

Iron Deficiency and Iron Metabolism

Jeong-Ok Lee

*Department of Internal Medicine, Seoul National University Bundang Hospital,
Seoul National University College of Medicine, Korea*

Iron is an essential micronutrient required for erythropoiesis, mitochondrial energy production, and cellular proliferation. Because both deficiency and excess are harmful, iron balance is maintained through tightly coordinated systemic and cellular mechanisms. Iron metabolism is now understood as an integrated, multi-organ system encompassing intestinal absorption, macrophage recycling of senescent erythrocytes, hepatic storage, and hepcidin-mediated systemic regulation. Hepcidin, a liver-derived peptide hormone, serves as the central regulator of systemic iron homeostasis by controlling ferroportin-mediated iron export. Its expression reflects body iron status, erythropoietic activity, and inflammatory signaling, thereby linking iron metabolism to immune responses and chronic disease. Suppressed hepcidin promotes iron mobilization during deficiency or increased erythropoietic demand, whereas inflammation-induced hepcidin elevation leads to iron sequestration and iron-restricted erythropoiesis. Iron deficiency remains the most common nutritional disorder worldwide and a leading cause of anemia. It occurs along a spectrum ranging from isolated depletion of iron stores to overt iron-deficiency anemia and may also present as functional iron deficiency in inflammatory states. Importantly, iron deficiency affects multiple organ systems, contributing to fatigue, reduced exercise tolerance, cognitive impairment, and immune dysfunction, even in the absence of anemia. Assessment of iron status is often challenging, particularly in patients with chronic inflammatory conditions, malignancy, heart failure, or kidney disease. While serum ferritin and transferrin saturation remain cornerstone biomarkers, individualized interpretation is required in complex clinical settings. Treatment strategies have evolved toward personalized iron replacement. Oral iron is appropriate for uncomplicated deficiency, whereas intravenous formulations enable rapid and effective repletion in patients with malabsorption, intolerance, or functional iron deficiency. This lecture will review key mechanisms of iron metabolism and the clinical spectrum of iron deficiency, integrating molecular insights with practical diagnostic and therapeutic approaches to optimize patient care in current clinical hematology practice.