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Case Report

Neurotherapy as a Complementary Approach for Beta-Thalassemia Intermedia

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Abstract

Beta-thalassemia intermedia (β-TI) is a genetic disorder characterized by chronic anaemia resulting from ineffective erythropoiesis. While management typically involves transfusions and pharmacological therapies, these carry risks like iron overload and variable efficacy. Alternative approaches, such as neurotherapy, offer potential for improving haematological outcomes without invasive interventions. The aim of this study was to evaluate the impact of neurotherapy as a complementary treatment in a 44-year-old female patient with β-TI. A 44-year-old female with β-TI underwent 48 neurotherapy sessions over three months (four sessions per week). Therapy targeted specific pressure points to stimulate organ functions critical for haemoglobin synthesis and overall health, including the pancreas, kidneys, thyroid, and liver. Techniques focused on improving erythropoietin production, enhancing iron metabolism, and promoting globin chain synthesis. Following therapy, the patient's haemoglobin level increased from 5.9 g/dL to 9.7 g/dL, with improvements in haematocrit, MCV, MCH, MCHC and ferritin levels. Platelets, serum TSH and ESR showed significant reductions, while vitamin B12 normalized. Clinically, the patient reported increased energy levels, reduced fatigue, and improved physical activity tolerance. No adverse effects were observed, and the patient expressed high satisfaction with the outcomes. This case highlights the potential of neurotherapy as a safe and effective adjunctive treatment for β-TI. By improving haematological parameters and alleviating fatigue, neurotherapy could serve as a valuable addition to the therapeutic toolkit for managing this condition.

Keywords: Beta-Thalassemia; Beta-Thalassemia Intermedia; Anaemia; Neurotherapy; Treatment; Complementary Therapy.

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Quick Response Code:

Introduction

Beta-thalassemia (β-T) is one of the most common inherited genetic disorders, caused by reduced production of beta-globin chain subunits. This imbalance disrupts the delicate ratio between alpha-globin and beta-globin chains, leading to ineffective erythropoiesis and chronic haemolytic anaemia [1]. Among its forms, beta-thalassemia intermedia (β-TI) is a distinct phenotype characterized by chronic anaemia, ineffective erythropoiesis, and complications primarily related to iron overload [2,3].

The clinical spectrum of β-T is highly heterogeneous, ranging from asymptomatic carriers to individuals with severe, transfusion-dependent anaemia, making diagnosis and classification challenges. At one end of the spectrum is thalassemia minor, which manifests as mild hypochromic microcytic anaemia with minimal or no clinical symptoms. At the other extreme is thalassemia major (TM), a severe form that typically presents within the first year of life, with affected individuals experiencing profound anaemia that necessitates lifelong, regular blood transfusions for survival [3,4].

Positioned between these extremes is thalassemia intermedia (TI), first described by Sturgeon, referring to individuals whose disease severity is milder than TM but more pronounced than thalassemia minor [5].TI is characterized by an intermediate clinical severity that does not meet the transfusion dependency criteria of TM [6].The earliest comprehensive description of this condition was provided in 1955 by Rietti-Greppi-Micheli, who documented cases with clinical features falling between those of thalassemia minor and TM [7].TI is a genetically diverse disorder, leading to a wide range of presentations from mild to severe haemolytic anaemia [7].

Patients with TI can be categorized into two broad subgroups:

Mild anaemia group – These individuals maintain haemoglobin levels between 7 and 11 g/dL, remain largely transfusion-independent, and may require occasional transfusions during periods of stress, illness, or pregnancy.

Severe anaemia group – These patients present with more pronounced anaemia. Although they may not require regular transfusions, they are at risk of developing complications such as skeletal deformities, growth retardation, and other morbidities if inadequately managed [7,8,9].

Differentiating TI from TM is crucial for optimizing management strategies and preventing long-term complications. While some clinical overlaps exist, TI is typically characterized by pallor, jaundice, splenomegaly, skeletal abnormalities, and chronic anaemia. Diagnosis is often supported by haemoglobin levels around 7 g/dL and a family history suggestive of an atypical beta-thalassemia carrier parent. Laboratory findings commonly reveal microcytic hypochromic anaemia [9,10].

The primary goal of TI treatment is to prevent complications and enhance quality of life. Current management strategies include transfusion therapy, iron chelation, splenectomy, pharmacological modulation of gamma-globin chain production, and stem cell transplantation [1]. However, these treatments have limitations and varying degrees of efficacy. In this context, a non-invasive alternative or complementary approach could be highly beneficial. By addressing the unmet needs of this patient population, such an intervention has the potential to significantly improve outcomes and provide a timely solution.

Case Presentation

The presented case of β-TI was managed over a three-month period (April–June 2023) at a clinic in eastern India. Before the study commenced, the patient provided written informed consent, including permission for photographs to be taken during therapy sessions. Ethical approval was obtained prior to the study's initiation. Additionally, there are no competing interests, and all data presented originates solely from specified sources.

Case History

The patient is a 44-year-old married woman who presented to the neurotherapy clinic with complaints of general fatigue, extreme weakness, and shortness of breath during physical exertion. She reported a history of splenomegaly, facial bone expansion, and mild visual protrusion of the lower jaw but denied any prior surgeries. Although the patient had normal serum iron levels, she had been taking iron supplements and was on levothyroxine for hypothyroidism. Her family history revealed that her father had TM and that hypothyroidism was prevalent on both the paternal and maternal sides.

The patient was diagnosed with TM during her first pregnancy in 2008 but did not experience any significant health issues in her daily life. However, in April 2023, a routine blood test revealed an Hb level of 5.9 g/dL, which became a critical concern. Despite receiving erythropoietin injections and attempting various homeopathic treatments and home remedies, she did not observe any significant improvement. Seeking alternative solutions, she was introduced to Neurotherapy through a family member who had experienced positive results for a different condition. Encouraged by this recommendation and abnormal laboratory findings, she decided to explore Neurotherapy as a complementary approach to managing her condition.

Biochemical Evaluation

The blood investigation report, analysed using an ELISA test analyser, revealed the following:

- **Thyroid Function Tests**: The serum thyroid-stimulating hormone (TSH) level was elevated at 8.334 µIU/mL (reference range: 0.350-4.940 µIU/mL), indicating hypothyroidism. However, the levels of T3 (107.9 ng/mL, reference range: 35-193 ng/mL) and T4 (8.47 µIU/mL, reference range: 4.87-11.71 µIU/mL) were within normal limits.
- **Vitamin B12**: The serum vitamin B12 level was significantly reduced, measuring <148 pg/mL (reference range: 187-883 pg/mL), suggesting a deficiency.
- **Inflammatory Marker**: The erythrocyte sedimentation rate (ESR) was elevated to 25 mm in the first hour (reference range: 0-20 mm), indicative of an underlying inflammatory or chronic condition.
- **Iron Storage**: Ferritin levels were markedly low at 2.30 ng/mL (reference range: 4.63-204.0 ng/mL), pointing to depleted iron stores.
- **Complete Blood Count (CBC)**:
    - Haemoglobin (Hb) was critically low at 5.9 g/dL (reference range: 12.0-15.0 g/dL), consistent with severe anaemia.
    - Platelet count was markedly elevated at 679 thousand/µL (reference range: 150-410 thousand/µL), indicating thrombocytosis, as corroborated by the peripheral smear.
    - Haematocrit was significantly reduced to 20.2% (reference range: 36-46%), reflecting severe anaemia.
    - Mean corpuscular volume (MCV) was notably low at 47.9 fL (reference range: 83-101 fL), indicating microcytosis.
    - Mean corpuscular haemoglobin (MCH) was also decreased to 14.1 pg (reference range: 27.0-32.0 pg), consistent with hypochromia.
    - Mean corpuscular haemoglobin concentration (MCHC) was slightly below normal at 29.4 g/dL (reference range: 31.5-34.5 g/dL).

These findings collectively indicate severe microcytic hypochromic anaemia with associated iron deficiency, vitamin B12 deficiency, hypothyroidism, and reactive thrombocytosis.

Intervention

Neurotherapy is an emerging technique in the field of alternative medicine that has shown promising results in managing patients with βT. This innovative approach offers a unique perspective by focusing on circulation-based pressure therapy, which aims to enhance blood flow and improve overall physiological function. By stimulating specific pressure points in alignment with blood circulation pathways, neurotherapy works to slow the progression of genetic or clinical disorders [11,12].

This holistic treatment method addresses multiple underlying factors contributing to anaemia and related complications in patients with β-TI. By improving blood circulation and addressing the body's imbalances, it promotes better oxygen delivery to tissues, helping alleviate symptoms like fatigue and weakness. Neurotherapy exemplifies the growing potential of non-invasive, complementary approaches in improving patient outcomes and enhancing the quality of life for those living with chronic conditions.

For the given case of β-TI, the patient underwent a total of 48 therapy sessions over a period of three months, with four sessions per week. Each session targeted specific pressure points, designed to stimulate organ functions critical for maintaining blood health and regulating iron metabolism. The detailed techniques employed during the therapy are as follows:

Pancreas Stimulation:

Pressure was applied to the area above the thigh muscle near the pelvic bone in a "V" shape. The pressure was applied in intervals of 6 seconds and repeated 8 times during each session (Figure 1). According to neurotherapy principles, this stimulation is believed to activate the pancreas, promoting RNA production. Enhanced RNA synthesis indirectly supports the formation of globin, a key component of haemoglobin, thereby addressing the impaired haemoglobin production characteristic of β-TI.

Kidney Stimulation

Specific pressure points on the left thigh muscle and left shoulder were stimulated to enhance blood flow to the right kidney, while pressure on the right thigh muscle and right shoulder was applied to stimulate the left kidney. Each pressure point was stimulated for 6 seconds and repeated 7 times (Figure 2). This technique aims to improve the kidneys' ability to produce erythropoietin (EPO), a hormone essential for stimulating red blood cell (RBC) production in the bone marrow. By boosting EPO levels, this method helps address the patient’s low haemoglobin levels and supports overall blood health.

Thyroid Stimulation

Gentle pressure and rubbing were applied to specific areas beside the trachea on the patient’s throat. This stimulation was done in half-second intervals, repeated 4 times during each session (Figure 3). The goal of this therapy was to enhance the production of thyroid hormones, specifically TSH. These hormones play a critical role in metabolic regulation and contribute to the synthesis of globin chains, an essential component of functional haemoglobin.

Liver Stimulation

Pressure was applied to two key areas: the left middle thigh and the left shoulder, as well as the left middle thigh and the region extending from the elbow to the wrist. The pressure was applied for 6 seconds, repeated 6 times, with 30-second intervals between applications (Figure 4a & 4b). This stimulation is believed to optimize the liver's role in regulating iron metabolism. By enhancing the production of hepcidin, a hormone responsible for maintaining iron balance, this technique ensures that iron is efficiently managed and readily available for haemoglobin synthesis.

The above therapies were specifically designed to address the unique challenges faced by the patient, including impaired haemoglobin production, low iron metabolism, and reduced RBC count. By targeting the pancreas, kidneys, thyroid, and liver, the treatment aimed to stimulate physiological processes that collectively improve blood health and iron utilization. This comprehensive approach highlights the potential of neurotherapy as a complementary treatment for β-TI, focusing on restoring balance and enhancing organ function.

Results

After 12 weeks of consistent therapy sessions (four sessions per week), the patient demonstrated remarkable improvements in various biochemical and haematological parameters. The serum TSH level decreased significantly to 5.51 µIU/mL, and the ESR reduced to 20 mm/hr, both showing considerable progress, although still slightly above the normal range. Additionally, the vitamin B12 level exhibited a dramatic increase to 740 pg/mL, and the ferritin level rose to 55 ng/mL, both returning to within normal limits. The patient's Hb level increased substantially, reaching 9.7 g/dL, reflecting improved RBC production. Other complete blood count (CBC) indices, such as MCV, MCH, and MCHC, also showed improvement, indicating enhanced red blood cell morphology and functionality (Table 1). Although some of these parameters were still slightly outside the normal range, the overall trends indicated significant progress.

Figure 1: Application of pressure on 'pancreas' point

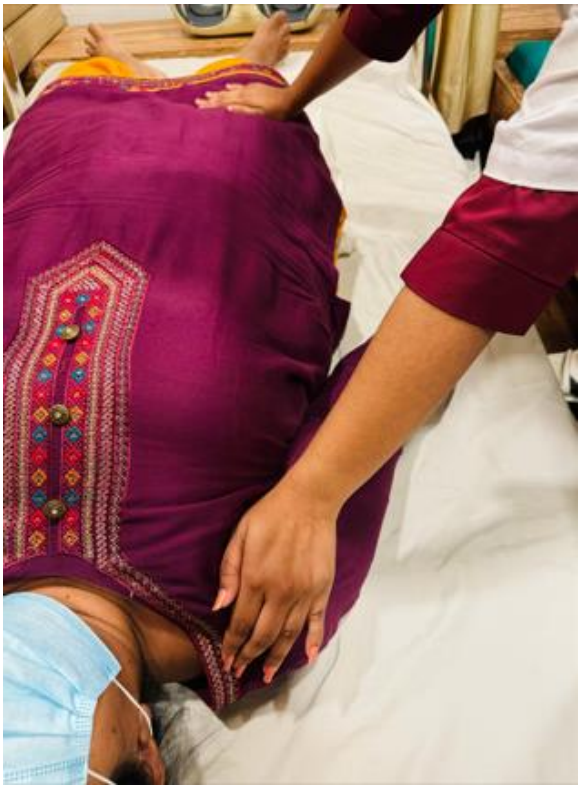

Figure 2: Application of pressure on 'kidney' point

Figure 3: Application of pressure on 'thyroid' point

Figure 4a & 4b: Application of pressure on 'liver' point

Table 1: Profile of laboratory results of patient with β-TI (Pre & Post Therapy

Parameters	Pre-Therapy	Post-Therapy
TSH	8.33 µIU/mL	5.51 µIU/mL
Vitamin B12	<148 pg/mL	740 pg/mL
ESR	25 mm in the first hour	20 mm in the first hour
Ferritin	2.3 ng/mL	55.0 ng/mL
Hb	5.9 g/dL	9.7 g/dL
Platelet	679 thousand/µL	420 thousand/µL
Haematocrit	20.2%	32.1%
MCV	47.9 fL	68.4 fL
MCH	14.1 pg	20.7 pg
MCHC	29.4 g/dL	30.2 g/dL

Abbreviations: TSH=Thyroid Stimulating Hormone, ESR=Erythrocyte Sedimentation Rate, Hb=Haemoglobin, MCV=Mean Corpuscular Volume, MCH=Mean Corpuscular Haemoglobin, MCHC=Mean Corpuscular Haemoglobin Concentration, µIU/mL=micro-international units per milliliter, pg/ml=picogram per milliliter, ng/ml=nanogram per milliliter, g/dL=gram per decilitre, thousand/µL=thousand per microliter, %=percentage, fL=femtoliter, pg=picogram.

Clinically, the patient reported a noticeable boost in energy levels and no longer experienced shortness of breath during physical activity. These improvements greatly enhanced her quality of life and daily functioning. After completing therapy, the patient was monitored through telephonic follow-ups to assess any potential side effects or after-effects. Our team is pleased to report that the patient provided overwhelmingly positive feedback, expressing satisfaction with the treatment journey and its outcomes. She appeared genuinely content with the results, further supporting the efficacy of this therapeutic approach.

Discussion

β-TI is a complex and heterogeneous condition that poses significant management challenges, particularly in adults. Conventional treatments, such as blood transfusions, iron chelation, and erythropoietin therapy, can mitigate symptoms to some extent. However, they often have limitations, potential side effects, and do not address the underlying metabolic and physiological factors driving disease progression.

In this case, a complementary and non-invasive therapeutic approach was explored, aimed at improving haemoglobin levels, optimizing iron metabolism, and alleviating clinical symptoms associated with β-TI. The observed outcomes suggest that this approach not only eased symptoms but also enhanced haematological parameters and overall well-being.

The apparent efficacy of this therapy may be attributed to its multifaceted mechanism, targeting key physiological systems. Pancreatic stimulation likely enhanced globin synthesis, while kidney stimulation appeared to boost erythropoietin production, promoting RBC generation. Liver stimulation may have contributed to optimized iron metabolism, and thyroid stimulation likely supported metabolic processes critical for effective globin chain synthesis. Together, these interventions seem to have addressed core contributors to anaemia in β-TI.

Despite these promising results, certain limitations must be acknowledged. The findings are based on a single patient, and the influence of other factors, such as dietary modifications or lifestyle changes, cannot be ruled out. Additionally, while no adverse effects were reported during follow-up, the long-term sustainability of these improvements remains uncertain.

Future research should focus on large-scale randomized controlled trials to assess the efficacy, safety, and reproducibility of this therapeutic approach.

Conclusion

Neurotherapy has shown promising results in managing β-TI, underscoring its potential as a valuable complementary treatment option for patient care.This case highlights the need for innovative and holistic approaches in the management of β-TI, emphasizing the importance of addressing underlying factors and fostering long-term patient well-being.

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