV, mean corpuscular hemoglobin (MCH), and mean corpuscular hemoglobin concentration (MCHC), which is simple, inexpensive, rapid to perform, and help early prediction of IDA.

Changes in Hb concentration and hematocrit occur only in later stages; both these tests are indicators of Irondeficiency. Low Hb with a reduced MCV is usually the initial finding on a routine complete blood count (CBC). The severity of anemia is based on the patient's Hb/hematocrit level.

Altitude above sea level and smoking are the known modifiers of Hb. Currently, the Hb cut-off according to trimester has not been defined by the WHO, but it should be recognized that the Hb falls about 0.5 g/dL in the second trimester. Hb concentration is the most common hematological estimation. There is a strong correlation between Hb concentration and serum ferritin levels. The generally recommended methods of Hb estimation are cyanomethemoglobin and HemoCue@ system. RDW has better sensitivity than MCV for diagnosis of IDA. Falling MCV accompanied by a rising RDW should alert the clinician to the presence of possible IDA which is then confirmed by marked RDW increase occurring early after initiation of therapy.

Peripheral smear shows the presence of microcytic hypochromic red cells and typical "photo pencil cells" being indicative of IDA. Other than IDA, the conditions which cause a microcytic blood picture are anemia of chronic disorder, beta-thalassemia, and sideroblastic anemia.

Of all the available indices, the Meltzer index (MCV/ RBC) has been shown as the most reliable index with high sensitivity.