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Evaluation of Electrophysiological Alterations in Iron Deficiency Anemia: A Comparative Study of Nerve Conduction and Electrocardiographic Parameters in Young Adults

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ABSTRACT

Background: Iron deficiency anemia (IDA) is the most common nutritional disorder worldwide, affecting various physiological systems beyond hematologic function. Emerging evidence suggests that IDA impacts neurocardiac physiology, including alterations in electrocardiographic (ECG) patterns and nerve conduction studies (NCS), even before clinical symptoms of neuropathy appear. This study aims to evaluate and compare electrophysiological parameters among individuals with different grades of IDA and healthy controls.

Methodology: This cross-sectional observational study was conducted at Government Medical College, Kannauj, involving 110 participants aged 20–40 years, divided into two groups: 55 cases with clinically and biochemically confirmed IDA and 55 age- and sex-matched healthy controls. Hematological indices (Hb, MCV, MCH, MCHC, ferritin), ECG (heart rate, T wave, QRS duration), and nerve conduction parameters (latency, amplitude, conduction velocity of median and ulnar nerves) were assessed. Statistical analyses included t-tests, ANOVA, Chi-square, and correlation tests with a significance level of $p < 0.05$.

Results: Hemoglobin and serum ferritin levels significantly decreased across anemia grades ($p < 0.001$). Heart rate increased with anemia severity ($p < 0.001$), while other ECG parameters showed no significant difference. Nerve conduction velocities (motor and sensory) were significantly reduced in IDA patients, particularly in moderate and severe cases. Latency was prolonged, and amplitudes were reduced in both median and ulnar nerves. Strong positive correlations were found between hemoglobin and nerve conduction velocities (r = 0.36–0.44), and serum ferritin showed moderate correlations with conduction parameters. Nerve conduction abnormalities were significantly more frequent in severe anemia ($p < 0.001$).

Conclusion: Iron deficiency anemia is associated with significant alterations in nerve conduction parameters and elevated heart rate, indicating subclinical neurocardiac involvement. These electrophysiological changes correlate with anemia severity and are potentially reversible with timely treatment. Routine evaluation of ECG and NCS may aid early detection and management of complications in IDA.

Keywords: *Iron deficiency anemia, nerve conduction study, electrocardiogram, hemoglobin, serum ferritin, electrophysiology, peripheral neuropathy, cardiac function*

1. INTRODUCTION

Anemia is a prevalent global health condition defined by a reduction in red blood cell count or hemoglobin concentration, which compromises the oxygen-carrying capacity of blood and results in tissue hypoxia. It affects approximately 25% of the world's population, with the highest burden observed in low- and middle-income countries, particularly among women, children under five, and the elderly[1]. According to the World Health Organization, 1.8 billion people are anemic, including 40% of children aged 6–59 months and 30% of women of reproductive age, with sub-Saharan Africa and Asia being the most affected regions[2]. Anemia contributes significantly to cognitive delays, reduced work capacity, increased maternal and child morbidity, and overall mortality. The Global Burden of Disease Study (2019) estimated an age-standardized prevalence of 23,176.2 per 100,000 people, with a modest 13.4% reduction over three decades. In regions like India, anemia is

multifactorial in origin, often driven by nutritional deficiencies, hemoglobinopathies, infections, and socio-economic barriers to healthcare[1, 3].

Iron deficiency is the most common underlying cause of anemia globally, typically resulting from poor dietary intake, malabsorption, or chronic blood loss, including menstruation and gastrointestinal bleeding[4, 5]. Other nutritional deficiencies, such as vitamin B12 and folic acid, can cause megaloblastic anemia, characterized by the presence of large, immature red blood cells[5].

Iron plays a critical role in neurodevelopment, especially during infancy and childhood. Its deficiency disrupts key processes such as myelination, neurotransmitter synthesis, synaptic development, and gene regulation, leading to long-term deficits in cognitive, motor, and socioemotional functioning[6]. In adults, IDA has been linked to changes in brain metabolism, increased blood-brain barrier permeability, and impaired cognitive functions like memory and spatial processing, all of which are associated with hemoglobin levels[7]. A major neurochemical consequence of iron deficiency is altered dopamine metabolism, which can impair learning, attention, and memory. School-aged children with IDA often show poor performance in attention and working memory tasks, likely due to dopamine pathway disruption and changes in neurotransmitter levels[8, 9].

Cardiovascular involvement in anemia is also well-documented. Electrocardiographic (ECG) changes are common, particularly in hemoglobinopathies like sickle cell anemia (SCA), and include left or right ventricular hypertrophy, QTc prolongation, and sinus tachycardia—changes that worsen with anemia severity and improve upon correction[10, 11]. Severe anemia may cause ST segment depression, T wave inversion, and myocardial ischemia due to reduced oxygen supply, prompting compensatory increases in cardiac output and sometimes ventricular dilation[11, 12]. These effects are generally reversible with appropriate treatment.

In addition to cardiac implications, electrophysiological techniques such as electroencephalography (EEG), surface electromyography (EMG), and nerve conduction studies (NCS) are invaluable in detecting neurophysiological alterations associated with anemia. These methods not only facilitate early diagnosis of neurological disorders like epilepsy, stroke, and neuropathies, but also benefit from recent machine learning advances—including convolutional neural networks (CNN), support vector machines (SVM), BBE-XL, and ML-ANN models—that improve diagnostic accuracy[13-16]. Such innovations are also valuable in psychiatric and functional neurological disorders, aiding in early intervention.

Given this background, the current study was designed to evaluate and compare electrophysiological changes—including nerve conduction abnormalities and ECG findings—across varying grades of iron deficiency anemia. The goal is to elucidate the systemic neurocardiac impact of anemia and its correlation with anemia severity.

2. METHODOLOGY

Study Design and Setting:

This cross-sectional observational study was conducted in the Department of Physiology at Dr. BRBA Government Medical College, Kannauj, Uttar Pradesh, over a period of 1.5 years. The study aimed to evaluate electrophysiological alterations in patients with iron deficiency anemia (IDA) compared to healthy, age- and sex-matched controls. Institutional Ethics Committee approval was obtained prior to commencement.

Study Population and Sample Size:

A total of 110 participants (aged 20–40 years) were enrolled, including 55 IDA cases and 55 healthy controls. Sample size was calculated using the formula $n = 4pq/L^2$, considering a 52% estimated prevalence of anemia, 10% allowable error, and a 5% non-response rate. Participants were recruited from hospital outpatient and inpatient departments. Cases were diagnosed based on hemoglobin levels (<13 g/dL in males, <12 g/dL in females) and serum ferritin (<15 ng/mL). Controls had normal hematological parameters and no chronic illnesses.

Inclusion and Exclusion Criteria:

Included were individuals aged 20–40 years with confirmed IDA (cases) or normal hematological status (controls). Exclusion criteria encompassed endocrine, neurological, renal, hepatic, or cardiac disorders; pregnancy; recent childbirth; neurotoxic drug use; genetic hemoglobinopathies; and recent iron supplementation.

Data Collection and Investigations:

After informed consent, all participants underwent clinical evaluation, hematological tests, electrocardiography (ECG), and nerve conduction studies (NCS). Blood samples were analyzed for hemoglobin, MCV, MCH, MCHC, and serum ferritin. ECG (12-lead) was performed to assess heart rate, ST segment, T wave morphology, and QRS duration. NCS (median and ulnar nerves) assessed motor and sensory amplitudes, latencies, and conduction velocities using standard protocols.

Grading of Anemia:

Anemia was classified as mild, moderate or severe according to NFHS-5 and WHO hemoglobin cut-offs:

- **Males:** Mild (12–12.9 g/dL), Moderate (9–11.9 g/dL), Severe (<9 g/dL)
- **Females:** Mild (10–11.9 g/dL), Moderate (7–9.9 g/dL), Severe (<7 g/dL)

Statistical Analysis: Data were analyzed using IBM SPSS. Descriptive statistics (mean ± SD, percentages), independent *t*-tests, ANOVA, and Chi-square tests were used for comparisons. Correlation between hematological and electrophysiological parameters was assessed using Pearson's or Spearman's coefficients. A *p*-value < 0.05 was considered statistically significant.

3. RESULTS

Table 1: Composite summary of hematological, ECG, and nerve conduction parameters across grades of anemia

Anemia Grade	Hb (g/dL)	Heart Rate (bpm)	Median Motor CV (m/s)	Ulnar Sensory CV (m/s)	Serum Ferritin (ng/mL)
Normal	14.08 ± 1.09	79.13 ± 4.95	65.52 ± 7.36	59.36 ± 5.81	60.03 ± 14.94
Mild	10.96 ± 0.59	77.75 ± 3.53	64.01 ± 9.53	60.04 ± 9.23	19.11 ± 17.59
Moderate	8.71 ± 0.78	85.90 ± 13.65	56.03 ± 10.59	50.35 ± 10.04	8.71 ± 2.04
Severe	5.91 ± 1.23	98.25 ± 20.95	46.27 ± 6.15	38.99 ± 4.31	6.74 ± 1.81

Table 1 presents a composite summary of hematological, ECG, and nerve conduction parameters across four anemia grades. Hemoglobin (Hb) levels declined progressively from **14.08 ± 1.09 g/dL** in the normal group to **10.96 ± 0.59, 8.71 ± 0.78**, and **5.91 ± 1.23 g/dL** in mild, moderate, and severe anemia, respectively. Correspondingly, **heart rate** increased from **79.13 ± 4.95 bpm** in normals to **77.75 ± 3.53, 85.90 ± 13.65**, and **98.25 ± 20.95 bpm,** indicating a compensatory tachycardia. Nerve conduction also showed progressive impairment, with **median motor conduction velocity** decreasing from **65.52 ± 7.36 m/s** in controls to **64.01 ± 9.53, 56.03 ± 10.59**, and **46.27 ± 6.15 m/s**, and **ulnar sensory conduction velocity** reducing from **59.36 ± 5.81 m/s** to **60.04 ± 9.23, 50.35 ± 10.04,** and **38.99 ± 4.31 m/s,** respectively. Serum ferritin, a marker of iron stores, also declined significantly with anemia severity—from **60.03 ± 14.94 ng/mL** in controls to **19.11 ± 17.59, 8.71 ± 2.04**, and **6.74 ± 1.81 ng/mL** in mild, moderate, and severe anemia, respectively.

Table 2: Correlation between hemoglobin and nerve conduction velocity (Cases Only)

Parameter	Pearson r	P-Value
Median Motor CV (m/s)	0.39	0.003 *
Median Sensory CV (m/s)	0.44	0.001 *
Ulnar Motor CV (m/s)	0.36	0.007 *
Ulnar Sensory CV (m/s)	0.41	0.002 *

Table 2 highlights the correlation between hemoglobin concentration and nerve conduction velocities among anemic participants. A moderate, statistically significant positive correlation was observed between Hb and median motor conduction velocity (r = 0.39, $p = 0.003$), median sensory CV (r = 0.44, $p = 0.001$), ulnar motor CV (r = 0.36, $p = 0.007$), and ulnar sensory CV (r = 0.41, $p = 0.002$). These findings reinforce the role of hemoglobin levels in maintaining normal nerve conduction, suggesting that neurophysiological dysfunction worsens as anemia deepens.

Table 3: Association between anemia severity and nerve conduction abnormalities

Anemia Grade	Normal NCS (n)	Abnormal NCS (n)	P-Value (Chi-Square)
Mild	8(50%)	8(50%)	<0.001 *
Moderate	6(19.4%)	25(80.6%)	<0.001 *
Severe	0	8(100%)	<0.001 *

Table 3 presents the association between anaemia severity and the frequency of nerve conduction abnormalities. Among those with mild anaemia, 8 out of 16 patients (50%) showed abnormal NCS results, whereas this increased to 25 out of 31

(80.6%) in moderate anaemia and 8 out of 8 (100%) in severe anaemia. The association was statistically significant with a *p*-value **<0.001** (Chi-square test). This progression confirms a strong relationship between anaemia severity and peripheral nerve dysfunction, indicating the importance of early neurodiagnostic screening in anaemic patients.

4. DISCUSSION

Our study found that increasing severity of iron deficiency anemia (IDA) was associated with a progressive and significant decline in hematological parameters, including hemoglobin and serum ferritin, as well as deterioration in nerve conduction function and a rise in heart rate. These findings are consistent with previous literature. For instance, Ochola and Onyemocho (2017)[16] reported that hemoglobin levels in IDA typically fall below 13.5 g/dL in males and 11.5 g/dL in females, with severe cases dropping further. Our observed serum ferritin values, which declined from 60.03 ± 14.94 ng/mL in normal individuals to 6.74 ± 1.81 ng/mL in severe anemia, align with the ferritin thresholds of 10–25 ng/mL suggested by Foy et al. (2020)[17] for significant iron depletion. The rise in heart rate observed in our study, from 79.13 ± 4.95 bpm in controls to 98.25 ± 20.95 bpm in severe anemia, corroborates earlier descriptions of anemia-induced tachycardia as a compensatory response to hypoxia. Importantly, our data also support neurophysiological findings reported by Paunikar et al. (2023)[18] and Sharma G et al. (2021)[19], who demonstrated reduced nerve conduction velocities and prolonged latencies in the median and ulnar nerves of anemic patients. In our cohort, median motor conduction velocity declined from 65.52 ± 7.36 m/s to 46.27 ± 6.15 m/s, and ulnar sensory conduction from 59.36 ± 5.81 m/s to 38.99 ± 4.31 m/s, mirroring the trend of worsening electrophysiological impairment with anemia severity. These findings reinforce the systemic nature of IDA, as emphasized by De Franceschi L et al. (2017)[20], affecting cardiovascular and peripheral nervous systems. Thus, our study adds to the growing body of evidence advocating for routine electrocardiographic and nerve conduction screening in patients with moderate to severe IDA to enable early detection of subclinical complications and timely intervention.

Our study demonstrated that hemoglobin levels significantly declined from 14.08 ± 1.09 g/dL in healthy controls to 5.91 ± 1.23 g/dL in severe anemia, while median motor conduction velocity (CV) decreased from 65.52 ± 7.36 m/s to 46.27 ± 6.15 m/s and ulnar sensory CV from 59.36 ± 5.81 m/s to 38.99 ± 4.31 m/s. A significant positive correlation was found between hemoglobin and nerve conduction parameters ($r = 0.36$ to 0.44, $p < 0.01$). These findings are consistent with Paunikar et al. (2023)**[18]**, who reported reduced CV and increased distal latency in both median and ulnar nerves across anemia grades, with significant differences ($p = 0.001$). Similarly, Ghosal et al. (2018)[21] observed slower conduction velocities and lower amplitudes in IDA patients compared to controls, and reported positive correlations between hemoglobin and nerve conduction velocities. Swaminathan et al. (2016)[22]also found prolonged latencies and reduced CMAP amplitudes in median, ulnar, and tibial nerves. Sharma G et al. (2021)[19] further showed that nerve conduction deficits in IDA (with hemoglobin 4–10.9 g/dL) were reversible after iron therapy. Together, these findings confirm that the progressive decline in hemoglobin in IDA is closely linked with deteriorating peripheral nerve function, which can be effectively detected and monitored using nerve conduction studies.

Our study demonstrated a strong and statistically significant association between the severity of iron deficiency anemia (IDA) and the prevalence of nerve conduction abnormalities, with abnormal NCS findings present in 50% of patients with mild anemia, 80.6% in moderate, and 100% in severe cases ($p < 0.001$). This progressive impairment aligns closely with previous studies, such as Sharma G et al. (2021)[19], which reported significantly increased distal latencies and reduced conduction velocities in IDA patients, particularly in severe grades. Similarly, Abraham et al. (2018)[23] observed marked neurophysiological deficits in severe anemia cases, attributing them to myelin disruption and axonal degeneration due to iron depletion. The 100% abnormal NCS rate in severe anemia in our study reinforces their conclusion that severe IDA is associated with critical peripheral nerve dysfunction. However, the 50% normal findings in mild cases also suggest that early-stage IDA may spare nerve conduction to some extent, indicating interindividual variability and possible compensatory mechanisms. These comparative insights emphasize the importance of early neurodiagnostic screening in IDA, especially as severity increases.

5. CONCLUSION

Iron deficiency anemia is associated with significant neurocardiac changes, including elevated heart rate and impaired nerve conduction, which worsen with increasing anemia severity. These alterations correlate strongly with hemoglobin and ferritin levels, indicating early subclinical involvement. Routine ECG and nerve conduction studies may aid in early detection and timely intervention, preventing long-term complications and highlighting the need for comprehensive management of IDA.

REFERENCES

[1] Ismail M, Ahmad P, Bashir SD, Naaz S, Khan F, Khan H. Prevalence of anemia in India and its multifactorial aetiology: a comprehensive review. Int J Adv Res. 2024;12(10):1259–65.

[2] Pasricha SR, Moir-Meyer G. Measuring the global burden of anaemia. *Lancet Haematol. 2023;*10(9):e696-e697.

[3] Safiri S, Kolahi AA, Noori M, Nejadghaderi SA, Karamzad N, Bragazzi NL, et al. Burden of anemia and its

underlying causes in 204 countries and territories, 1990–2019: results from the Global Burden of Disease Study 2019. J HematolOncol. 2021;14(1):185.

[4] Brittenham GM, Moir-Meyer G, Abuga KM, Datta-Mitra A, Cerami C, Green R, Pasricha SR, Atkinson SH. Biology of Anemia: A Public Health Perspective. J Nutr. 2023;153 Suppl 1:S7-S28.

[5] Maciel GA, Maciel DP, Vieira IC, dos Santos Silva T, de Brito SM, Junior AD, de Almeida AC, Nogueira MC, Beltrami AC, Soares PD, Quixabeira LA. Deficiency anemia: Causes, clinical picture, diagnosis and treatment. InIII Seven International Medical and Nursing Congress. Seven Congress.2023

[6] Korczak A, Wójcik E, Olek E, Łopacińska O, Stańczyk K, Korn A, Jędrzejczyk J, Szewczyk O, Burda K, Czarnecka K. The Long-Term effects of Iron Deficiency in Early Infancy on Neurodevelopment. Journal of Education, Health and Sport. 2024;70:51104-.

[7] Beard J. Iron deficiency alters brain development and functioning. The Journal of nutrition. 2003;133(5):1468S-72S.

[8] Sitorus NL, Salsabila IN. Impact of Iron Deficiency on Cognition in School Age Children. Journal of Indonesian Specialized Nutrition. 2023;1(2):1-7.

[9] Shah HE, Bhawnani N, Ethirajulu A, Alkasabera A, Onyali CB, Anim-Koranteng C, et al. Iron deficiency-induced changes in the hippocampus, corpus striatum, and monoamines levels that lead to anxiety, depression, sleep disorders, and psychotic disorders. Cureus. 2021;13(9).

[10] Priyan IG, Wasnik P, Kannauje PK, Das P, Singh S, Patel S, Priyan IG. Electrocardiographic Changes and Their Association With Disease Severity in Adults With Sickle Cell Anemia at a Tertiary Care Center: A Cross-Sectional Study. Cureus. 2024;13:16(5).

[11] Biradar SM, Holiyachi R, Shannawaz M, Teja VR. Electrocardiographic abnormalities in severe anaemia and its reversibility after correction of anaemia in North Karnataka, India. Int J Adv Med. 2020;7(10):1566–9.

[12] Mbakamma A, Alikor C, Enwere O, Odia O. Electrocardiographic abnormalities in adults with sickle cell anaemia. Saudi J Med Pharm Sci. 2019;5(12):1148–52.

[13] Radhika R, Mishra S, Birok R. EEG Graph-Based Disease Analysis: A Novel Approach to Neurological Disorder Diagnosis. In 2024 First International Conference on Electronics, Communication and Signal Processing (ICECSP). 2024;8:1-5

[14] Shigapova RR, Mukhamedshina Y. Electrophysiology methods for assessing of neurodegenerative and post-traumatic processes as applied to translational research. ReprodDev Biol. 2024;14(6):737.

[15] Jha A, Kuruvilla N, Garg P, Victor A. Harnessing Creative Methods for EEG Feature Extraction and Modeling in Neurological Disorder Diagnoses. In2023 7th International Conference on Computation System and Information Technology for Sustainable Solutions (CSITSS) 2023 Nov 2 (pp. 1-6). IEEE.

[16] Ochola IO, Onyemocho A. An Overview of Anaemia. Nurse Care Open Acces J. 2017;3(6):00091.

[17] Foy, B. H., Li, A., McClung, J. P., Ranganath, R., & Higgins, J. M. Data-driven physiologic thresholds for iron deficiency associated with hematologic decline. American Journal of Hematology.2020; 95(3):302–309.

[18] Paunikar VM, Rawekar AT, Barapatre SA. Assessment of peripheral nerve conduction in iron deficiency anemia patients in Indian rural adult population attending a tertiary care hospital. International Journal of Nutrition, Pharmacology, Neurological Diseases. 2023;13(3):173-80.

[19] Sharma G, Gupta S, Atri SK, Sharma P, Research M. Reversible alteration of nerve conduction velocity in iron deficient anemic patients in response to treatment. J Adv Med Biomed Res. 2021;33(7):40-4.

[20] De Franceschi L, Iolascon A, Taher A, Cappellini MD. Clinical management of iron deficiency anemia in adults: Systemic review on advances in diagnosis and treatment. European Journal of Internal Medicine. 2017;42:16-23.

[21] Ghosal S, Chattaraj W, Pramanik D, Mukherjee S, Banerjee U. Assessment of Peripheral Neuronal Activity with Nerve Conduction Studies in Iron Deficiency Anaemia Patients from Rural Areas of Bankura District of West Bengal. J Med SciClin Res. 2018;6(9):1051-1054.

[22] Swaminathan A, Kumarasamy S, Shanmugam S, Ayyavoo S, Velayutham S. Motor nerve conduction parameters in patients with iron deficiency anemia. Natl J Physiol Pharm Pharmacol. 2016;6(6):553-558.

[23] Abraham, A., Breiner, A., Barnett, C., Katzberg, H. D., Ngo, M., Lovblom, L. E., Perkins, B. A., &Bril, V. Laboratory Abnormalities in Polyneuropathy and Electrophysiological Correlations. Canadian Journal of Neurological Sciences.2018; 45(3): 346–349.