INTERNATIONAL
AYURVEDIC
MEDICAL JOURNAL

IAMJ

Case Report ISSN: 2320 5091 Impact Factor: 5.344

ARGINASE DEFICIENCY – A CASE STUDY

Palak Rathod[1], Manjunath Adiga[2], Pushpa Jogihalli[3]

[1]PG Scholar, Department of Kayachikitsa; [2]Associate Professor, Department of Kayachikitsa;
[3]Associate Professor, Department of Kaumarabhritya;
SKAMCH & RC, Bengaluru, Karnataka, India

Email: palakrathod2407@gmail.com

ABSTRACT

Arginase deficiency is an inherited disorder that causes ammonia to accumulate gradually in the blood. It is caused due to mutations in the ARG1 gene. It is inherited in autosomal recessive pattern. Arginase deficiency is a very rare disorder; it has been estimated to occur once in every 300,000 to 1,000,000 individuals[1]. A 4 year old patient with complaints of delayed milestones (unable to walk, sit without support and speak), a known case of Seizures was diagnosed with Arginase deficiency at an age of 1 year approached the *Balaroga* OPD of SKAMCH&RC, Bangalore and This condition considered under the group of *Sahaja vikaras*, was managed with different *Panchakarma* treatments, Oral medications and Physiotherapy. A good improvement was noticed in terms of speech, ability to sit without support after set of treatments.

Keywords: Arginase deficiency, Seizures, *Panchakarma, Shamana Aushadhi*

INTRODUCTION

Arginase deficiency is an inherited disorder that causes the amino acid - arginine and ammonia to accumulate gradually in the blood. Ammonia, which is formed when proteins are broken down in the body, is toxic if levels become too high. The nervous system is especially sensitive to the effects of excess ammonia. Arginase deficiency usually becomes evident by about the age of 3. It most often appears as stiffness, especially in the legs, caused by abnormal spasticity of the muscles. Other symptoms may include slower growth, developmental delay and eventual loss of developmental milestones, intellectual disability, seizures, tremor, and ataxia. Occasionally, high protein meals or stress caused by illness or fasting may cause ammonia to accumulate more quickly in the blood. In some affected individuals, signs and symptoms of arginase deficiency may be less severe and may not appear until later in life. Arginase deficiency is a very rare disorder; it has been estimated to occur once in every 300,000 to 1,000,000 individuals[1]. It is caused due to mutation in ARG gene 1.

This condition can be understood in Ayurveda under the banner of *Sahaja Vikara* and *Samvardhana Vikara*. There is no detail description given of such disease by any Acharya and since this is one of the rare diseases, no treatment protocol was followed, observations were made.

Case Report

A 3 year 8 months old male child visited the OPD of *Balaroga*, SKAMCH&RC on. He presented with complaints – unable to sit without support, unable to stand with or without support, unable to walk with or without support, unable to speak.

History of Present Illness

At 6th month of age, restless cry was noticed without reason and parents went hospital as he was suffering with high fever and started with febrile seizures which lasted for 5 mins. Treatment was done for 8 days in ICU and 20 days in hospital. Patient then complained of frequent cold, cough and difficulty in breathing and was taken to hospital frequently. At 10th month, parents complained that patient is not able to sit without support and was then diagnosed as Developmental delay secondary to Arginase deficiency. He was then treated with anti-epileptic drug and physiotherapy but did not much improvement. Patient was taken brought to our college for further management. Patient had 3 episodes of attack (1st attack at 6th month, second at 2018 and third at Oct' 2019).

Patient's parents had a consanguineous marriage. Antenatal scan did not confirm any abnormality, mode of delivery was LSCS and birth weight was 3.5kgs with good sucking reflex and no pathological jaundice was observed.

Treatment: Treatment was planned on every visit based on the improvements observed in the patient. On 1st visit - *Kumara kalyana rasa* ¼ OD along with honey, Bravobol 2.5ml BD with Low protein and milk diet was advised for 1 month. On 2nd visit - Memtone 5ml BD and *Matra basti with Maha kalyanaka ghrita* 20ml for 10 days. On 3rd visit - Syp Memtone 2.5ml BD, Syp Colic Carmin 5ml BD Before food, *Brahmi Ghrita* ½ tsp OD, Physiotherapy twice a week for 1 month. *Sarvanga Abhyanga* with *Ksheerabala taila* followed by *Shastika Shali Pinda Sweda* with *Maha Paichaka Ghrita* ½ tsp OD with food for 16 days.

Observation: On 2nd visit – Patient was able to speak letters. On 3rd visit - Patient can sit without support till 10-15mins, stand with support, and walk with support (Scissor Gait), Stiffness reduced in hands. On 4th visit – Stiffness of legs slightly reduced.

Table 1: Observation made before and after treatment.

Before Treatment	After Treatment
Unable to sit without support	Sit without support till 10-15mins
Unable to stand with or without support	Stand with support
Unable to walk with or without support	Walk with support
Hands were always in flexed position	Stiffness reduced in B/L hands
Unable to speak	Able to speak letters

DISCUSSION

Initially, *Kumara kalyana Rasa* was given with intention of observation. Ingredients of *Kumara kalyana rasa* includes *suddha parada, swarna, mukta, abhraka, loha, makshika bhasma* with *kumara rasa* and these are *pitta – vata hara* in action and *muktwa* is one the *vata nanatmaja rogas.* When patient started responding with improvement in speech, *Matra basti* in *bahumatra* (20ml) was started with intension to tackle *vata* which was aggravated and to strengthen muscles of the limbs. Since there was *vata prakopa* noticed in patient, *matra basti* was selected. *Maha kalyanaka ghrita*[2] is proved *medhya* and *brumhaniya*. *Brahmi ghrita* was taken into consideration as it has *brahmi – medha janana* and its *phalashruti – unmada, apasamara, and alakshmi*[3]. Physiotherapy was started later with an intention of trial and error keeping in mind the stress in muscle tissue and baby responded to it. Later *Sarvanga Abhyanga* with *Shastika Shali Pinda Sweda* was started. This was not adopted initially as in some demyelinating disorder *abhyanga* is not adopted initially so after keen observation this treatment was started. *Abhyanga* and *Shastika Shali Pinda Sweda* are proved to be *brimhana* and *balya* in action. *Maha paichaka ghrita*[4] was administered as it is *buddhi, smruti karam, balanam anga vardhanam*.

CONCLUSION

Arginase deficiency is an autosomal recessive urea cycle disorder and there is no cure available for this disease. In Ayurveda, we can manage the condition and improve the quality of life for these patients. *Charaka Acharaya in Sutrasthana*[5], explained that we always need not name a disease and can treat that with the understanding of involvement of *hetu vishesha, sthana of dosha* vitiation and *prakruti of vikruti*. There was an attempt made to understand this disease and manage accordingly.

REFERENCES

1. U.S National Library of medicine, Genetic Home reference, Jan 21st 2020, https://ghr.nlm.nih.gov/condition/arginase-deficiency#statistics
2. Harishachandra Singh Kushwaha, Charaka Samhita, 1st edition, Chikitsa sthana, 9th chapter, 42 – 44 shloka, Chaukhamba Surbharathi prakashan, Varanasi, pg. 257
3. Harishachandra Singh Kushwaha, Charaka Samhita, 1st edition, Chikitsa sthana, 10th chapter, 25 – 27 shloka, Chaukhamba Surbharathi prakashan, Varanasi, pg. 267
4. Harishachandra Singh Kushwaha, Charaka Samhita, 1st edition, Chikitsa sthana, 9th chapter, 45 – 48 shloka, Chaukhamba Surbharathi prakashan, Varanasi, pg.257
5. Harishachandra Singh Kushwaha, Charaka Samhita, 1st edition, Sutras sthana, 18th chapter, 44 – 47 shloka, Chaukhamba Surbharathi prakashan, Varanasi, pg. 287

Source of Support: Nil
Conflict of Interest: None Declared

How to cite this URL: Palak Rathod: Arginase Deficiency – A Case Study. International Ayurvedic Medical Journal {online} 2020 {cited February, 2020} Available from: http://www.iamj.in/posts/images/upload/2859_2861.pdf