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CHARCHOT MARRIE TOOTH DISEASE -ANUKTA VYADHI - A CASE STUDY

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ABSTRACT

Charchot-Marie-Tooth (CMT) disease is the most common type of hereditary neuropathy. The various subtypes of CMT are classified according to the nerve conduction velocities, predominant pathology (e.g., demyelination or axonal degeneration), inheritance pattern (autosomal dominant, recessive or X- linked) and specific mutated genes. There are no medical therapies for any of the CMT'S but physical and occupational therapy can be beneficial.[1] Such disease is not explained in Ayurveda. Therefore, CMT disease can be treated with the concept of *Anukta Vyadhi* explained by Charak. Management of such disease depend on factors involved in disease process such as *Dosha*, *Dushya* (*Vikar Prakriti*), *Adhishtan* (place of lesion) and *Nidan* (*Samutthan Vishesh*).[2] Being a hereditary neuropathy, disease in most of the cases, is expressed in childhood and early adult life. A case recorded and was treated in Government Ayurved College and Hospital Nagpur. Results obtained were encouraging which are presented in full paper.

INTRODUCTION

Charcot-Marie-Tooth (CMT) disease is the most common type of hereditary neuropathy. Rather than one disease, CMT is a syndrome of several genetically distinct disorders. The various subtypes of CMT are classified according to the nerve conduction velocities and predominant pathology (e.g., demyelination or axonal degeneration), inheritance pattern (autosomal dominant, recessive, or X-linked), and the specific mutated genes. Loss of touch sensation in the feet, ankles, and legs, as well as in the hands, wrists, and arms occurs with

various types of the disease. High-arched feet (pes cavus) or flat-arched feet (pes planus) are classically associated with the disorder. Scoliosis is common, causing hunching and loss of height.[3] Difficulty in chewing, swallowing, and speaking (due to atrophy of vocal cords).[4] Type 1 CMT (or CMT1) refers to inherited demyelinating sensorimotor neuropathies, whereas the axonal sensory neuropathies are classified as CMT2. motor conduction velocities in the arms are slowed to less than 38 m/s in CMT1 and are greater than 38 m/s in CMT2. However, most cases of CMT1 actually have motor nerve conduction velocities (NCVs) between 20 and 25 m/s. CMT1 and CMT2 usually begin in childhood or early adult life; however, onset later in life can occur, particularly in CMT2. Both are associated with autosomal dominant inheritance, with a few exceptions. CMT3 is an autosomal dominant neuropathy that appears in infancy and is associated with severe demyelination or hypomyelination. CMT4 is an autosomal recessive neuropathy that typically begins in childhood or early adult life.

CMT1

CMT1 is the most common form of hereditary neuropathy, with the ratio of CMT1:CMT2 being approximately 2:1. Affected individuals usually present in the first to third decade of life with distal leg weakness (e.g., foot Drop), although patients may remain asymptomatic even late in life. People with CMT generally do not complain of numbness or tingling, which can be helpful in distinguishing CMT from acquired forms of neuropathy in which sensory symptoms usually predominate. Reduced sensation to all modalities is apparent on examination. Muscle stretch reflexes are unobtainable or reduced throughout. There is often atrophy of the muscles below the knee (particularly the anterior compartment), leading to so-called inverted champagne bottle legs. Nerve biopsies usually are not performed on patients suspected of having CMT1, because the diagnosis usually can be made by less invasive testing (e.g., NCS and genetic studies). However, when done, the biopsies reveal reduction of myelinated nerve fibres with a predilection for the loss of the large-diameter fibres and Schwann cell proliferation around thinly or demyelinated fibres, forming so-called onion bulbs. CMT1A is the most common subtype of CMT1, representing 70% of cases, and is caused by a 1.5-megabase (Mb) duplication within chromosome 17p11.2-12 wherein the gene for peripheral myelin protein-22 (PMP-22) lies. This results in patients having three copies of the PMP-22 gene rather than two. Approximately 20% of patients with CMT1 have CMT1B, which is caused by mutations in the myelin protein zero (MPZ). CMT1B is for the

most part clinically, electro-physiologically, and histologically indistinguishable from CMT1A.

CMT2

Affected individuals usually become symptomatic in the second decade of life. some cases present earlier in childhood, whereas others remain asymptomatic into late adult life. Clinically, CMT2 is for the most part indistinguishable from CMT1. NCS are helpful in this regard in contrast to CMT1. The velocities are normal or only slightly slowed. The most common cause of CMT2 is a mutation in the gene for mitofusin 2 (MFN2), which accounts for one-third of CMT2 cases overall.

CMT3

CMT3 was originally described by Dejerine and Sottas as a hereditary demyelinating sensorimotor polyneuropathy presenting in infancy or early childhood. Affected children are severely weak. Motor NCVs are markedly slowed, typically 5–10 m/s or less. Most cases of CMT3 are caused by point mutations in the genes for PMP-22, MPZ, or ERG-2, which are also the genes responsible for CMT1.

CMT4

CMT4 is extremely rare and is characterized by a severe, childhood-onset sensorimotor polyneuropathy that is usually inherited in an autosomal recessive fashion. Electrophysiologic and histologic evaluations can show demyelinating or axonal features. CMT4 is genetically heterogenic.

There are no medical therapies for any of the CMTs, but physical and occupational therapy can be beneficial, as can bracing (e.g., ankle-foot orthotics for foot drop) and other orthotic devices.[1] Prognosis according to 2007 review stated that, "life expectancy is not known to be altered in the majority of cases.[5]

Charcot-Marie-Tooth (CMT) disease is not described in Ayurveda. Therefore, it can be termed as *Anukta Vyadhi* in Ayurveda.

As such disease was not explained in ayurvedic texts or classic management of such disease mainly depends on concept of *Anukta Vyadhi* as explained by Charak. There are three types of *Agni* explained in Ayurvedic Text i.e. *Jataragni*[6] *Dhatvagni*[7] and *Bhutagni*.[8] *Dhatvagni* of all *Dhatu* plays an important role in formation *Dhatu* in *Sama*

Matra. which maintain whole-body structure of individual. Therefore, when disease is not explained, management is contemplated mainly depending on *Dosh*, *Dushya*, *Adhisthan* and *Nidan*.[2]

A Case Profile

27 years old, male patient B/B relative and admitted in GACH, Nagpur in Kayachikitsa department had:

Chief Complaint

Patient had *Chankraman Kashtata, Asantulit Chankraman, Ubhaypad Pradeshi Daurbalya, Ubhaypad Pradeshi Vakrata Avum Shosha, Skashta Vak Pravritti, Kvachit Katishula Avum Pindikodvesthan* since 2 years.

Past H/O:H/O Fall at home and trauma over right knee 12 years ago.
No H/O of any systemic illness.

Family History

H/O Grandfather had similar deformity of bilateral foot.

Vaiyaktik Vrittant

Ahar*: *Ahar Pramana: *Sama,*

Pradhana Rasa: *Sarva Rasa,*

Guna: *Ushan, Shita, Snigdha and Tikshna*

Dietetic Habbit: *Samasan, Paryushita Ahar*, *Ushapan* and *Nishapan.*

Type of food: Both Vegetarian and Non-vegetarian (once/month).

***Vihara*:** Sedentary

Occupation: Student

***Vyasan*:** *Avishesh*

Asthavidh Parikshana

Nadi: 98/mi ***Shabda:*** *Skashtavak Pravritti*

Mala: *Prakrit* ***Sparsha:*** *Samshitoshna*

Mutra: *Prakrit* ***Drik:*** *Prakrit*

Akriti: *Krish* ***Jivha:*** *Niram*

***Urah Parikshana*:** No abnormality detected.

***Udar Parikshana*:** No abnormality detected.

Investigations

CBC with ESR: **Hb%:** 14.2 gm/dl **Platelets:** 1.49 lac/cumm

TLC: 7800/cumm **ESR:** 10 mm/hr

BSL Fasting: 84 mg/dl **Post-prandial:** 101mg/dl

Urine Routine and Microscopic: No abnormality detected,

Serum vitamin B12: 163 pg/ml

Nerve Conduction Test

Study showed predominantly sensory axonal polyneuropathy affecting all four limbs.

Motor Nerve Conduction: Conduction Velocities of Arms are > 38 m/s.

MRI Lumbo-Sacral Spine

Mild postero-central bulge of L3-L4 and L4-L5 disc indenting thecal Sac. Schmorl's node involving inferior end plate of L2 vertebral body.

Mild posterior bulge of C3-C4 and C5-C6 disc indenting anterior subarachnoid space.

MRI Brain: No significant abnormality detected.

Vikrititah Parikshana

Dosha* Involved: *Vata: *Vyan*:*Gati Vikriti*

Udana: Skasta Vaka Pravritti

Pitta: *Pachaka Pitta*: *Dhatu Agni Manda*

Kapha: *Shleshaka Kapha*: *Snigdha* and *Slakshna Guna* of *Kapha* was affected because of vitiated *Vata* responsible for defect in structure of articulation.

Dushya* Involved:** *Mansa, Meda, Asthi* and *Majja.**

***Mala* Involved:** No abnormality detected

Srotas* Involved:** *Mansavaha, Medovaha, Asthivaha* and *Majjavaha.**

***Upadhatu* involved:** *Snayu Avum Kandara*

Type of *Srotas Dushti*: *Sanga*

Adhishtana: *Ubhaya Hasta Pada (Karmendriya) Avum VakIndriya.*

Samuthan Vishesh: *Shukrashonita Dushti*

Samprapti

History of family revealed that grandfather was suffering from disease suggested that *Shukrashonita Dusti* was there which is further responsible for *Garbha Vikriti* because of *Shukra Dushti*.[9] *Dhatvagni Mandya was* responsible for *Ama* formation. Specifically, *Mansagni Mandya was there* which is responsible for *Ama* formation at *Mansa Dhatu* results into symptoms of *Ama at Mansa Dhatu* like *Srotorodha* and *Balabhransha*.[10] Formation of *Ama i*s responsible for *Srotas Avrodha* at *Mansa Dhatu* level. As result *Poshakansh* are unable to reach at *Mansa Dhatu* result into *Mansa Dhatu Kshaya. Uttarotar Dhatu Nirmiti* by *Kedar Kulya Nyaya* of *Dhatu Poshan* was also affected because of *Srotas Avrodha*,[11] Results in *Medo-Asthi-Majja Kshaya.* This *Kshaya* at *Dhatu* level is responsible for vitiation of *Vata Dosha.*[12] *Snayu Upadhatu* of *Meda* also get affected, which is *Pitrij Bhava*.[13] *Dhatukshaya* and vitiation of *Vata* are further responsible for symptoms like *Chankraman Kashtata, Asantulit Chankraman, Ubhaypad Pradeshi Daurbalya, Ubhaypad Pradeshi Vakrata Avum Shosha, Skashta Vak Pravritti, Kvachit Katishula Avum Pindikodvesthan.* Disease having such symptoms was not explained in Ayurvedic text. Therefore, termed as *Anukt Vyadhi.*

Principle of Management

Pachana Chikitsa

Initially *Mansa-Asthi-Majja Pachaka Kvath* containing *Guduchi, Amalki, Musta, Triphala, Neem, Mrudvika, Chandana* and *Patola* was given in 20 ml dose twice a day for 18 days.

Bahya Chikitsa

Sarvang Abhyang Svedana with *Tila Taila* was advised throughout the treatment in morning along with *Manyaprustvansha Basti* with *Tila Taila* was also done for same duration. Two settings of *Shasti Shali Svedana (Ubhaya Hast Pad Pradeshi)* with *Bala, Aswagandha* and *Dashmool Siddha Dugdha* for 15 days and 8 days respectively. *Ubhaya Pad Pradeshi Mahamasha Taila Pat Bandhan* once day was done for 15 days. For the complaint of *Skashta Vak Pravriiti Vacha* and *Akarkarabha Churna Jivahapratisarana* with *Madhu* was adviced.

Shodhan Chikitsa

Rajyapana Basti 400ml in morning was administered through anal route for 30 consecutive days i.e. Karma Basti.

Shamana Chikitsa

Bhrihat Vata Chintamani Rasa 1 OD with *Goghrita* for 10 days.

Combination of *Trayodashang Gugglu* 250mg, *Abhrak Bhasma* 25mg and *Chopchinyadi Churna* 2 gm twice a day with *Koshnajala.*

Erandmula Ghanvati 250mg 2 BD with *Koshnajala.*

Brihan* and *Rasayan Chikitsa

Mansa Rasa 40 ml once a day for 30 days.

Ashvagandha and *Shatavari Siddha Kshirpaka* 40 ml twice a day.

Ajamansa Rasayan 5 gm BD with milk.

OBSERVATIONS AND RESULT

Table-1: Table Showing Deep Reflex of patient Before and After Treatment.

S.N	Reflex	Right side BT	Right side AT	Left side BT	Left side AT
1.	Biceps	+	+	+	+
2.	Triceps	+	+	+	+
3.	Brachioradialis	+	+	+	+
4.	Supinator	+	+	+	+
5.	Knee	+	+	+	+
6.	Ankle	+++	+++	+++	+++
7.	Babinski's	Negative	Negative	Negative	Negative

Table-2: Table Showing Muscle Power Grade of patient Before and After Treatment.

S.N	Muscle Power Grade	Right BT	Right AT	Left BT	Left AT
1.	Upper Limb	5/5	5/5	5/5	5/5
2.	Lower Limb	4/5	5/5	5/5	5/5

Table-3: Table Showing Muscle Tone of patient Before and After Treatment.

S.N	Muscle Tone	Right BT	Right AT	Left BT	Left AT
1.	Upper Limb	Normal	Normal	Normal	Normal
2.	Lower Limb	Normal	Normal	Normal	Normal

Table-4: Table Showing time required for 50-meter walk of patient Before and After Treatment.

S.N	Walking Time	
1.	Before Treatment	1 Min 20 sec
2.	After Treatment	50 sec

Table-5: Table Showing Dimension of lower limb of patient Before and After Treatment.

S.N		Right BT	Right AT	Left BT	Left AT
1.	20 cm above knee joint	39 cm	41.5 cm	39 cm	41.5 cm
2.	16 cm above knee joint	37 cm	39 cm	37 cm	39 cm
3.	10cm below knee joint	26 cm	29 cm	26 cm	29 cm
4.	20 cm below knee joint	27 cm	28 cm	27 cm	28 cm
5.	5 cm above ankle joint	18 cm	18cm	18 cm	18 cm
6.	At ankle joint	23 cm	24 cm	23 cm	24 cm
7.	5 cm below ankle joint	25 cm	25 cm	25 cm	25 cm
8.	10 cm below ankle joint	22 cm	22 cm	22 cm	22 cm

Table-6: Table Showing assessment of weight and BMI of patient Before and After Treatment.

S.N	Date	Weight (Kg)	BMI
1.	30/3/2019 (On zero day)	43	14.53
2.	9/4/2019 (11th day)	43	14.53
3.	16/4/2019 (18th day)	46	15.55
4.	30/4/2019 (32st day)	46.5	15.72
5.	5/5/2019 (37th day)	47.5	16.06

Table-7: Table Showing assessment of sensory function test of patient Before and After Treatment.

S.N	Sensory function Test	Right Upper limb	Right Lower limb	Left Upper limb	Right Lower limb
1.	Tactile Localization	Normal	Normal	Normal	Normal
2.	Tactile Discrimination	Normal	Normal	Normal	Normal
3.	Tactile Stereognosis	Normal	Normal	Normal	Normal

Table-8: Table Showing assessment of Motor Nerve Conduction Test Before Treatment and After Treatment.

S.N	Before Treatment Nerve conduction test	After Treatment Nerve conduction test
	Predominantly sensory axonal polyneuropathy affecting all four limbs.	Mild predominantly sensory axonal polyneuropathy affecting all four limbs. (Compare to previous study there is improvement in sensory nerve action potentials and No Change in motor nerve action potentials)

Table-9: Table Showing assessment of Motar Nerve Conduction Test Before Treatment and After Treatment.

S.N	Nerve and Site	BT NCV	AT NCV
1.	Right median Nerve (Elbow)	55 m/s	52 m/s
2.	Right ulnar Nerve (Below Elbow)	60 m/s	61 m/s
3.	Left median Nerve (Elbow)	54 m/s	49 m/s
4.	Left ulnar Nerve (Below Elbow)	59 m/s	55 m/s
5.	Right peroneal Nerve (Head of fibula)	45 m/s	42 m/s
6.	Right tibial nerve (Popliteal fossa)	39 m/s	40 m/s
7.	Left peroneal Nerve (Head of fibula)	43 m/s	43 m/s
8.	Left tibial nerve (Popliteal fossa)	43 m/s	39 m/s

Table-10: Table Showing assessment of Sensory Nerve Conduction Test Before Treatment and After Treatment.

S.N	Nerve and Site	BT NCV	AT NCV
1.	Right Median Nerve (Wrist)	41 m/s	48 m/s
2.	Left Median Nerve (Wrist)	42 m/s	46 m/s
3.	Left Ulnar Nerve (Wrist)	42 m/s	40 m/s
4.	Right Sural Nerve (Lower Leg)	36 m/s	45 m/s
5.	Left Sural Nerve (Lower Leg)	37 m/s	40 m/s

Table-1 shows that there is no change in Deep reflex of patient. **Table-2** shows that muscle power grade of right lower limb was improved from 4/5 to 5/5. **Table-3** highlighted that muscle tone of all four limbs are normal **Table-4** shows improvement in 50-meter walking time from 1 min 20 sec to 50 sec in this case. **Table-5** shows that there is significant increase in dimensions of bilateral lower limb at different levels i.e. 2.5 cm increased in muscle mass was observed at 20 cm above knee joint, 2 cm at 16 cm above knee joint, 3 cm at 10 cm below knee joint, 1 cm at 20 cm below knee joint and at ankle joint. **Table-6** shows weight gain of patient from 43 kg to 47.5 kg i.e. 4.5 kg weight increased after management. **Table-7** manifest that sensory function test is normal before and after treatment. **Table-8** showed that compare to previous study of nerve conduction there is obvious improvement in sensory nerve action potential. **Table-9** stated that there was no significant change in motor nerve action potential. **Table-10** shows improvement in Sensory nerve conduction velocities after treatment.

DISCUSSION

According to Ayurveda, CMT can be corelated with *Anukta Vyadhi*. The treatment of such diseases can be done by considering the vitiated status of *Dosha, Dushya, Adhishtan* and

Nidan.[2] In this case before starting the management status of *Agni* was taken into consideration because all diseases are because of *Agnimandya* of individual.[14] In this case there is gradually wasting of bilateral lower limbs were seen which suggest that there was *Vikriti in Mansagni* i.e. *Manda Mansagni* was there responsible for *Ubhay Pad Pradeshi Shosha*. According to concept of *Dhatu Poshan Nyaya* i.e. *Kedarkulya Nyaya.*[11] Plung in *Mansa Dhatu* was liable for *Uttarotar Dhatukshaya.*[15] As result symptoms of *Meda, Asthi* and *Majja Kshaya* was observed in patient. To leaden the *Dhatwagni Vikriti Pachana Chikitsa* was advocated. Therefore, *Mansa-Asthi-Majja Pachaka Kvath* was used.[16] In Ayurveda *Dhatukshaya* is responsible for Vitiation of *Vata*.[13] Hence, *Mansakshaya* induced *Vataprakopa* further aggrevated *Meda-Asthi-Majja* Kshaya. Therefore, line of management for *Meda-Asthi-Majja Kshaya* and *Vatashamana* was planned in the form of *Bhaya Chikitsa, Shamana* and *Shodhana Chikitsa.* While all these management was adviced after understanding involvement of *Agni, Dosha, Dushya* and *Srotas.* Basic principles of Ayurveda were used to treat the case.

In *Bhaya Chikitsa Prustavansha-Manya Basti* procedure with *Tila Taila* was advised for symptoms of *Katishula* develop because of *Asthikshaya* and *Vataprakopa.* This procedure provides local *Snehana* and *Svedana* at *Prustavansha* and *Manya Pradesh.* Which alleviate vitiated *Vata* by lowering *Shita* and *Ruksha Guna* of *Vata*[17] by *Ushana* and *Snigdha Guna* of luke warm *Tila Taila*. The concept of *Guna Vishesh* was applied here.[18] Charak stated that *Apatarpanjanya Vyadhi* are always treated by *Santarpana / Brihan Chikitsa*.[19] Hence, For *Mansakshaya* and *Vataprakopa Shasti Shali Pinda Sveda* with *Aswagandha, Bala* and *Dashmoola Siddha Kshira* was done. Which help to lower Vitiated *Vata* at *Kshaka* by opposite and Similar *Guna* of *Dravya* used in this procedure.[18] *Aswagandha, Bala* and *Kshira* help to improve *Mansakshaya. Dashmool* is used as *Vatahara* drug which alleviate *Vata Prakopa.*

For *Shosha, Sthabdata* and *Sankoch* situated at *Ubhaya Pad Pradesh Patabandha* of *Koshna Mahamasha Taila* was applied. As we know continuous *Snehana* and *Svedana* can even bend hard wooden stick i.e. *Kashta*.[20] Therefore, *Patabandha* with *Koshna Mahamasha Taila* indicated in *Vatavyadi* help to reduce *Shosha* (atropic changes) and *Sankoch.*[21]

Vacha and *Akarkarabha Churna Jivhapratisarana* with *Madhu* was adviced for complaints of *Skasta Vakapravritti.* Harit stated *Vacha* as choice of drug in *Mukharoga.*[22] Similarly, in

Dhanvantari Nighantu it was used to treat diseases of *Kanta Roga*. *Akarkarabha* used as *Vatashamak*.[23] Therefore, both drugs are helpful to treat Difficulty in talk.

As we know, *Vata Dosha* is responsible for formation of diseases of *Kshakhagata, Koshtagata, Maramaurdhva* and all parts of the body.[24] *Vata Dosha* is responsible for *Shosha, Sthabdhata* and *Sankoch*.[25] Therefore, bestowed management explained for *Vata Dosha* in ayurvedic text was *Basti*.[26] Therefore, *Rajyapana Basti* administered in patient which help to increase *Mansa, Agni* and *Bala* of patient.[27] *Sarvang Abhyang Svedana* was adviced as *Poorvakarma* of *Basti*. Where *Abhyang* help to lower vitiated *Vata*, brings *Mardavta* in body and *Anulomana* of *Vata* and remove *Mala* responsible for obstruction.[28] *Svedana* help to liquify the *Lina Dosha* and help to remove it.[29]

Along with *Shodhana Chikitsa* i.e. *Basti*. *Shamana Chikitsa* was also adviced to patient. *Bhrihatvata Chintamani Rasa* is drug used for *Vata Roga* and *Pitta Roga*.[30] Hence, helpful in alleviating vitiated *Vata* as result helpful for maintaining equilibrium of *Dhatvagni*.[31]

Some drugs like *Trayodashang Gugglu, Shuddha Abhrak* and *Chopchinyadi Churna* was used in combination. Where *Trayodashang Gugglu* help in *Katigraha, Sthabdhata* in both lower limbs, *Vata* situated in *Sandhi* and *Asthi* and *Maja, Snayugat Vata*.[32]

Ayurved Prakash and Rasratnasamuchhaya advocated *Abhrak Bhasma* in *Shosha, Kshaya* and *Madagni* condition.[33] Therefore, it was used in this case for *Roga Vinashanarth*. *Chopchinyadi Churna* is *Vata Shamak* used to treat pain and inflammation at joints.[34]

CONCLUSION

The combination of this Ayurvedic treatment can be helpful in treating the cases of CMT. Improvement in Dimension of lower limbs, walking time, increase in weight and muscle power grade and sensory axonal improvement in nerve conduction test suggestive of effect of given therapy is definitely effective in management of such cases of Charchot Marrie Tooth disease (Hereditary Neuropathy). However, the trial was on only one patient and multiple such cases can be taken for study by similar line of management.

Before Treatment and After Treatment Images

Image of Curling of Left Foot (Before Treatment)

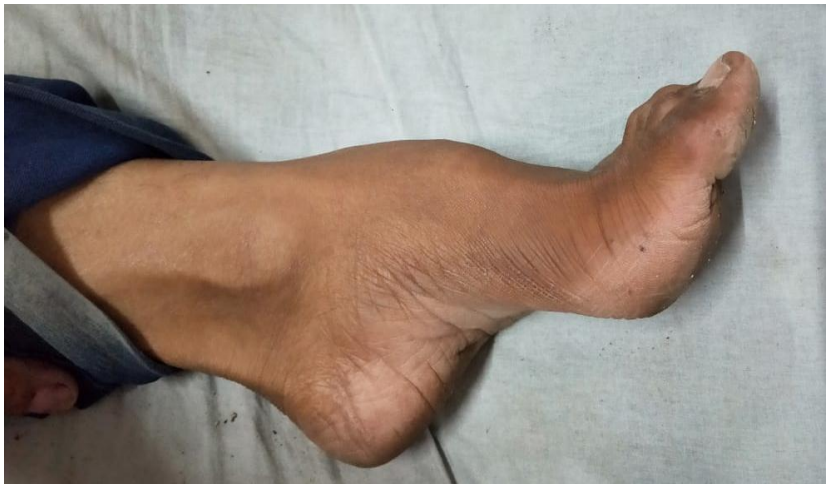


Image of Curling of Left Foot (After Treatment)

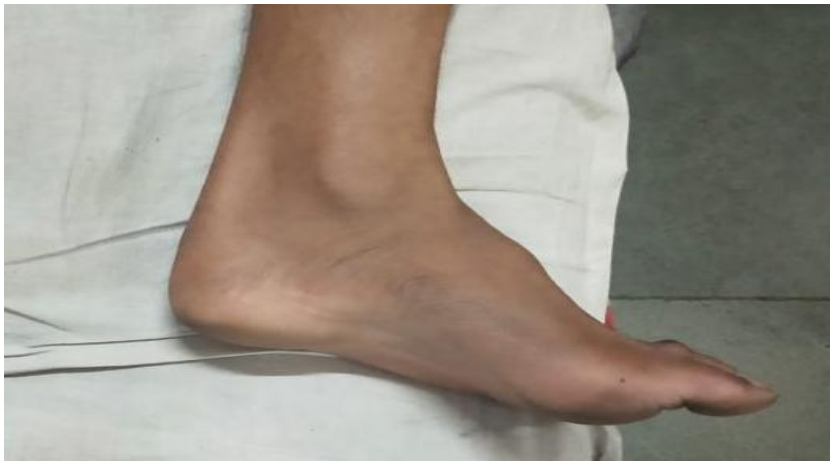


Image of Curling of Right Foot (BeforeTreatment)

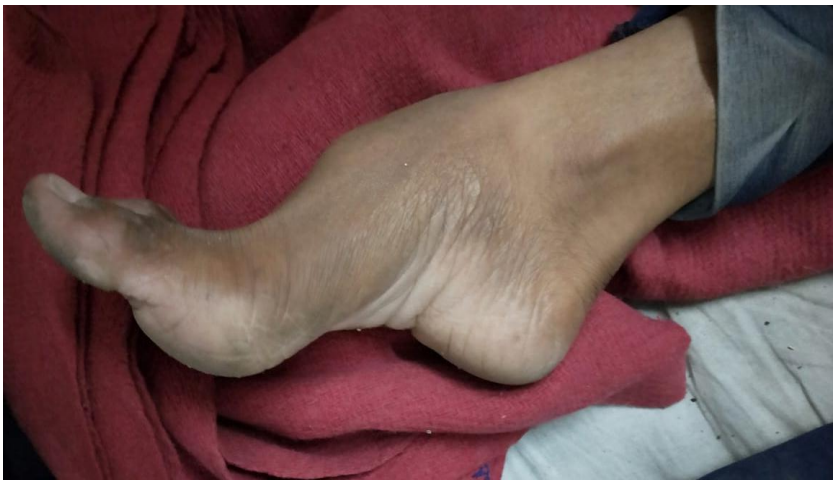


Image of Curling of Right Foot (After Treatment)

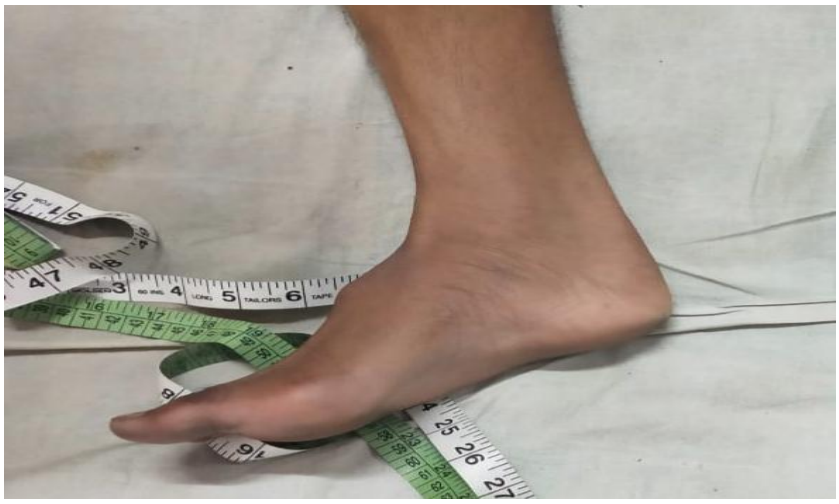
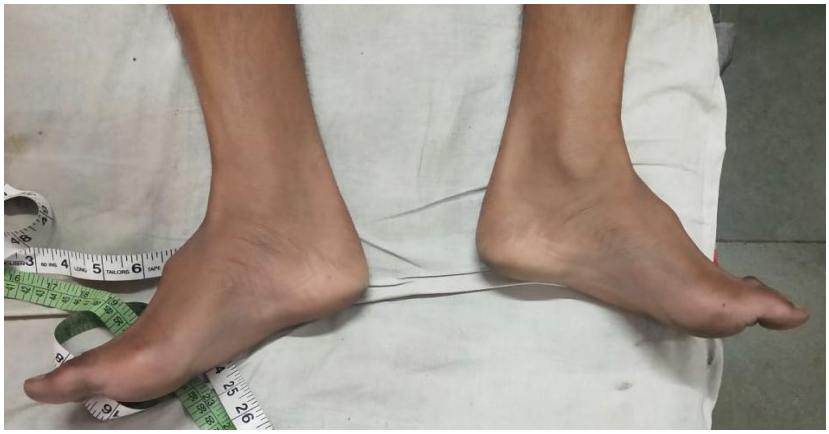


Image of bilateral foot after treatment



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