

## Deficiency of Vitamin E and Dehydration as a Cause of Deep Venous Thrombosis in a Child with Cystic Fibrosis

Virtut Velmishi<sup>1\*</sup>, Melpomeni Bizhga<sup>2</sup>

<sup>1</sup>Department of Pediatric Gastroenterology Mother Teresa Hospital, Tirana, Albania; <sup>2</sup>Department of Pediatric Pulmonology, Mother Teresa Hospital, Tirana, Albania

### ABSTRACT

Deep Vein Thrombosis (DVT) is caused by a blood clot that occurs in the deep venous system. DVTs are rare in healthy children, but may occur more commonly in children that are hospitalized or have chronic diseases. Children with cystic fibrosis have a higher risk to develop deep vein thrombosis. Signs and symptoms of DVT include swelling, pain, and changes in skin color of the affected limb. Diagnosis of DVT is usually confirmed with an imaging test such as an ultrasound. Treatment for DVT includes waiting and watching to see what happens, medication, thrombolysis, or surgery. We describe a child with cystic fibrosis who presented a deep vein thrombosis of left leg as a cause of dehydration and vitamin E deficiency.

**Keywords:** Deep vein thrombosis; Cystic fibrosis; Vitamin E

### INTRODUCTION

Venous thromboembolism is increasing in the pediatric population, especially in children with cystic fibrosis. Due to central venous catheters, acquired thrombophilia due to chronic inflammation, deficiency of anticoagulant proteins due to vitamin K deficiency or liver dysfunction [1]. Vitamin E for these supplements are given to children with fat malabsorption such as in cystic fibrosis and cholestatic liver disease [2].

### CASE PRESENTATION

Our patient is the first child of an Albanian couple without consanguinity. Birth weight was 2400 g. Pregnancy and delivery were normal. This child was breastfed but his mother noticed poor weight gain and several episodes of diarrhea. He was sent to our center at the age of 4 months. He presented failure to thrive and sweat test resulted 130 mmol/L confirming cystic fibrosis. No history of important disease in family. The rest of examination was quite normal except mild hypoalbuminemia and hyponatremia. After two albumin infusions and regulation of electrolyte imbalance this child was discharged at home with Creon, vitamin ADK supplement every day and vitamin E 100 mg/day in alternate days. A month later this child is returned to our service because of dehydration, poor feeding and

developed swelling and tenderness of left leg. Skin over the affected area was turned reddish (Figure 1). He presented pain during leg movements associated with warm skin over affected zone. According to his mother treatment was not administered properly because of frequent vomiting and child refusal.



**Figure 1:** This picture shows left leg which presents swelling and reddish color.

Hemorrhage and coagulation test showed: hemorrhage time 2 min; coagulation time 3 min 30 sek; PT 89.5%; APTT C 27 sek.; D dimer was positive. Testing for secondary infections showed: CRP negative. Tests for viral and bacterial agents: Negative were a

**Corresponding Author:** Virtut Velmishi, Department of Pediatric Gastroenterology, Mother Teresa Hospital, Tirana, Albania, E-mail address: tutimodh@yahoo.com

**Received:** May 21, 2021; **Accepted:** June 04, 2021; **Published:** June 11, 2021

**Citation:** Velmishi V, Bizhga M (2021) Deficiency of Vitamin E and Dehydration as a Cause of Deep Venous Thrombosis in a Child with Cystic Fibrosis. *Pediatr Ther.* 11:390

**Copyright:** © 2021 Velmishi V, et al. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

Other tests: Homocysteine 6.8 micromol/l ;Protein C 61% (N 70%-110%); Protein S 67% (N 65%-58%). Level of E vitamin in 0.8 mcg/mL (3-18.4µg/dL). We excluded antiphospholipid in antibodies (aPLs), FVLeiden and antithrombin deficiency. Doppler ultrasound of the leg showed a blood clot at distal part of posterior tibial vein but overall flow of the blood is not being constrained or restricted. We started immediately rehydration 3l/m<sup>2</sup> correcting hyponatremia. At the same time we restarted a creon , vitamin A,D,K and vitamin E 50 mg per day making sure a proper administration. We used antibiotics to prevent infection. Blood clot dissolved and leg took normal color 4 days later. During follow up this boy didn't show any thrombosis phenomenon.

## DISCUSSION

The incidence of venous thromboembolism is increasing in pediatric population especially in hospitalized children [3]. In the Children with cystic fibrosis have an increased risk of thrombosis due to central venous catheters , as well as acquired thrombophilia secondary to inflammation or deficiencies of anticoagulant proteins due to vitamin K deficiency and liver dysfunction [4]. In our case we excluded these etiologies because our patient had not a central vein, he had a normal PCR excluding a severe inflammation. According to normal INR we logically can say that is excluded deficit of vitamin K. Liver function was also normal. Most of our tests regarding thrombophilia resulted negative. It is well known that dehydration remains a serious cause of deep vein thrombosis such as in our case [5]. The second point which is not frequently described in literature is the deficiency of vitamin E as a cause of thrombosis. Vitamin E is all the following eight compounds alpha, beta, gamma, and delta-tocopherol and alpha, beta, and gamma, and delta-tocotrienol. Alpha-tocopherol is the only they compound of eight that are known to meet human dietary needs. All of the vitamin E forms are absorbed in small intestine, and then liver metabolizes only alpha-tocopherol. The liver then removes and excretes the remaining vitamin E forms [6]. Vitamin E is a lipid soluble nutrient. Patient with cystic has fibrosis fail to secrete pancreatic enzymes to absorb vitamins A, D, E and K. Vitamin E inhibits platelet adhesion by there in preventing oxidative changes to LDLs and inhibition of platelet aggregation by reducing prostaglandin E<sub>2</sub>. In our patient we found a level of vitamin E 0.8 µg/dL [7]. In infants a level of vitamin E lower than 3 µg/dL is considered as deficiency. In this context we started immediately a prompt administration of vitamin E 100 mg per day during 7 days and later 50 mg per day.

continuously [8]. We didn't start thrombolytic agents because of small age of our patient and lack of experience. Preferring a strategy wait and see the outcome of conservative treatment including aggressive rehydration, creon and vitamin in our case to supplements resulted very efficient [9].

## CONCLUSION

Indications of Alfa Tocopherol Acetate (Vitamin E) in cystic fibrosis were well designed. Vitamin E is given by mouth 50 mg/once day adjusted as necessary in children 1 month to 1 years old with cystic fibrosis. Deficiency of vitamin E associated with other risk factor such as dehydration in our case may lead to deep vein thrombosis. A prompt treatment can avoid serious complications. Other studies should be done to confirm our findings.

## CONFLICT OF INTEREST

The authors declare that they don't have conflict of interest

## REFERENCES

1. Askegard-Giesmann JR, Brien SH, Wang W, Kenney BD. Increased use of enoxyparine in pediatric trauma patients. *J Pediatr Surg.* 2012;47(5):980-9833.
2. Sapiejka E, Krzyzanowska-Janowska P, Wenska- Chuzu E, Szczepanik M, Walkowiak D, Cofta S, et al. Vitamin E status and its determinants in patients with cystic fibrosis. *Adv Med Sci.* 2018;63(2):341-346.
3. Raffini LJ, Raybagkar D, Blumenstein MS, Rubenstein RC, Manno CS. Cystic fibrosis as a risk factor for recurrent venous thrombosis at a pediatric tertiary care hospital. *J Pediatr.* 2006;148(5):659-664.
4. Clifford MT. Venous thromboembolism in cystic fibrosis. *Pediatr Pulmonol.* 2012 47(2):105-112.
5. Ishola T, Kirk SE, Guffey D, Voigt K, Shah MD, Srivaths L, et al. Risk factors and co-morbidities in adolescent thromboembolism are different than those in younger children. *Thromb Res.* 2016;141:178-82.
6. Kim HK, Han SN. Vitamin E: Regulatory role on gene and protein expression and metabolomics profiles. *IUBMB Life.* 2019;71(4): 442-455.
7. Suzuki H, Kume A, Herbas MS. Potential of Vitamin E Deficiency, Induced by Inhibition of α-Tocopherol Efflux, in Murine Malaria Infection. *Int J Mol Sci.* 2018;24;20(1):64
8. Jilani T, Iqbal MP. Vitamin E deficiency in South Asian population and the therapeutic use of alpha-tocopherol (Vitamin E) for correction of anemia. *Pak J Med Sci.* 2018;34(6):1571-1575.
9. Khadangi F, Azzi A. Vitamin E: The Next 100 Years. *IUBMB Life.* 2019;71(4):411-415.