Letter of Transmittal

Nashville, Tennessee | February 21st, 2026

"revise" is a better term here!

Subject: Request a re-write of a treatment plan according to a new and unique SCOPE, particularly in the context of the recent arrival of ChatGPT/AI tools for Drug Research (Disease/Symptoms of Muscle Atrophy (Bệnh teo cơ))

To: Patient Care Team

I am writing to you on behalf of my friend. I am coincidentally a chemical engineer/pharmacist with a new and unique scope of treatment, yet still aligned with current medical protocols of practice. Please see my case draft for Dementia Syndrome (The Wife of Jay Leno - The Great Comedian of Our Time - The Late-Night Show) in the attachment. I also detailed a re-growth and nutrition supplement for the case of Tatiana Kennedy-Schlossberg (JFK's granddaughter) with acute myeloid leukemia (AML) of the bone marrow, involving a rare chromosome 3 inversion gene defect.

I have conducted an interview with his aunt, Mrs. Tu Le, who is retired after a long nursing career in a hospital setting here in Nashville, Tennessee. This patient developed the disease and symptoms of Muscle Atrophy (Bệnh teo cơ) right after his marriage at a young age, and his siblings also happened to acquire it around the same time - close in age - which fits very well with the characteristics of a gene defect and Type 3 for the case Muscle Atrophy (Bệnh teo cơ). This Type 3 is also well defined as the juvenile form, or Wohlfart–Kugelberg–Welander disease.

Even though he is skinny, he is still able to make movements (walk) and engage in limited social interactions such as attending church services and enjoying social media, as these skills date back to before he contracted/acquired this disease.

This disease has already received attention, but there is still no clear-cut treatment. Now, with the new wave of scientific revolution, I feel confident in my new and unique scope of treatment- and now, additionally, with ChatGPT/AI applications for drug research coming into play.

Here is my comment and also the key to my proposal:

This disease is identified as a gene defect - a type of tumor cancer - but it is not toxic/damaging and not aggressive to other organs or cells. It simply PREVENTS The Production of SMN Protein. This can be replenished by a supplement (similar to the case of enzymes in the digestive system). Here, I suggest using [Option 1] Evrysdi (risdiplam) 0.75 mg/mL [ORAL SOLUTION] bottle. It is very expensive in the form of an injection IV drug, so use the oral solution instead, which also allows easier adjustment as needed. It's $7,000 per bottle for the Roche brand and only $200 for a generic drug.

importing from India!

We certainly need to follow up with a plan for re-growth and nutritional supplementation as detailed in a HARD case of Tatiana Kennedy-Schlossberg (JFK's granddaughter) with acute myeloid leukemia (AML) of the bone marrow ABOVE!

I once was:

A pioneer– placed the Clinical Pharmacist in an active role in medication management services, as a job/career, not just dispensing and answering at requests since August 2005 as a Doctor Pharmacy Student at Duquesne University – Mylan School of Pharmacy in Pittsburgh, Pennsylvania. Pharmacist Intern (ranked 4th Year)- a job, not at school's rotation at University of Pittsburgh Medical Center – South Side.

A first and early inventor (03.10.2020) of Covid-19 vaccine based on a DNA – fake Covid Virus model (like flu virus in FLU vaccine) from a new field- Pharmacogenomics. The official Covid-19 vaccine (Nov. 2020) is a mRNA coded strand of Covid Virus- similar to ours (DNA- more accurate) but required up to 3rd booster from their Microbiology field – these two physician scientists later were awarded with Nobel in Medicine in 2022.

A Chemical Engineer & Mathematician with some training/studies in hospital pharmacy. Graduated from the Ivy Leagues: Vanderbilt & Johns Hopkins.

I sincerely appreciate your time to review this new treatment proposal.

Thank you!

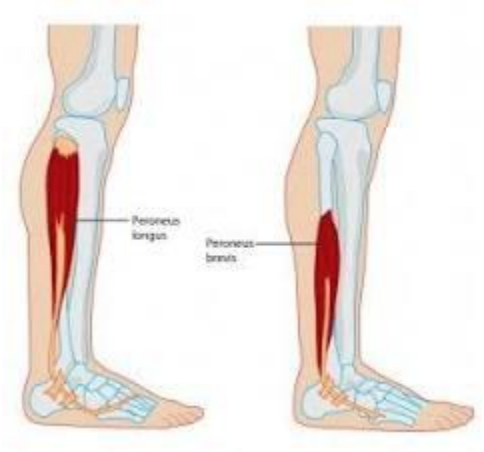
- Winston Hieu-Duc Vo

From the Desk of Winston Vo- specializing in chemical engineering, hospital pharmacy, & control systems- under the guidance of Lawyers Dad & Uncle.

** Schooled at Ivy League(s): Vanderbilt and Johns Hopkins.*

Disease / Symptoms of Muscle Atrophy (Bệnh teo cơ)

AI Overview (Generated by ChatGPT/AI apps)



Muscle atrophy (Bệnh teo cơ) is the wasting or loss of muscle tissue, leading to decreased strength and size, often caused by disuse, aging, malnutrition, or nerve damage. Symptoms include visible muscle shrinkage, weakness, and limited mobility. Reversible with exercise and improved nutrition, it can be managed with physical therapy, though severe, neurogenic cases may require medical intervention.

This case is later identified as a defect in the SMN1 gene!

Symptoms of Muscle Atrophy

- Visible Muscle Reduction: One limb or muscle group appears visibly smaller than the other.
- Muscle Weakness:
  Noticeable loss of strength in affected muscles.
- Reduced Physical Activity/Mobility: Difficulties with movements like walking or climbing stairs.
- Fatigue: Easily tired or limited exercise capability.

Here is just BRAINSTORM - this case is later identified as a defect in the SMN1 gene.

Causes of Muscle Atrophy

- Disuse (Physiologic) Atrophy: Caused by not using muscles enough due to sedentary lifestyles, bed rest, or immobilization (e.g., cast).
- Neurogenic Atrophy: The most severe type, caused by damaged nerves connecting to muscles (e.g., ALS, spinal cord injuries, neuropathy).
- Pathologic Atrophy: Triggered by illness (cancer, AIDS, heart failure), severe burns, starvation, or aging (sarcopenia).

Just look in detail, even though these health conditions are not applicable to your situation.

Certain diseases and chronic health conditions can contribute to muscle atrophy, such as:

Amyotrophic lateral sclerosis (ALS), also known as Lou Gehrig's disease , is a condition encompassing several types that damage the motor neurons that control muscles.

Sounds good! But here … the gene SMN1 has a defect

Multiple sclerosis: This chronic condition occurs when the immune system attacks the central nervous system, causing inflammation in the nerve fibers.

Arthritis: Arthritis involves inflammation in the joints, causing pain and stiffness. Arthritis can severely limit a person's mobility, leading to muscle atrophy.

Myositis: This condition causes muscle pain and weakness. You may develop myositis due to a viral infection or as a result of autoimmune diseases.

Polio: This infectious disease attacks the nervous system, causing flu-like symptoms and permanent paralysis.

Another explanation/definition in laymen's terms (Vinmec Health System)

Muscle atrophy, also known as muscular dystrophy, is a term referring to the loss of muscle mass over time. This occurs when there is an imbalance between the body's ability to synthesize protein and its breakdown. Over time, if left untreated, the symptoms can become severe and lead to disability.

Once diagnosed as a gene defect - here it is detailed as in modern medicine (2025)

Spinal muscular atrophies usually result from autosomal recessive mutations that affect the survival motor neuron 1 (*SMN1*) gene on the long arm of chromosome 5 (thiếu hụt các tế bào thần kinh vận động), most often causing a homozygous deletion of exon 7. Spinal muscular atrophies may involve the central nervous system and thus are not purely peripheral nervous system disorders. *SMN2* is a modifier gene; it is 99% identical to the *SMN1* gene and is located on 5q; if present in multiple copies, *SMN2* may modify the severity of the disease and explain phenotypic differences between children with SMA. Also, there are rare forms of SMA that are not due to 5q mutations.

There are 5 main types of spinal muscular atrophy.

In **spinal muscular atrophy type 0,** onset is prenatal; it manifests as decreased fetal movement in late pregnancy and severe weakness and hypotonia at birth. Affected neonates have facial diplegia, areflexia, cardiac defects, and sometimes arthrogryposis. Death due to respiratory failure occurs within the first 6 months.

Spinal muscular atrophy type 1 (infantile spinal muscular atrophy, or Werdnig-Hoffmann disease) is also present in utero and becomes symptomatic by about age 6 months. Affected infants have hypotonia (often notable at birth), hyporeflexia, tongue fasciculations, and pronounced difficulty sucking, swallowing, and eventually breathing. Death, usually due to respiratory failure, occurs within the first year in 95% and by age 4 years in all.

In **spinal muscular atrophy type 2** (intermediate form, or Dubowitz disease), symptoms usually manifest between 3 and 15 months of age; < 25% of affected children learn to sit, and none walk or crawl. Children have flaccid muscle weakness and fasciculations, which may be hard to see in young children. Deep tendon reflexes are absent. Dysphagia may be present. Most children are confined to a wheelchair by age 2

to 3 years. The disorder is often fatal in early life, frequently resulting from respiratory complications. However, progression can stop spontaneously, leaving children with permanent, nonprogressive weakness and a high risk of severe scoliosis and its complications.

My friend (patient) fits this category well!

Spinal muscular atrophy type 3 (juvenile form, or Wohlfart-Kugelberg-Welander disease) usually manifests between age 15 months and 19 years. Findings are similar to those of type I, but progression is slower and life expectancy is longer; some patients have a normal life span. Some familial cases are secondary to specific enzyme defects (eg, hexosaminidase deficiency). Symmetric weakness and wasting progress from proximal to distal areas and are most evident in the legs, beginning in the quadriceps and hip flexors. Later, arms are affected. Life expectancy depends on whether respiratory complications develop.

Spinal muscular atrophy type 4 (late-onset) can be recessive, dominant, or X-linked, with adult onset (age 30 to 60 years) and slow progression of primarily proximal muscle weakness and wasting. Differentiating this disorder from amyotrophic lateral sclerosis that involves predominantly lower motor neurons may be difficult.

Another short and recap about this disease cause

Spinal Muscular Atrophy (SMA) is a genetic disorder primarily caused by a deficiency in a specific protein needed for motor neurons to survive.

Option 1: Target the SMN2 gene to increase SMN protein production with Evrysdi (risdiplam) 0.75mg/ml [ORAL SOLUTION] bottle. The oral solution seems ideal in terms of COST and Acceptable Route!

Primary Genetic Cause: SMN1 Gene

- **SMN Protein Deficiency:** In approximately **95% of cases**, SMA is caused by a mutation or deletion of both copies of the **SMN1** (Survival Motor Neuron 1) gene on chromosome 5.
- **Motor Neuron Loss:** This gene is responsible for producing the **SMN protein**, which is essential for the health and survival of motor neurons—the nerve cells in the spinal cord that control voluntary muscle movement.
- **Muscle Atrophy:** Without enough SMN protein, these motor neurons shrink and eventually die. As a result, the brain can no longer send signals to the muscles, leading them to waste away (atrophy).

The Role of the "Backup" Gene: SMN2

Every person with SMA has at least one copy of a "backup" gene called **SMN2**.

- **Limited Production:** While SMN2 is nearly identical to SMN1, it only produces about **10–15%** of the functional SMN protein the body needs.
- **Severity Modifier:** The number of SMN2 gene copies a person has (usually between 0 and 8) directly influences the severity of the disease. Generally, **more copies of SMN2** lead to a milder form of SMA because more functional protein is being produced.

Inheritance Pattern

SMA is typically an **autosomal recessive** condition:

Carrier Parents: Most children with SMA inherit one mutated SMN1 gene from each parent. The parents are usually "carriers" who have one normal gene and one mutated gene, meaning they show no symptoms themselves.

- **Risk per Pregnancy:** If both parents are carriers, there is a **25% chance** with each pregnancy that the child will have SMA.
- **Rare "De Novo" Mutations:** In about **2% of cases**, a child may inherit one mutation from a parent while the second mutation occurs spontaneously during embryonic development.

Treatments

(Comments by author – Winston Vo): *This disease is identified as a gene defect - a type of tumor cancer - but it is not toxic/damaging and not aggressive to other organs or cells. It simply PREVENTS The Production of SMN Protein. This can be replenished by a supplement (similar to the case of enzymes in the digestive system). Here, I suggest using [Option 1] Evrysdi (risdiplam) 0.75 mg/mL [ORAL SOLUTION] bottle. It is very expensive in the form of an injection IV drug, so use the oral solution instead, which also allows easier adjustment as needed. It's $7,000 per bottle for the Roche brand and only $200 for a generic drug.*

Prices in India

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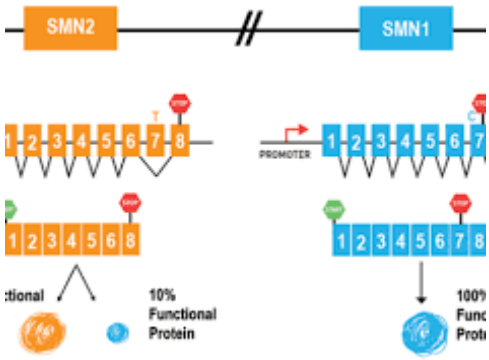

Treatments for *SMN1*-related spinal muscular atrophy (SMA) focus on increasing SMN protein levels through genetic therapies, primarily including Zolgensma (gene replacement for children <2), Spinraza (antisense oligonucleotide for all ages), and Evrysdi (oral medication). Early intervention, especially pre-symptomatic, is critical for best outcomes.

FDA-Approved Disease-Modifying Therapies (SMN1-Targeted)

- Onasemnogene abeparvovec-xioi (Zolgensma): A one-time intravenous gene therapy for children under 2 years old that delivers a functional copy of the *SMN1* gene.

- Nusinersen (Spinraza): An antisense oligonucleotide (ASO) injected into the spinal canal (lumbar puncture) that modifies the *SMN2* gene to produce more functional SMN protein.
- Risdiplam (Evrysdi): An oral, daily medication that also targets the *SMN2* gene to increase SMN protein production, approved for individuals 2 months and older.

Supportive and Rehabilitative Therapies

- Physical and Occupational Therapy: Exercises to improve strength, maintain joint flexibility, and manage contractures.
- Respiratory Care: Techniques and devices to support breathing, which is crucial as muscles weaken.
- Nutritional Support: Feeding assistance for swallowing difficulties.

Emerging Research

- Research is ongoing into enhancing the efficacy of these treatments and exploring new therapeutic targets to further boost SMN levels.

Drug Cost

Again - here the term "Target" is used, not "Modify", like the Option 2.

Option 1: Target the *SMN2* gene to increase SMN protein production, approved for individuals 2 months and older.

(Imported from India) Evrysdi (risdiplam) 0.75mg/ml [ORAL SOLUTION] bottle:

Cost (cheap)

- Roche (Evrysdi): ~₹6.2 Lakh (approx. $7,440)
- Natco Generic (Natsmart): ₹15,900 (approx. $190)

(in USA) Evrysdi (risdiplam) [INJECTION] is a high-cost specialty medication for spinal muscular atrophy (SMA) with an annual list price of up to $340,000–$354,000 for patients weighing 44 lbs (20 kg) or more.

"Modify" is HARD to achieve!

Option 2: Modify the **SMN2** gene to produce more functional SMN protein

(imported from India) Spinraza (nusinersen) [INJECTION, not available in Oral Solution]: A single vial (12mg/5ml) can range from ₹70,000 to ₹150,000 or more, with treatment requiring multiple doses.

(in USA) Spinraza (nusinersen) [INJECTION] is a highly expensive, life-long treatment for spinal muscular atrophy (SMA), with a wholesale acquisition cost of approximately $750,000 for the first year (covering initial doses) and around $375,000 annually thereafter. It costs roughly $125,000 per injection.

Therapy Beside Drug Use

Plan for a re-growth and nutrition supplement as the case of Tatiana Kennedy-Schlossberg (JFK's granddaughter) with acute myeloid leukemia (AML) of the bone marrow!

Physiotherapy

Physical therapy includes specific exercises and stretching routines aimed at preventing immobility. Physical therapy offers the following benefits for people with muscle atrophy:

- Preventing immobilization.
- Strengthen muscles.
- Improves blood circulation.
- Reduces muscle stiffness.

Functional electrical stimulation

Functional electrical stimulation (FES) is an effective treatment for muscle atrophy. Doctors use electrical impulses to stimulate muscle contraction in the affected muscles.

In this procedure, electrodes will be attached to a limb that is atrophied. The electrodes transmit an electric current, triggering movement in the limb.

Plan for a re-growth and nutrition supplement

FOR DRUG USE and MINDSET, especially MY NEW SCOPE of Treatments - very unique, yet STILL aligned with current medical protocols of practice - SEE MY CASE DRAFT for Dementia SYNDROME (The Wife of Jay Leno - The Great Comedian of Our Time - The Late-Night Show) in the attachment.

The case of Tatiana Kennedy-Schlossberg (JFK's granddaughter) with acute myeloid leukemia (AML) of the bone marrow is a much better sample, one that I have already fully developed!

Looking forward to hearing from you!

- Winston Hieu-Duc Vo

From the Desk of Winston Vo- specializing in chemical engineering, hospital pharmacy, & control systems- under the guidance of Lawyers Dad & Uncle.

** Schooled at Ivy League(s): Vanderbilt and Johns Hopkins.*

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Short Biography of Winston Hieu-Duc Vo

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Neuroplasticity Art AIKIDO Portfolio LLC (http://www.NeuroplasticityArtAIKIDO.com): to advocate and promote: Neuroplasticity. Scatterbrain. Stretching. Then, AIKIDO- a

Japanese martial art – is a choice that any sport should adopt – at least “Just body warm-up exercise”. MADE FOR Bright, Creative, and Wellness-Focused!

The Church Townhall Gala (http://www.TheChurchTownhallGala.com). Now: Re-Discovery and Sharing its treasures- which can beat Any New Book Club, as it even goes further to benefit in a Wellness or Healing Recovery Process! Even further, incorporate these new influences into the Church’s Preaching and Sunday Bible School.

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