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ABSTRACT

Torticollis in Latin word means twisted neck and was first defined by Tubby in 1912. [1] Torticollis is the postural deformity of head and neck, which may be acquired or congenital. Acquired Torticollis may be due to trauma, inflammatory, psychogenic or neoplastic. Congenital muscular Torticollis is a postural deformity of head and neck detected at birth or shortly after birth, primarily resulting from unilateral fibrosis & shortening of the Sternocleidomastoid muscle. Torticollis is also known as twisted neck. [2,3,4] The most common Physiotherapy treatment were passive stretch, handling advice, facilitation of neck muscle strength and facilitation of active cervical ROM.

Key words: Torticollis, Cervical dystonia, Stretching, facilitation

INTRODUCTION

Torticollis is the postural deformity of head and neck, which may be acquired or congenital. Acquired Torticollis may be due to trauma, inflammatory, psychogenic or neoplastic. Congenital muscular Torticollis is a postural deformity of head and neck detected at birth or shortly after birth, primarily resulting from unilateral fibrosis & shortening of the Sternocleidomastoid muscle. Torticollis is also known as twisted neck. [2,3,4]

Congenital muscular torticollis (CMT) is the third most common musculoskeletal abnormality in infants next to hip dysplasia and clubfoot.[2,5] Congenital muscular torticollis is a musculoskeletal deformity, observed in infancy, characterized by unilateral contracture, unilateral shortening of the sternocleidomastoid muscle resulting in lateral inclination of the neck associated with contralateral torsion.[2,6,7,8,9] Cervical dystonia is also known as acquired torticollis, is a chronic neurologic disorder characterized by involuntary patterned contractions of cervical musculature resulting in abnormal movements or postural changes of the head, neck, and shoulders.[10]

CMT is a result of shortening or excessive contraction of the sternocleidomastoid muscle (SCM) with limited range of motion (ROM) in both rotation and lateral flexion of the neck and an imbalance of muscle function around the neck.[11,12,13] The shortening of Sternocleidomastoid muscle results in traction of mastoid process toward the sternoclavicular joint.[14] The head is therefore tilted towards the involved sternocleidomastoid muscle and the chin is rotated in opposite direction, this condition is sometime called “Wryneck”. (15) Infants born prematurely also have a greater incidence of skull deformity attributable to molding after birth. Associated torticollis or “Wryneck” may occur because of hemorrhage (within the sternocleidomastoid muscle) and/or subsequent scarring within the sternocleidomastoid muscle, or muscle shortening caused by persistent, unidirectional positioning and limited neck motion. The term wry neck is derived from old and middle English, meaning to wind or to twist. Greek and Roman authors named this condition caput obstipurn to indicate the abnormal posturing of the head and, in the German medical literature, this term has been used synonymously with torticollis.

Jean Froissart, in the 14th century, coined the term au tort col from the Latin words tortus, meaning twisted, and collum, meaning neck. From this, the more common term, torticollis, is derived. (16) The term plagiocephaly is a Greek derivative meaning "oblique head." Most skull deformities present at birth are the result of in utero or intrapartum molding. Associated conditions involve uterine constraint, especially in cases of multiple birth infants, and birth injury associated with forceps or vacuum-assisted delivery.[17,18] Infants born prematurely also have a greater incidence of skull deformity attributable to molding after birth. Most of these deformities improve spontaneously during the first few months of life if the infant does not rest his or her head on the flattened area of the skull. If the infant continues to rest his or her head on the flattened side of the occipital, an initially occipital plagiocephalic deformity may be perpetuated or worsened by gravitational forces.[19]

CLASSIFICATION OF TORTICOLLIS (20)

Type	Nonparoxysmal (Nondynamic)	Paroxysmal (Dynamic)
1	1.Congenital muscular torticollis Intrauterine constraint (eg, breech presentation) Birth trauma (eg, difficult delivery)	1 Benign paroxysmal torticollis
2	2. Osseous torticollis Congenital (eg, Klippel-Feil syndrome) Traumatic (eg, cervical vertebra fracture) Inflammatory (eg, atlantoaxial rotary subluxation)	2. Spasmodic (cervical dystonia) Primary Secondary (eg, Huntington disease, Wilson disease)
3	3. Central nervous system/peripheral nervous system torticollis Brain Posterior fossa (eg, brainstem or cerebellar tumor)	3. Sandifer syndrome (gastroesophageal reflux)
4	4. Ocular torticollis Superior oblique muscle palsy Other ocular deviations (eg, paralytic horizontal strabismus) Spasmus nutans	4. Drug-induced torticollis (eg, neuroleptics)
5	5. Nonmuscular, soft tissue torticollis Infectious (eg, retropharyngeal abscess)	5. Torticollis from increased intracranial pressure (eg, pseudotumor cerebri)

CMT is further subdivided into 3 groups on the basis of clinical presentation.(21,22,23)

Congenital muscular torticollis (CMT)		
Sternomastiod tumor	Muscular torticollis	Postural torticollis
It is the most severe form	When neck deformity is associated with muscle tightness and	The mildest form, with postural deformity in the neck but without restricted
It has a fibrotic mass in the SCM	It has not a fibrotic mass in the SCM	No fibrotic mass present
Passive ROM of neck is limited	Passive ROM is restriction.	Passive ROM or mass/tightness in the SCM.
About 8% of cases require invasive intervention if not treated early	About 3% of cases require invasive interventions if not treated early.	Shorted-term conservative interventions are used to treat infants because resolution is quick and occasionally spontaneous. (24)

Anatomy of Sternocleidomastoid muscle (26):

ORIGIN	INSERTION	NERVE SUPPLY	ACTIONS
The sternal head is tendinous and arise from superolateral part of the front of the manubrium.	By a thick tendon into the lateral surface of the mastoid process, from its tip to its superior border.	The spinal accessory nerve provides the motor supply. It passes through the muscle.	A)When one muscle contracts: It turns the chin to the opposite side, It can also tilt the head towards the shoulder.
The clavicular head is musculo-tendinous and arise from the medial one third of the superior surface of the clavicle.	By a thin aponeurosis into the lateral half of the superior nuchal line of the occipital bone.	BLOOD SUPPLY: One branch each from superior thyroid artery and suprascapular artery and two branches from the occipital artery veins follow the arteries. Veins follow the arteries.	(B)When both muscles contract together: They draw the head forwards, as in eating and in lifting the head from a pillow, With the longus colli, they flex the neck against resistance, The reverse action helps in forced inspiration.

Pathophysiology [25]: The muscle has been replaced by dense fibrous tissue. CMT may be caused by intrauterine or perinatal SCM muscle compartment syndrome. Ischemia and edema within the SCM muscle compartment can result from flexion with lateral bending and rotation of the head and neck, leading to SCM muscle trauma.

Biomechanics: [27]

SCM imbalance present with an intermittent head tilt, unlike the patient with CMT who present with a fixed head tilt due to restricted SCM. Intermittent means that, when the child was examined in either upright or supine position, the head is tilt fluctuated in severity, not appearing to remain fixed at a specific angle. SCM imbalance also persistently favored rotating the head to one side while supine and upright, appearing unable or unwilling to actively rotate away from it, even if a strong stimulus was presented to the other side.

Aetiology: Although evidence about CMT aetiology is vague it is postulated that fetal position abnormalities, intrauterine or perinatal compartment syndrome and birth trauma ensuing a difficult delivery embody the main causes.[28,29]

Other possible causes encountered are hereditary and venous or arterial occlusion which may create fibrous tissue within the Sternocleidomastoid.[30,31]

CAUSE OF TORTICOLLIS:[32]

1.	Congenital	Sterno-mastoid tumour
2.	Infection	Tonsillitis Atlanto-axial infection Labyrinthitis
3.	Reflex spasm	Acute disc prolapsed(cervical)
4.	Neurogenic	Spasmodic condition Paralytic condition
5.	Ocular	Compensation for squints
6.	Other	Spasmodic Torticollis Rheumatoid arthritis

EPIDEMIOLOGY:

The incidence of congenital muscular torticollis in newborn infants has ranged from 0.3% to 2%.[33]

In a study of 288 pediatric patients with torticollis, 82% were congenital muscular and 18% non-muscular. [34] In a prospective outcome study of manual stretching in 821 consecutive patients with congenital muscular torticollis, 55% had a sternocleidomastoid tumor, 34% were muscular (thickening and tightness of the sternocleidomastoid) and 11% were postural torticollis (head tilt but no tumor, thickening, or tightness of the sternocleidomastoid). [35]

The reported incidence is 0.4-2.0% however a recent study indicates that it might be higher.[36] Congenital torticollis occurs in 1-2% of the population, clinically presented as a lateral inclination of the head accompanied by the rotation of the chin to the opposite side.[37]

The incidence of CMT is one in every 300 live births.[38] The exact pathophysiology and etiology of Sternocleidomastoid impairment in CMT is still unknown.[15] In most cases, right side is more frequently affected, by a factor of three to one, and it is more prevalent in males than in females.[37,39, 40]

Clinical feature:[41]

Clinically, in infants with CMT, the head is typically tilted toward the side of the affected muscle and rotated toward the opposite side.

In CMT, skull and facial asymmetry (in addition to plagiocephaly) may be present.

Jaw asymmetry with mandible hypoplasia may be the first indication that CMT is present, and some mothers notice that infants with CMT have difficulty breastfeeding equally well from both breasts.

Ears are asymmetric as well, with the ear on the side of the torticollis, or affected SCM muscle, often smaller and the ear opposite the torticollis displaced forward with the contra-lateral occipital flattening.

Differential diagnosis:[42]

Congenital	Muscular torticollis. C1 –C2 articular malformation. Atlantoaxial dislocation Rotatory subluxation Klippel-Feil syndrome Sprengel`s deformity. Congenital postural torticollis-transient, Present at birth, secondary to abnormal fetal position
Acquired	Trauma Nasopharyngeal" torticollis Grisel`s disease/C1-C2 subluxation Drugs Sandifer`s syndrome Gastroesophageal reflux Psychiatric
Neurologic	Syringomyelia Dystonia Herniated cervical disks Any posterior fossa pathologic finding Spastic torticollis
Ocular strabismus/paresis of extraocular movements vestibular	Congenital nystagmus Paroxysmal torticollis of infancy Episodic head tilt, vomiting, pallor, and dizziness

DIAGNOSIS

- Diagnosis is based mainly on past medical history and clinical examination of the infant. A meticulous prenatal history record is essential and detects complicated labor and the coexistence of previous birth trauma such as clavicular fracture. The presence of perinatal asphyxia, jaundice, seizures, medication, gastroesophageal reflux disease (GERD) or Sandifer syndrome are also recorded.[44]

A firm painless not tender pseudotumor mass is typically palpable in the first few weeks of life.[45,46]

This lump can affect both the sternal and clavicular parts of the muscle.[47] This endomysial mass is consisted of fibrotic issue linked with deposition of collagen and migration of fibroblasts around the atrophic sternocleidomastoid fibers. [27]

Clinical examination includes evaluation of neck range of motion, and thorough neurological assessment. The type of deformation is also investigated, as well as the combination of flexion and rotation, whether the deformation is rigid or flexible, and whether it can be corrected by the child itself.[45,47,48]

Associated congenital musculo-skeletal conditions i.e. hip dysplasia is also investigated. Ophthalmological examination may reveal extra ocular muscle imbalance as the causing factor of torticollis.[49]

Ultrasonographic imaging is a useful diagnostic tool with important diagnostic and prognostic application.[50] This method is characterized by high sensitivity and specificity of 95.83% and 83.33%, respectively. The ultrasonographic findings vary in accordance with different CMT stages.[51]

Magnetic resonance imaging (MRI) is a modern radiologic examination with increasing role in CMT diagnosis. In a recent study MRI findings have been found to be correlated with histopathological findings.[52]

Physical therapy Treatment:

The most common treatment were passive stretch, handling advice, facilitation of neck muscle strength and facilitation of active cervical ROM.[53]

The parents were taught a stretching program to increase the infant's range of neck rotation to the affected side and neck lateral flexion to the contralateral side. Two people were required to stretch the infant's neck. One person secured the infant's shoulders, stabilizing the clavicle, while the other person did the stretching. [54]

Passive stretching of SCM before the age of 12 month is the most effective mode of physical therapy.[55] Other physical

treatment included massage of tight neck muscles and subcutaneous tissues which increase pain free range of motion, joint mobilization, myofascial release, therapeutic taping. [47]

Postoperative Rehabilitation

Phase 1: Inpatient Acute Care:

Post operative Days 1 to 2: The patient is usually discharged from the hospital on postoperative day 1 when the patient and caregivers are independent with all mobility and orthotic management, and pain is well controlled .Before discharge, the therapist reviews the outpatient plan of care, emphasizing the importance of compliance with therapy, and daily home exercise program (HEP). The therapist therefore reviews safe transfers, gait, and stair climbing.

Phase 2: Outpatient Rehabilitation: ROM, Strength, and Midline:

Postoperative Day 3 to Week 8: The subacute phase of rehabilitation begins on postoperative day 3 or 4 at follow-up with the physician. Initially physical therapy is recommended 3 times per week with a daily home exercise program.

The goals of this phase are to optimize cervical passive and active ROM, maximize functional strength of the cervical musculature, and develop midline awareness and control.

The therapist works to advance active head righting reactions and improve the patient's proprioceptive awareness. It is important that all stretching is performed gently and the child is monitored for signs of discomfort during therapeutic exercise and functional mobility. In addition, the therapist needs to be aware of compensatory movement patterns and postures. The family should be instructed to monitor the incision site for signs of irritation or infection.

The child's HEP consists of active and passive cervical rotation and lateral flexion, active strengthening of the uninvolved cervical lateral flexors, and scar massage (once incision is healed). It should be performed 4 to 6 times per day.

Therapeutic treatment recommendations include the same 5 first-choice interventions recommended by the CPG: neck passive range of motion (PROM), neck and trunk active range of motion, development of symmetrical movement, environmental adaptations, and caregiver education.

In addition, active assistive ROM and cervical and core-strengthening therapeutic exercises are important. Development of symmetrical movement in older children with well-established compensations may be challenging. Visual-motor activities, therapeutic ball activities, scapula stabilization, upper extremity weight bearing, and bimanual activities are used with a focus on the child incorporating tactile, visual, and vestibular feedback. Additional intervention strategies focus on facilitation of midline head orientation, affected upper extremity strength and fine motor skills, bimanual activities, and visual-motor exercises combined with vestibular activity.

Criteria for Advancement

At 6 to 8 weeks post surgery, the child is re-examined by the physician. If progress is satisfactory, the wearing schedule of the brace is decreased to night use. The child is ready to progress to phase 3 of rehabilitation when his or her active and passive ROM measures are within 5° bilaterally and cervical lateral flexion strength is within 1 grade bilaterally on the Muscle function scale.

Phase III: Outpatient Rehabilitation: Functional Maintenance of Midline Weeks 8 to 24

The goals for phase III are to achieve equal active and passive ROM bilaterally, establish symmetrical head righting in all positions, maintain head in midline in all developmental positions at least 95% of time, demonstrate symmetrical gross motor skills, and have the child and caregivers independent with an updated home exercise program (HEP). Therapists must monitor for recurrence of the head tilt upon discharge of the pinless halo (typically

around 10-12 weeks post-surgery) and habitual compensatory movement patterns.

Treatment recommendations include continuation of activities from phase II with the addition of kinesiotaping and a Tubular Orthosis for Torticollis (TOT) collar as needed to promote sustained midline head orientation. To address persistent motor asymmetries, activities from phase II are progressed to be more challenging and complex to meet specific needs of the child. [55]

Criteria for Discharge

The child is ready for discharge from physical therapy when he or she has achieved symmetrical active and passive cervical ROM, symmetrical head righting, and symmetrical cervical lateral flexion muscle strength. The child must demonstrate the ability to maintain head in midline in all developmental positions at least 95% of time and perform age-appropriate, symmetrical gross motor skills. Finally, the family must understand how to monitor for signs of regression and be independent with HEP.[55]

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