



Traumatic Myositis Ossificans: Treatment Protocol and Review of Literature.

Dental Science

Khadar Vali Shaik Assistant Professor, Department of Oral and Maxillofacial Surgery, MNR Dental College and Hospital Corresponding author

Haripriya Chari MDS, Assistant Professor, Department of Oral and Maxillofacial Surgery

ABSTRACT

Myositis ossificans traumatica (MOT) is known mostly in the orthopedic literature as non-neoplastic, heterotopic bone formation within muscle or fascia, presumably due to acute trauma, burns, surgical manipulation, or repeated injury.

Aim of the study: To review the literature available, on Myositis ossificans in the head and neck region and to formulate a protocol for its treatment.

Materials and methods: We have reviewed the literature through random search in Pubmed, for MOT in Head and neck and as well as in the orthopedic literature

Summary: we suggest early surgical intervention and prevention of further calcifications with drugs like indomethacin along with active physiotherapy to maintain jaw movements.

KEYWORDS:

Myositis ossificans traumatica, Temporalis muscle, medial pterygoid muscle, muscles of mastication.

Introduction:

Myositis ossificans is a rare autosomal dominant condition, which is characterized by bone deposition in soft tissues. Myositis ossificans traumatica (MOT) is defined as a heterotrophic, non-neoplastic proliferation of bone in muscle and other soft tissues previously exposed to trauma and hematoma.^{1,2,3,4} MOT is also called as Traumatic Myositis Ossificans, Localized Myositis Ossificans, Myositis Ossificans Circumscripta and Fibrous Dysplasia Ossificans Circumscripta.⁵ Guy Patin in 1692, first described an extra skeletal bone formation, in literature and Von Dusche in 1868, named it as, Myositis Ossificans Traumatica.^{3,6} This condition has been published elaborately in the orthopaedic literature suggesting of its common occurrence in the extremities. Common examples are Horse Riders Bone, Cavalrymen's Osseous Plate on the outer thigh and Infantrymen's drill bone on the deltoid. These indicate that the commonly involved muscles are quadriceps femoris, brachialis anticus, adductor muscles of the thigh and deltoid muscle.^{7,8,9}

In Maxillofacial region, its occurrence is very rare, though it is the most commonly involved area. There are only 26 reported cases of MOT involving muscles of mastication over the past 10 decades in the literature, out of which, two-thirds affected masseter, suggesting its involvement more commonly than the rest, followed by lateral and medial pterygoid muscles. Temporalis muscle is very rarely affected and only three cases have been reported in literature, where it is involved solely.

Aetiopathogenesis:

Clinically, three types of Myositis Ossificans have been described in the literature. They are congenital, idiopathic and traumatic (Table - 1).^{1,10-13} Of the three types mentioned above, a congenital variant, known as Myositis Ossificans Progressiva, also called as Munchmeyer's disease, is a rare autosomal dominant disease, characterized by multiple congenital malformations and osseous metaplasia of the muscles and connective tissue, leading to progressive ossification of relevant areas. Its main aetiopathology is considered to be, an over expression of the gene coding for the bone morphogenetic protein BMP4. Progression of the disease is manifested with development of numerous outbreaks of heterotopic ossification in the muscles, tendons and fascia. Striated muscles are the commonly affected group. In this condition, death may occur due to calcification of respiratory muscles.⁶

Myositis Ossificans Traumatica is associated with a history of trauma and has been named depending on the area of involvement.^{7,8,9,14} Two classifications were proposed to classify MOT one by Arrington¹⁵ (Table - 2) mentioning involvement of stalk, periosteal, broad base muscle and other by Mestan and Bassano⁸ (Table - 3) involving paraosteal, periosteal and extra osseous.

Pathogenesis of the condition remains unclear. Carey¹⁰ proposed four

main theories for its development:

- 1) Displacement of bony fragments into the soft tissues with subsequent proliferation.
- 2) Detachment of periosteal fragments into the surrounding tissues with proliferation of osteoprogenitor cells.
- 3) Migration of sub periosteal osteoprogenitor cells into surrounding soft tissues, through periosteal perforation induced by trauma.
- 4) Metaplasia of extra osseous cells exposed to Bone Morphogenic Proteins (BMP) derived from the lysis of bone fragments, displaced within the soft tissue during traumatic injury.

Among the proposed theories, a widely accepted one stated by Arima et al,¹⁷ says that, autolysis of scattered bone fragments releases Bone Morphogenic Proteins (BMP), inducing the differentiation of perivascular mesenchymal cells into muscular tissue, resulting in a relatively homogenous bony mass. They also found that the interval between trauma and first detection of calcified mass ranged from 3 weeks to more than 20 years.

In head and neck region, the clinical presentation, especially, when masticatory muscles are involved, is trismus. Usually mouth opening is limited, ranging from 1 cm to complete trismus. There will be a history of trauma especially, blunt in nature in these presentations. The limitation of jaw movements, is not sudden, instead, it is progressive. Examination usually reveals a non-tender swelling and the affected muscles are firm to hard in consistency at rest.

Ryan et al,¹⁶ suggests that, blunt trauma creates a compression wave travelling through soft tissue, crushing the deepest muscle against the bone. The force is transmitted through the fluid compartment of all the layers of muscle, but the damage usually occurs in the layer that is next to the bone.

There are several steps following an injury that leads to the development of MOT. There is cellular damage causing necrotic debris, which is subsequently removed through invasion of histiocytes. Fibroblasts from the endomysium then assail the injured cells and mesenchymal cells thereafter begin to proliferate. The fibroblasts and mesenchymal cells produce osteoid and chondroid tissue that lays down the groundwork for the formation of bone, within the damaged tissue. This can occur as early as 4 to 5 days after the injury has occurred.^{18,19}

Radiological evaluation:

Shirkhoda et al,²² described four different phases of MOT.

- 1) The initial phase features capillary and mesenchymal cell proliferation at the periphery of the wound. Due to lack of calcification, this phase is inconspicuous on radiographic examination.
- 2) Initial phase of bone formation (1 – 2 weeks).
- 3) Intermediate phase (4 weeks) and a

4) Late phase (6 weeks).

The mature phase appears as a central radiolucency surrounded by a rim of bone. Excision of MOT is best performed during the mature phase of the lesion, when it is well – delineated from the surrounding skeletal muscles.

Radiographically, a varied presentation can be noticed in plain radiographs depending on the maturity of the calcifications. Amorphous calcifications are found in the early lesions and dense, well-circumscribed calcifications are seen in mature lesions. A computed tomography shows similar presentation. Typically, the calcifications are found away from normal bone, mostly confined within the muscle mass. However, mature and old lesions can appear attached to normal bone by a stalk.^{20,21}

Danchik et al,⁷ suggested that additional imaging modalities like Computer tomography (CT), Magnetic Resonance Imaging (MRI), Ultra Sonography and Bone scan could be helpful in diagnosis. Zeanah and Hudson,²³ stated that CT scan accurately demonstrates characteristics of well-defined peripheral shell of bone, its location and extent of the lesion into the surrounding normal tissues. Kirkpatrick et al,²⁴ preferred ultrasound examination in early cases of suspected MOT, because they suggest that, early cases do not show positive findings in radiography and in such cases, an ultrasonography can be helpful.

Histological Evaluation:

Histologically MOT classically presents a Zonal Architecture with peripheral ossification and central cellular region. The outer zone is composed of mature lamellar bone with active osteoclasts. An intermediate zone consists of cartilage or woven bone and active osteoblasts. Central zone is usually composed of loose fibro vascular tissue containing spindle cells and giant mesenchymal cells. This zonal presentation is similar to osteosarcoma and should be differentiated by history and clinical findings.²⁰

Differential diagnosis:

MOT, since long has been confused with osteosarcoma by many clinicians. Depending on the clinical, radiological and histological presentations, the reasonable differential diagnosis includes extra osseous osteosarcoma, synovial osteosarcoma, osteochondroma, posttraumatic periostitis, osteomyelitis and tumoural calcinosis.²⁵ There had been reports of osteosarcoma that had developed from traumatic myositis ossificans, but they are very rare. Aboulafia A J,²⁶ reported a case of osteosarcoma arising from heterotopic ossification that occurred, because of an electrical burn 10 years previous. MOT can be differentiated from osteosarcoma by history of trauma, site of the lesion, nature of pain and alkaline phosphatase levels briefly discussed in table - 4.

Treatment:

There had been a difference of opinion in management in orthopaedic literature and maxillofacial literature. MOT is generally considered a self-limiting condition and can have spontaneous resolution.^{1,27} Hamida et al,²⁸ reports that, without treatment the radiological and clinical findings will stabilize and/or resolve within 1.5 to 3 years following the onset of symptoms. Danchik et al,⁷ notes that, within 4 to 6 months the lesions stabilized. Full re-absorption can take place especially in lesions occurring within the muscle belly, whereas lesions located near an origin or insertion of a muscle are less likely to reabsorb and may result in functional impairment. Parikh et al,³ reported that full reabsorption is more likely to occur in small, upper extremity lesions compared to a larger lesion located in the lower extremity. Le Roux et al,²⁵ noted that smaller upper extremity lesions were more likely to reabsorb except for those lesions occurring at the elbow. There are several management options that are used to treat MOT: aspiration of the hematoma, usage of multiple drugs like bisphosphate, NSAIDS, acetic acid iontophoresis and the last option being surgery. Various treatment options are discussed briefly in table - 5.

Prevention: There is little to be done once the process of MOT has been initiated. However, proper management of the risk factors may decrease the possibility of its onset. According to Larson et al,²⁹ these include:

1. Early consideration of MOT as a differential diagnosis, at the same time proper management of the hematoma to prevent

calcification.

2. Protection from recurrent injury to the injured area and advice against forced movements. Some authors recommend extra protection 3 to 6 months post injury.
3. Educating patients on the causes and consequences of MOT and importance of early treatment.

Conservative management include mouth opening without force and medical management with etidronate, alendronate, NSAIDS especially indomethacin, steroids and warfarin. Other treatment modalities like low dose radiation therapy, acetic acid iontophoresis and shockwave therapy have also been suggested.

Some orthopaedic surgeons, for the prevention of extra skeletal bone formation have used low dose radiation therapy. Unlike conventional radiation therapy, used in malignancies, anti-inflammatory action is seen in low dose radiation therapy, in both acute and chronic inflammatory diseases. Dosage of radiation is 600 - 700 cGys with minimum side effects.^{30,31,32}

Bisphosphates especially, Sodium etidronate has been used in Myositis Ossificans Progressiva, with some success. However, on discontinuation, it leads to recurrence. Although, safety was reported by many clinicians, it is advised to carefully monitor serum calcium and phosphate levels as long term use may cause osteomalacia. However, it has been found to be ineffective in MOT.³⁰ Hamida et al²⁸ reported a case of MOT in which they have used Alendronate for 6 months with positive results.

Corticosteroids have not been shown to affect the calcification. However, they may be used to reduce acute inflammation. Corticosteroids have also been used with little success intra-lesionally according to Molloy J C et al.³³

Indomethacin was used by Kienapfel et al,³¹ Knelles et al³² and cella JP et al,³⁴ and shown to be effective against heterotrophic bone formation. The mechanism of action is its ability to decrease the synthesis of prostaglandin E and F. Its action is similar to low dose radiation in inhibiting the differentiation of pre-osteoblasts. It is effective if used for 4 to 6 weeks, but, not effective with shorter - term usage. It is administered at a dose of 25 mg 8 hourly or 75 mg sustained release capsules.^{31,32,34}

There are a few documented reports, particularly one report by Buschbacher et al,³⁵ regarding use of warfarin, which was theorized to prevent heterotrophic bone formation by interfering with vitamin - K dependant osteocalcin production. However, the obvious haemostatic considerations may be a limiting factor.

Isotrentoin, which is a retinoid, was used by Vernale C A,³⁶ as a prophylactic measure against extra skeletal bone formation due to its ability to inhibit mesenchymal differentiation into cartilage and bone. Nevertheless, high instances of side effects have rendered it as a less favourite option.

Iontophoresis is a method, which involves movement of an electrically charged substance through the skin using low electrical current. In this procedure, 2% acetic acid was used, as documented by Wieder³⁷.

Extracorporeal shockwave therapy is one of the effective treatment modality most commonly advocated by orthopaedicians and physiotherapists with positive results. Busseli et al,³⁸ administered three sessions over a period of 6 weeks for every 2 weeks at frequency of 100 impulses/cm² of the affected area at a medium power setting of 0.13 to 0.23 mj/mm² in an effort to keep pain at a tolerable level. Treatments were started at an average of 4 ± 2.09 months after the trauma, effectiveness and evaluations were made at 1, 2, 3, 6 and 12 months after. These results have shown complete clinical resolution in 21 out of 24 patients.

Surgery alone is considered as only treatment modality for this condition, in maxillofacial region with varied outcome. Almost all reported literature; suggest immediate surgical intervention, to relieve trismus. However, the results obtained were not satisfactory. Orthopaedic surgeons have suggested that, surgery should not be attempted until the lesion is fully mature, because in immature lesions, the osteoblastic activity is high and aggravation of the condition may occur. Usually, full maturation of the lesion may take 6-12 months of

time and eventually stop. A variety of surgical procedures were performed in head and neck region, ranging from simple excision of the calcified mass to excision of the entire muscle tissue as described by Thangavelu et al.³⁹ In cases of temporalis muscle involvement coronoidectomy has been advocated along with excision of the lesion.

Discussion:

Myositis Ossificans, as described previously, is a heterotrophic bone formation or extra skeletal bone formation within muscle or of tissue including fascia and tendons. There has been a controversy regarding the name itself, as many authors consider it a misnomer arguing that, there is no inflammatory process involved in its aetiology.^{19,25}

There are 29 cases of MOT reported in the head and neck region. There is no age relation observed. A definite male predilection had been observed. The commonly involved muscle is masseter, since it is most likely to receive force directly. Next group involved is medial pterygoid muscle, usually follows and erroneous local anaesthetic technique. Lateral pterygoid muscle and temporalis muscle are less commonly affected group. Though temporalis muscle is vulnerable for trauma like masseter, less chance of occurrence of MOT in this group is seen and the reason for this is not known. When the muscles of mastication are involved, patients will experience limited mouth opening. Radiologically these lesions present themselves as abnormal calcifications within the muscle tissue. MOT rarely occurs in head and neck region and reports suggest that it might take 3 weeks to 6 weeks subsequent to injury before manifestation of symptoms and for ossification to be observed radiographically.²⁴ This condition is difficult to detect on plain radiograph due to superimposition of cranial bones. In such cases, CT scan and MRI have proven to be useful, while planning surgery. Bone scintigraphy has also been used as a modality of diagnosis. De Smet et al,⁴⁰ evaluated seven cases with MRI and described a typical appearance that included a low intensity rim. However, it is important to differentiate these findings from other conditions like osteosarcoma, osteoma, nodular fasciitis, rhabdomyosarcoma, chondroma and osteochondroma, as MOT mimics these conditions radiologically.

MOT is many a times misdiagnosed as osteosarcoma, which mimics it is clinical and microscopic presentation. Diagnosis of MOT is based on history of blunt injury, although it is reported in only 70% of the cases, with supportive clinical, radiographic and microscopic features.

Ackerman,²⁰ recognized and described the “zone phenomena” with inner, middle and outer zones. The central or inner zone contains undifferentiated cells, haemorrhagic and necrotic muscular tissue, loose fibro vascular tissue and spindle cells and prominent giant mesenchymal cells containing abundant mitosis. The middle zone contains active osteoblast and immature osteoid, chondroid and woven bone tissue. The peripheral or outer zone contains mature lamellar bone with active osteoclasts and collagenous fibrous stroma. Although the zonal pattern is considered the hallmark of MOT, many reports suggest that the histological examination reveal a well circumscribed mature osseous tissue with or without cellular component.^{17,41}

Booth and Westers⁴² identified three important criteria in order to establish a diagnosis of MOT. These are:

1. History of significant local injury,
2. Clinical and radiological evidence of ossification within 2 months of the initial injury and
3. Location of the lesion.

Treatment:

Best treatment modality in MOT is prevention of further ossification and an understanding of risk factors, which may lead to formation of heterotrophic bone. Danchick et al,⁷ Ryan et al,¹⁶ and Jackson et al,⁴³ suggested few risk factors, which are as follows:

- 1) Severity of injury: Although MOT can occur following relatively minor trauma or repetitive micro trauma. The more severe the initial injury, the greater the likelihood of developing MOT.
- 2) Localized tenderness and swelling.
- 3) Restricted movements.
- 4) Re-injury during the recovery phase.
- 5) Delay in treatment > 72 hours.
- 6) Improper management of the muscle contusion. These include:

- a. Massage, heat, ultrasound or specific analgesic liniments used in the initial stages of the injury, applied to the area of hematoma;

- b. Soft tissue manipulation, passive exercises and active stretching of the contused area is performed too soon.
- c. Increased bleeding in the tissue secondary to haemophilia or anticoagulant therapy.

As mentioned earlier varieties of treatments were proposed which include surgery and conservative management. Surgery has been an answer for this condition since long, but there had been a controversy regarding the time of surgical intervention. Many authors suggest that it is advisable to intervene only after ossification has stopped, because if surgery is attempted during immature phase there will be aggravation of ossification.

Many orthopaedic counter parts have researched elaborately on the conservative management alone as they argue that it is a self-limiting condition especially in areas where the ossification does not necessarily hamper the daily activity: For example ossification in abdominal muscles and thigh muscles. In cases where joints are involved they suggest that surgery may not be necessary until it completely restrict the movements. Medical management by NSAIDS, particularly Indomethacin and Acetyl salicylate, Osteoclastic drugs like bisphosphonates show good results in MOT. Many authors suggested low dose radiation therapy, as it acts in the same way as NSAIDS. Other methods like acetic acid Iontophoresis and shockwave therapy have also been performed in many cases with positive results.

Some authors advocate a combined surgical and medical management as a treatment modality and showed good results in their follow-up. Knelles et al,³² between 1988 and 1994 performed a study in 723 patients following hip replacement surgeries. They compared the results between patient groups treated with acetyl salicylic acid, Indomethacin and low dose radiation therapy with doses of 3, 5, and 7 Gys, with those of a matched control series. They found that Indomethacin, 2x50 mg for 7 and 14 days, and irradiation of 4x3 Gy or 1x7 Gy, significantly reduced the development of heterotrophic ossification compared with the control group. Patients in the acetylsalicylic acid group and those with a single irradiation of five Gy after operation developed significantly more ossification than those in the indomethacin and other irradiation groups. Similarly, Cella et al,³⁴ studied the effectiveness of Indomethacin in 74 patients. They administered 75 mg Indomethacin once daily for 6 weeks. Their findings support the evidence, that indomethacin can effectively prevent higher grades of heterotrophic ossification. Kienapfel et al,³¹ performed a study in 154 patients in which, they compared the effectiveness of radiation therapy at a dose of 600 cGy once with Indomethacin 2x50 mg for 6 weeks. They found out that both radiation and Indomethacin therapy were effective in prevention of heterotrophic bone formation.

Current literature in maxillofacial region has documented surgery alone as a treatment option with no suggestions regarding the time of intervention. Besides management by conservative methods has not been detailed anywhere. A treatment protocol for MOT has been shown in the figure 1.



Conclusion:

Lack of long-term follow-up and poor documentation of a standard treatment protocol, has made it difficult to draw a conclusive treatment plan. In the absence of proper guidelines for, whether or not surgery should be undertaken and its timing has further thickened the problem. A combined surgical and medical management showed definite improvement and can be tried as an option. However, further studies are needed for the above treatment protocol to be standardized.

Compliance with ethical standards

Conflict of Interest: Authors Khadar vali Shaik, HariPriya chari, Taneem Ahmed declare that they have no conflict of interest.

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Classification of myositis ossificans

Table - 1

| | | | |
|---|------------|---|---|
| 1 | Congenital | Fibrousdysplasia ossificans progressiva (FOP) or stoneman's disease | <ol style="list-style-type: none"> 1. Characterized by the formation of bone in tendons and muscles. 2. Manifests itself in childhood and is slowly progressive resulting in the formation of a "second skeleton" in patients. 3. Very uncommon occurring in approximately 1 in 2 million. |
| | | Progressive osseous heteroplasia (POH) | <ol style="list-style-type: none"> 1. First described by Kaplan in 1994 2. Originally thought to be FOP 3. Ossification begins in the dermis and spreads to the subcutaneous tissue and fascia with no skeletal abnormalities 4. POH is very rare and begins in childhood, similar to FOP. |
| | | Albright's hereditary osteodystrophy (AHO) | <ol style="list-style-type: none"> 1. Manifests with a certain phenotypical presentation along with subcutaneous calcifications about the limb joints 2. Phenotypical features includes: short stature, a round facies, hypertelorism, and shortness of the 4th and 5th digits. 3. Mental retardation is also common in these patients. |
| 2 | Idiopathic | | <ol style="list-style-type: none"> 1. Regarding the idiopathic nature of myositis ossificans and its naming. 2. Some authors use the term myositis ossificans circumscripta (MOC) to describe the heterotopic ossification of soft tissue of an idiopathic nature 3. Other authors use MOC as synonymous with myositis ossificans traumatica has been reported following burns, tetanus, and polio and neurogenic injuries including spinal cord injuries, closed head injuries, central nervous system infections, and stroke |

| | | |
|---|-----------|--|
| 3 | Traumatic | <ol style="list-style-type: none"> 1. Difficult to adequately research MOT due to its many synonyms. 2. These include post-traumatic myositis ossificans, myositis ossificans circumscripta, pseudomalignant heterotopic ossification, pseudomalignant myositis ossificans, pseudomalignant osseous tumor of soft tissue, extraosseous localized non-neoplastic bone or cartilage formation 3. Some radiologists consider the traumatic ossification of ligaments specifically to be called dystrophic calcification. 4. Dystrophic calcification is defined as "calcium salt deposits in the presence of normal calcium/phosphorus metabolism involving tissues that do not physiologically calcify" which may not adequately describe our case |
|---|-----------|--|

(Courtesy by: Dr. Brad Muir)

Classification of MOT

Table - 2

| Arrington classification | | |
|--------------------------|-------------|--|
| 1 | Stalk | There is a stalk attachment to the underlying bone |
| 2 | Periosteal | There is total continuity between the underlying bone and the heterotopic bone |
| 3 | Broad based | is a large base of attachment to the underlying muscle |

(Courtesy by: Dr. Brad Muir)

Classification of MOT

Table - 3

| Mestan and Bassano Classification | | |
|-----------------------------------|---------------|--|
| 1 | Parosteal | the ossification is found near or against bone |
| 2 | Periosteal | the periosteum is torn away from the underlying cortex due to the trauma. The resulting injury may have a sunburst appearance. |
| 3 | Extra osseous | the heterotopic bone is found in the substance of the involved muscle |

(Courtesy by: Dr. Brad Muir)

Difference between MOT and Osteosarcoma

Table - 4

| Myositis Ossificans Traumatica | Osteosarcoma |
|--|---|
| <ul style="list-style-type: none"> • history of trauma • Starts in Soft tissue • cortex usually intact even with a periosteal reaction • pain and mass decrease with time • activity-related pain • alkaline phosphatase levels often normal but can be elevated | <ul style="list-style-type: none"> • no history of trauma (although trauma can occur in up to 40% of cases) • Starts in bone • cortex often violated • pain and mass increase with time • night pain • alkaline phosphatase levels elevated |

(Courtesy by: Dr. Brad Muir)

Table – 5

Treatment and Regime for MOT

| Treatment | Regime |
|-----------------|---|
| Aspiration | <ul style="list-style-type: none"> • Early aspiration of the hematoma with subsequent injection of steroids, lysosomal enzymes, and xylocaine. |
| Bisphosphonates | <ul style="list-style-type: none"> • Alendronate is commonly used • Dose 40mg per day for 6 months |
| NSAIDS | <ul style="list-style-type: none"> • Indomethacin 75 mg per day for 6 months • Acetylsalicylic acid 750 mg thrice daily |

| | |
|------------------------------------|--|
| Acetic acid Iontophoresis | <ul style="list-style-type: none"> • 2% acetic acid is used along with iontophoresis • 3 times a week for 3 weeks |
| Surgery | <ul style="list-style-type: none"> • Last option • Better to intervene after stoppage of once the lesion reaches osseous maturity • Chances of recurrence |
| Extra corporeal shock wave therapy | <ul style="list-style-type: none"> • Non-invasive procedure • Administered for 6 weeks |

(Courtesy by: Dr. Brad Muir)

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