GMR
The Original

# CLINICO-RADIOLOGICAL CORRELATION OF MRI BRAIN FINDINGS AT DIAGNOSIS AND AFTER SIX MONTHS OF TREATMENT IN INFANTILE TREMOR SYNDROME

**Dr Amrita Mayanger[1], Dr Gopi Kishan Sharma[2*], Dr Pankaj Meena[3], Dr Shiv Prakash Rathore[4], Dr Sachin Kumar[5], Dr Harmandeep Kaur[6]**

[1]MD (Paediatrics), Professor, Department of Paediatrics, Government Medical College, Kota. Email: dramrita@hotmail.com
[2*]MD (Paediatrics), Associate Professor, Department of Paediatric, Government Medical College, Kota. Email: dr.gopikishansharma6162@gmail.com
[3]MD (Radio diagnosis), Assistant Professor, Department of Radiodiagnosis, Government Medical College, Kota. Email: pankajmeena2007@gmail.com
[4]MD (Biochemistry), Assistant Professor, Department of Biochemistry, Government Medical College, Kota. Email: rathore977@gmail.com
[5]MBBS, Junior Rresident, Department of Paediatrics, Government Medical College, Kota. Email: Sachup.1411@gmail.com
[6]MBBS, Junior Resident, Department of Paediatrics, Government Medical College, Kota. Email: d.harman024@gmail.com
*Corresponding Author: Dr Gopikishan Sharma, dr.gopikishansharma6162@gmail.com

## ABSTRACT

**Background:** Infantile tremor syndrome (ITS) continues to be reported from South-East Asian countries despite improvements in child nutrition programs, and its etiopathogenesis remains incompletely understood.

**Objectives:** This study aimed to evaluate neuroimaging abnormalities on magnetic resonance imaging (MRI) of the brain and correlate them with clinical findings at diagnosis and after six months of treatment in children with ITS. A secondary objective was to generate hypotheses regarding the possible etiopathogenesis of the syndrome.

**Methods:** This prospective cohort study was conducted over an 18-month period at a tertiary care hospital. Children aged six months to three years diagnosed with ITS, were enrolled. Clinical, hematological, and biochemical parameters of the children and their mothers were recorded at baseline using a predesigned proforma. Non-contrast MRI of the brain was performed after initial stabilization and repeated at six months during follow-up. Clinical, hematological, and biochemical assessments were also repeated at follow-up.

**Results:** A total of 49 children were included in the final analysis. Males constituted 57.1% of cases, and a majority had a history of faulty feeding practices. At admission, both children and their mothers demonstrated macrocytic anemia with low serum vitamin B12 levels. After six months of treatment, the prevalence of anemia declined to 57.1%, and vitamin B12 deficiency reduced to 14.3%. At follow-up, abnormal clinical findings persisted in 14.3% of children, developmental delay in 18.4%, and abnormal MRI findings in 57.1%. Normal MRI scans increased from 8.2% at baseline to 42.9% after six months ($p < 0.01$). A significant correlation was observed between improvement in clinical features and improvement in MRI findings.

**Conclusion:** The study demonstrates a significant association between clinical recovery and radiological improvement in children with ITS. Partial reversibility of neuroimaging abnormalities was observed over a six-month period following appropriate intervention, supporting the reversible nature of the disease when diagnosed and treated early.

**KEYWORDS:** Hematologic tests, Infant nutrition disorders, MRI scans, tremor, Vitamin B12

## INTRODUCTION

Infantile tremor syndrome (ITS) is a well-recognized pediatric disorder characterized by a constellation of pallor, developmental delay or regression, hyperpigmentation of the skin, and sparse, brownish scalp hair.1 Tremors are frequently observed but are not universally present. The syndrome typically affects infants between six and 24 months of age and shows a male preponderance.2 Systemic involvement is generally absent, and the neurological manifestations are distinctive, particularly the presence of tremors that occur during wakefulness and disappear during sleep.

The tremors of ITS predominantly involve the distal parts of the limbs and may also affect the head, face, and tongue. Despite being described several decades ago, the etiopathogenesis of ITS remains incompletely understood.3 Multiple hypotheses have been proposed, including nutritional deficiencies, metabolic abnormalities, and genetic predisposition. Among these, the nutritional deficiency hypothesis—particularly vitamin B12 deficiency—has gained the widest acceptance.

ITS is commonly reported in infants who are exclusively breastfed for prolonged periods by mothers with vitamin B12 deficiency, especially those following vegetarian diets.4 Delayed initiation or inadequacy of complementary feeding further exacerbates nutritional deficits during a critical period of rapid brain growth and myelination.5 Several studies have demonstrated a strong association between ITS and vitamin B12 deficiency, while deficiencies of other micronutrients such as iron and zinc have also been reported.

Hematologically, ITS is often associated with macrocytic or dimorphic anemia. Although vitamin B12 deficiency is considered a central etiological factor, the heterogeneity in hematological presentation and clinical response suggests that ITS is likely a manifestation of multiple micronutrient deficiencies rather than an isolated deficiency disorder. The persistence of ITS in South-East Asia, despite a decline in hospital-based incidence, highlights gaps in maternal and infant nutrition as well as in early detection.

Neurologically, vitamin B12 deficiency interferes with myelin synthesis, neuronal maturation, and neurotransmitter metabolism, leading to developmental delay, regression, and movement disorders. Altered melanin metabolism due to

metabolic defects has been proposed as a mechanism underlying the characteristic skin and hair pigmentation. Involvement of the substantia nigra has been suggested as a possible explanation for the tremors observed in ITS.
Advances in neuroimaging have enabled better characterization of central nervous system involvement in ITS.6,7 Several studies have reported cerebral atrophy, white matter changes, thinning of the corpus callosum, and hypomyelination on MRI. Case series have demonstrated reversibility of some of these changes following nutritional rehabilitation, although systematic longitudinal studies correlating clinical and radiological recovery remain scarce.
Clinical improvement in ITS is often rapid, with noticeable changes occurring within days to weeks of initiating therapy. However, data on the timeline and extent of radiological recovery are limited. This study was therefore designed to evaluate MRI brain findings at diagnosis and after six months of treatment and to correlate these findings with clinical parameters and to generate hypotheses regarding the possible etiopathogenesis of the syndrome.

## MATERIALS AND METHODS

### Study Design and Setting

This prospective cohort study was conducted in the Department of pediatrics at a tertiary care teaching hospital.

### Study Duration

The study was carried out over a two-year period from sixth April 2023 to fifth April 2025.

### Study Population

Children aged six months to 3 years diagnosed with infantile tremor syndrome were included.

### Case Definition

Children presenting with pallor, hyperpigmentation of the skin, and psychomotor changes such as apathy or developmental abnormalities, with or without tremors, were diagnosed as having ITS.
Ethical approval was obtained from the Institutional Ethics Committee, and informed consent was taken from parents or guardians.

### Exclusion Criteria

Children with known neurodegenerative disorders, cerebral palsy, pre-existing developmental delay, epilepsy, post-infectious neurological sequelae, or whose parents declined consent were excluded.

### Participant Selection

Due to the rarity of ITS, sample size estimation was not feasible. All eligible cases presenting during the study period were enrolled using consecutive sampling.

**Intervention & follow-up –** A detailed history was taken including presenting complaints, past history, family history, birth history, developmental milestones, immunization status and dietary history. Skin hyper pigmentation at terminal phalanges, hands, wrist, elbow, ankle, knee, or any other parts of the body were noted. Hair colour was recorded. Pallor was looked at palpebral conjunctiva, tongue, nails, and palmer creases. Type of tremor was noted as generalized or local.
Investigations including complete blood count (CBC) with blood indices (mean corpuscular volume [MCV], mean corpuscular haemoglobin [MCH], and MCH concentration [MCHC]), peripheral blood smear, serum Vitamin B12, serum folate, serum iron and serum ferritin levels were done in every case. Investigations of the patient's mother including complete blood count (CBC) with blood indices (mean corpuscular volume [MCV], mean corpuscular haemoglobin [MCH], and MCH concentration [MCHC]), peripheral blood smear, serum Vitamin B12, serum folate, serum iron and serum ferritin levels were also sent.
Peripheral venous blood sample was taken in an ethylenediaminetetraacetate [EDTA] vial (two ml) for the determination of CBC and five ml of blood was taken in a plain vial for the estimation of above-mentioned serum investigations. Serum Iron level was done by Fully automatic Biochemistry Analyzer Spectrophotometry by Transasia XL 640 & Beckman Coulter – AU-480 machine, Serum Vit B12 levels, Serum Folate levels and Serum Ferritin levels were done by Chemiluminescence Analyzer (Immunoassay Analyzer) by Roche-Cobas e411 & Beckenham Coulter ACCESS 2 machine. Deficiency of Hb, Iron, Ferritin, Folate and Vit B12 were considered if the serum values were less than 11g/dL, 30µg/dL, 120 ng/ml, 3ng/dl and 200ng/dl respectively in patients. 8,9,10,11
Non contrast MRI brain was done by Philips Acheiva 1.5T MRI machine, just after stabilization of the patient after the diagnosis is made. Follow up of the patient was done after six months of initiation of treatment and clinical features and biochemical investigations were recorded along with non-contrast MRI brain changes. The findings of the pre and six months follow up MRI were recorded by similar single experienced (with 11 years of experience) radiologist. MRI protocol and sequences used for all patients for pre & follow up scan were Axial T1, Axial FLAIR (Fluid attenuated inversion recovery sequence), Axial DWI (diffusion weighted image), Axial SWI (Susceptibility weighted image) and Sagittal T2 weighted image.

**Treatment protocol –** All patients were managed according to the standard unit protocol as per symptoms and signs. Tremors were treated with propranolol, with phenobarbitone added when clinically indicated. Associated infections and secondary complications were managed appropriately. Nutritional rehabilitation was implemented as per standard guidelines for severe acute malnutrition. 12
Vitamin B12 was administered at 500 µg/day in children younger than one year and 1000 µg/day in those older than one year, given intravenously or intramuscularly for seven days, followed by oral methylcobalamin 1500 µg daily (half tablet

in children less than two years and one tablet in children of two years and above) for six months. Oral elemental iron was given at 6 mg/kg/day for one month, followed by 3 mg/kg/day up to six months. Multivitamin supplementation was provided at twice the recommended daily allowance for two weeks, followed by the recommended daily allowance for six months. Folic acid was administered as 5 mg on day one, followed by 1 mg/day for two weeks. Elemental zinc was given at 2 mg/kg/day for two weeks. Magnesium sulphate was administered intramuscularly at 0.3 mL/kg (maximum 2 mL), followed by oral magnesium 0.3 mL daily for two weeks. Calcium supplementation was provided as 500 mg elemental calcium with 400 IU vitamin D per day in children younger than one year and 800 mg elemental calcium with 600 IU vitamin $D_3$ per day in children older than one year, continued for six months. Mothers were also supplemented with vitamin B12, iron, folic acid, multivitamins, and calcium. Dietary counselling and information regarding disease prognosis were provided to parents or caregivers.

**Outcome variables –** We have compared hematological and biochemical parameters (Hemoglobin, Serum Iron, Serum Ferritin, Serum Folate and vitamin B12 level) obtained after six months with the baseline values along with pre & follow up MRI findings. We have also studied the correlation of clinical feature improvement with MRI findings.
Clinical improvement is defined if the case improved in reference of anemia, improved mental activity (alert and playful), disappearance of tremor (If the tremor present initially), started depigmentation of skin hyper pigmentation and achieved next developmental milestone.

Radiological improvement is considered if follow-up MRI scans show signs of improvement in the following ways:
1) Improvement in cerebral atrophy
2) Resolution of white matter changes
3) Improvement in Cortical atrophy with hypomyelination
4) Improvement in any white matter changes mentioned in pre treatment scan
5) Other changes which were present in pre treatment scan showed improvement in form of resolution or reduced severity.

**Statistical Analysis**
Data were collected as entered in MS Excel and analyzed using SPSS version 23. Continuous data were summarized in terms of mean & standard deviation. Discrete data would be summarized in terms of proportion. Chi square test and McNemar test were applied for comparison as appropriate. P value less than 0.05 was considered significant.

**RESULTS**
The present study enrolled 58 children of six months to three years of age as per inclusion criteria. Out of them 6 children were excluded (Neurodegenerative disorder – two, Cerebral palsy -one, Post Meningoencephalitis sequel -one, Epilepsy-one and Refusal for consent-one) as per exclusion criteria and three were left out during study period. So, 49 cases variables were studied and analyzed for the purpose of study.
It is revealed that the majority were aged between six–12 months (63.3%) with a mean age of 11.84±5.24 months. Males constituted a slightly higher proportion (57.1%) compared to females (42.9%). The baseline characteristics of study subjects are summarized in Table 1

**Table 1: Baseline characteristics of study subjects**

| | **Variable** | **No. (%)/Mean±SD** |
|---|---|---|
| **Age group** | 6-12 months | 31(63.3%) |
| | 13-18 months | 16(32.7%) |
| | >18 months | 2(4.1%) |
| | **Mean age** | 11.84±5.24 years |
| **Gender** | Female | 21(42.9%) |
| | Male | 28(57.1%) |
| **Area of residence** | Rural | 34(69.4%) |
| | Urban | 15(30.6%) |
| | **Mean duration of stay** | 9.22±4.75 |

| | | |
|---|---|---|
| **Presenting complaints** | Cough/Cold/Difficulty in breathing | 34(69.4%) |
| | Diarrhoea/Vomiting/Pain abdomen | 11(22.4%) |
| | Weakness/Decreased activity/Oral stomatitis/Refusal to feed | 12(24.5%) |
| | Others | 15(30.6%) |
| **Feeding history** | Exclusive breastfeeding | 12 (24.4%) |
| | Faulty feeding | 37 (75.5%) |
| **Tremor** | No tremor (Prodromal stage) | 26 (53.1) |
| | Established tremor phase | 23 (46.9) |

At admission, in the study subjects the mean hemoglobin level was 7.76 ± 2.43 g/dL, indicating widespread anemia with Mean MCV was 94.46 fL, Biochemical markers showed a wide range, with mean serum Vitamin B12 at 190.61 pg/mL. The hematological profile of the mothers showed a mean hemoglobin level of 7.78 ± 2.24 g/dL and Mean MCV 98.41fL indicating widespread macrocytic anemia with deficient level of Serum Vitamin B12 levels, serum iron and ferritin levels. Different hematological parameters at admission in study subjects and in their mothers are shown in table 2.

**Table 2: Different hematological parameters at admission in study subjects and their mothers**

| **Hematological parameter** | **Study Subject** | | **Mothers** | |
|---|---|---|---|---|
| | Mean | SD | Mean | SD |
| Hb(g/dL) | 7.76 | 2.43 | 7.782 | 2.242 |
| MCV(fL) | 94.46 | 12.64 | 98.41 | 11.43 |
| MCHC(g/dL) | 31.93 | 2.76 | 32.51 | 2.74 |
| MCH(ph) | 30.21 | 5.08 | 28.845 | 3.341 |
| Platelets(mL) | 232.77 | 210.37 | 216.55 | 83.54 |
| WBC/µL | 8.164 | 4.537 | 7.30 | 2.209 |
| S.Vitamin B12 (pg/mL) | 190.61 | 293.28 | 209.82 | 182.22 |
| S.Iron (µg/mL) | 56.184 | 32.16 | 182.22 | 31.63 |
| S.Ferritin (ng/mL) | 123.96 | 160.27 | 35.763 | 44.565 |
| S.Folate (ng/mL) | 10.34 | 15.45 | 38.037 | 184.54 |

In comparison to baseline, there was a marked improvement after 6 months in different hematological and biochemical parameters. Across all parameters: anemia reduced to 57.1% and vitamin B12 deficiency to 14.3%, indicating significant recovery in nutritional status over the follow-up period. The comparative data are shown in Table 3

**Table 3: Nutritional deficiency in study subjects (n=49)**

| Variable | Baseline | 6 months | P value* |
|---|---|---|---|
| Hb <11 g/dL | 44(89.8%) | 28(57.1%) | **<0.001** |
| S.Iron <30 µg/dL | 15(30.6%) | 4(8.2%) | **0.002** |
| S.Ferritin <12 ng/mL | 14(28.6%) | 2(4.1%) | **0.001** |
| S. Folate <3 ng/mL | 3(6.1%) | 0(0.0%) | 0.24 |
| Vitamin B12<200 pg/mL | 41(83.7%) | 7(14.3%) | **<0.001** |

***McNemar Test applied**

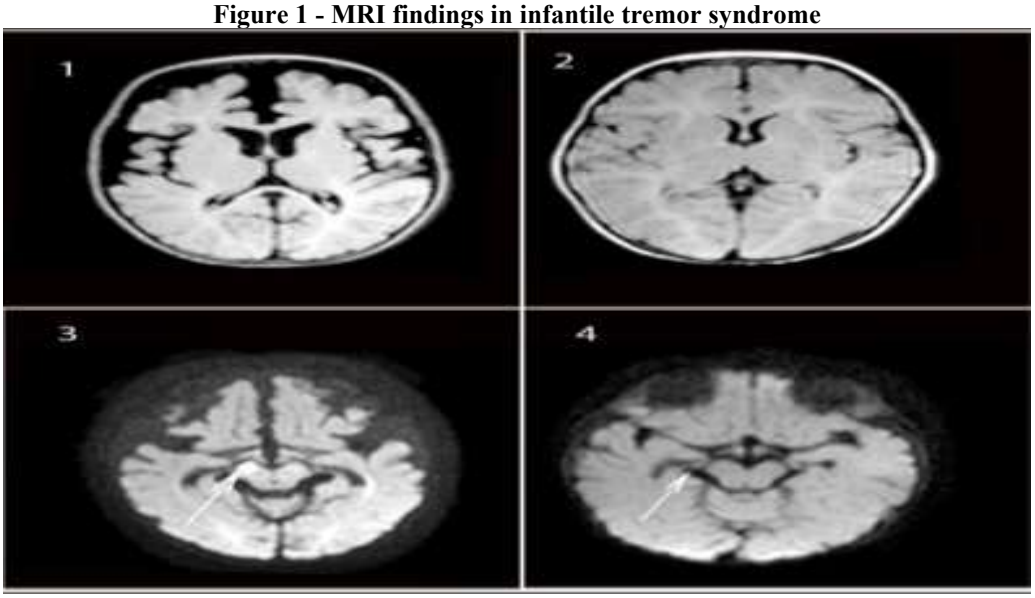
At the six month follow-up (n=49), clinical evaluation showed that seven (14.3%) subjects had abnormal clinical features, developmental assessment revealed delays in nine (18.4%) children while MRI findings were abnormal in 28 (57.1%) subjects, indicating that more than half had underlying neurological changes, despite clinical or developmental improvements in many cases. At baseline, only 4(8.2%) had normal MRI scans, which increased to 21 (42.9%) after six months of treatment. Six of the cases had partial resolution/recovery of the MRI findings in the follow up scan (but not completely normal) than previous scan, also considered radiological improvement. So, radiological improvement was observed in 23 of the 45 cases that had previously demonstrated abnormal MRI findings. MRI finding and different types of MRI abnormalities at baseline and at six months are shown in table 4 and Figure 1.

**Table 4: MRI Finding in study subjects at baseline and at 6 months (n=49)**

| MRI finding | Types of abnormality | Before treatment | After 6 months | P value* |
|---|---|---|---|---|
| Normal | Normal | 4(8.2%) | 21(42.9%) | <0.01 |
| Abnormal | Diffuse cerebral atrophy | 23(46.9%) | 13(26.5%) | |
| | Diffuse cortical atrophy with thinning of corpus callosum | 19(38.8%) | 13(26.5%) | |
| | Cortical atrophy with white matter changes | 2(4.1%) | 1(2.0%) | |
| | Cortical atrophy with hypomyelination | 1(2.0%) | 1(2.0%) | |

***McNemar test**

**Figure 1 - MRI findings in infantile tremor syndrome**




Axial FLAIR (1) at the time of initial presentation showing severe cerebral atrophy and diffusion-weighted (3) brain MRI at the time of initial presentation showing severe abnormal diffusion restriction (white arrow) while Axial FLAIR (2) and diffusion-weighted (4) brain MRI, at six months after initiation of treatment, showing resolution and normalization of

cerebral morphology and diffusion abnormality at the same levels respectively.
A significant correlation was observed between improvement in MRI findings and clinical feature improvement among the study subjects. Among those with improved clinical features, 23 also showed MRI improvement, while 15 did not. The data are shown in table 5.

**Table 5: Correlation of improvement of MRI Finding with clinical feature improvement in study subjects (n=49)**

| Clinical feature | MRI finding | | P value * |
|---|---|---|---|
| | Improved | Not improved | |
| **Improved** | **23[#]** | **15** | **<0.01** |
| **Not improved** | **0** | **7** | |

*Chi-square test (Initial 4 normal MRI cases are not included in this table)

**DISCUSSION**
Infantile tremor syndrome (ITS) remains a clinically important yet incompletely understood pediatric disorder, predominantly reported from developing countries. Despite being described several decades ago, uncertainties persist regarding its etiopathogenesis, clinical spectrum, and the reversibility of neurological changes. The present study provides longitudinal evidence correlating clinical, hematological, and radiological findings at diagnosis and after six months of treatment, thereby addressing a significant gap in existing literature.
In the current study, the majority of affected children were between six and 18 months of age, with a male predominance and rural background. This demographic pattern is consistent with previous reports from India and other low-resource settings, where male infants appear more frequently affected, possibly due to sociocultural feeding practices and delayed healthcare access.13,14 The high prevalence of respiratory and gastrointestinal infections at presentation can be explained by impaired immune function secondary to micronutrient deficiencies, a finding similarly reported by Gautam et al., who highlighted increased susceptibility to infections in children with ITS.11
Hematological evaluation at admission revealed widespread macrocytic anemia with low serum vitamin B12 levels in both children and their mothers, supporting the nutritional hypothesis of ITS. These findings are in concordance with earlier studies that identified vitamin B12 deficiency as a central etiological factor, particularly in exclusively breastfed infants born to vitamin B12–deficient mothers.3,5 The co-existence of iron and ferritin deficiency in a subset of cases further supports the concept that ITS represents a multimicronutrient deficiency state rather than an isolated vitamin B12 deficiency disorder.11,15
Following six months of nutritional rehabilitation and medical management, significant improvement was observed in hematological and biochemical parameters, with anemia reducing to 57.1% and vitamin B12 deficiency to 14.3%. Similar improvements in hemoglobin concentration and vitamin B12 levels have been documented in earlier interventional studies, reinforcing the effectiveness of timely nutritional supplementation.15 Vitamin B12 is essential for myelination and neuronal maturation; its deficiency disrupts central nervous system development, leading to tremors, developmental delay, and regression.16,17. The substantial clinical improvement observed in our cohort is therefore biologically plausible and consistent with established pathophysiological mechanisms.18
A key strength of the present study is the systematic evaluation of neuroimaging changes over time. At baseline, only 8.2% of children had normal MRI findings, while cerebral atrophy and white matter abnormalities predominated. After six months of treatment, normal MRI scans increased to 42.9%, and partial radiological recovery was observed in several cases, indicating reversibility of structural brain changes. Similar MRI abnormalities have been reported in earlier studies, including diffuse cerebral atrophy, corpus callosal thinning, and hypomyelination.6,9,19,20
Notably, while clinical recovery was evident in the majority of children, MRI abnormalities persisted in over half of the cases at six months. This discordance suggests that radiological recovery may lag behind clinical improvement. Gupta et al. demonstrated near-complete reversibility of cerebral atrophy after 15–18 months of follow-up, indicating that longer duration of nutritional rehabilitation may be required for full structural recovery.10 The comparatively partial reversibility observed in our study may therefore be attributed to the shorter follow-up duration.
A significant correlation between improvement in MRI findings and clinical recovery was observed in the present study, emphasizing that neuroimaging abnormalities in ITS have functional relevance. Similar associations between developmental improvement and radiological resolution have been reported previously, supporting the concept that early nutritional intervention can reverse both functional and structural brain changes.18 However, persistent abnormalities in some children may reflect prolonged deficiency or irreversible neuronal injury sustained during critical periods of brain development.
So Infantile tremor syndrome is a potentially reversible disorder when identified and treated early. The present study demonstrates significant clinical and hematological recovery accompanied by partial reversibility of MRI abnormalities within six months of treatment. Vitamin B12 deficiency, in the context of multimicronutrient deficiency, appears to play a central role in the pathogenesis of ITS. Preventive strategies focusing on maternal nutrition, early complementary feeding, and nutritional education are essential to reduce the burden of this preventable condition.
The strengths of this study include its prospective design, relatively larger sample size for a rare condition, and comprehensive correlation of clinical, hematological, and radiological parameters. However, limitations include the absence of longer-term follow-up beyond six months and the lack of assessment of additional metabolic markers such as homocysteine, methylmalonic acid, zinc, and vitamin D, which could have provided further insight into disease mechanisms.

**Acknowledgement -** We would like to express our gratitude for the support given by Multidisciplinary research unit, Govt. medical college Kota during research work and manuscript writing.

**REFERENCES**

1. Goraya JS, Kaur S. Infantile tremor syndrome: A review and critical appraisal of its etiology. J Pediatr Neurosci. 2016;11(4):298-304.
2. Chaparro CM, Suchdev PS. Anemia epidemiology, pathophysiology, and etiology in low- and middle-income countries. Ann N Y Acad Sci.
1. Garewal G, Narang A, Das KC. Infantile tremor syndrome: a vitamin B12 deficiency syndrome in infants. J Trop Pediatr. 1988;34(4):174-178.
2. Deshpande TV, Ingle VN. Tremor syndrome in children. Indian Pediatr. 1969;6(8):550-556.
3. Grattan-Smith PJ, Wilcken B, Procopis PG, Wise GA. The neurological syndrome of infantile cobalamin deficiency: developmental regression and involuntary movements. Mov Disord. 1997;12(1):39-46.
4. Sharma CM, Sharma D, Kumar R, Ranjan R. Rare association of thin corpus callosum with infantile tremor syndrome in a 5.5-month-old infant. J Pediatr Neurosci. 2015;10(3):273-275.
5. Gehlot,P.Gupta,R.Mandliya,J.C.,Singh,P.Pathak,A.Cranial Neuroimaging in Infantile Tremor Syndrome: The Road Ahead,J Clin of Diagn Res. 2018;12(3): SC01-SC04.
6. Holla RG, Prasad AN. Infantile Tremor Syndrome. Med J Armed Forces India. 2010 Apr;66(2):186-7.
7. Thora S, Mehta N. Cranial neuroimaging in infantile tremor syndrome (ITS). Indian Pediatr. 2007 Mar;44(3):218-20.
8. Gupta R, Pathak A, Mandliya J, Mandliya P, Sonker P. Reversible Cerebral Atrophy in Infantile Tremor Syndrome. Indian Pediatr. 2016;53(8):727-729.
9. Gautam P, Sharma N, Chaudhary S, Kaushal A. Infantile Tremor Syndrome in Modern Times. J PediatrNeurosci. 2017;12(3):232-236.
10. Ministry of Health and Family Welfare. Operational Guidelines on Facility Based Management of Children with Severe Acute Malnutrition. New Delhi: Ministry of Health and Family Welfare; 2017.
11. Sirolia V, Arya S. Study of clinical profile and estimation of Vitamin B12 level in infantile and pre-infantile tremor syndrome. J Evol Med Dent Sci 2014;3:10134-7.
12. Agarwal SP, Katiyar GP. Infantile tremor syndrome. Pediatr Clin India 1972;7:203-8.
13. Bolaman Z, Kadikoylu G, Yukselen V, Yavasoglu I, Barutca S, Senturk T. Oral versus intramuscular cobalamin treatment in megaloblastic anemia: a single-center, prospective, randomized, open-label study. Clin Ther. 2003 Dec;25(12):3124-34. doi: 10.1016/s0149-2918(03)90096-8.
14. Amin NM, Zeki JM. Infantile tremor syndrome in Iraqi Kurdistan. Indian J Pediatr. 2005;72(10):839-842.
15. Kaul KK. Syndrome of tremors in Indian infants. J Indian Med Assoc. 1972;59(11):473-475.
16. Singla DA, Sharma M, Sharma S, Sharma V. The changing profile of infantile tremor syndrome in hilly terrain of India. J Neurosci Rural Pract 2015;6:610-2.
17. Goraya JS, Kaur S. Infantile tremor syndrome -- down but not out. Indian Pediatr. 2015;52(3):249-250.
18. Bajaj, M., Thakur, A., Sharma, J., Sharma, M., & Pathania, S. Study of clinical, haematological and radiological profile of children admitted with infantile tremor syndrome to a tertiary care teaching hospital in Northern India. International Journal of Contemporary Pediatrics 2022;9(7):684–689.