REVIEW ARTICLE

e-ISSN: 2349-0659 p-ISSN: 2350-0964
doi: 10.21276/apjhs.2018.5.1.03

Enigma of myofascial pain-dysfunction syndrome - A revisit of review of literature

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ABSTRACT

Myofascial pain-dysfunction syndrome (MPDS) is a form of myalgia that is characterized by local regions of muscle hardness that are tender and cause pain to be felt at a distance, i.e., referred pain. The central component of the syndrome is the trigger point (TrP) that is composed of a tender, taut band. Stimulation of the band, either mechanically or with activity, can produce pain. Masticatory muscle fatigue and spasm are responsible for the cardinal symptoms of pain, tenderness, clicking, and limited function that characterize the MPDS. Since MPDS covers a wide range of symptoms, it might be difficult to diagnose and provide definitive treatment. A better understanding and working knowledge of TrPs and MPDS offers an effective approach to relieve pain, restore function, and contribute significantly to patient's quality of life.

Key words: Myalgia, myofascial pain-dysfunction syndrome, referred pain, trigger points

INTRODUCTION

Muscle pain is a common problem that is underappreciated and often undertreated. Myofascial pain-dysfunction syndrome (MPDS) is a myalgic condition in which muscle and musculotendinous pain are the primary symptoms and is the most common form of temporomandibular disorders. MPDS has always been an enigma, some have even questioned the mere existence of MPDS.[1]

The conventional definition of MPDS is "an entity characterized by regional pain originating from hyperirritable spots located within taut bands of skeletal muscle, known as myofascial trigger points (MTrPs). American Academy of Craniomandibular Disorders classified MPDS under craniomandibular disorders of non-organic (functional) origin."[2,3]

A "trigger zone" is a local zone of palpable hyperirritability in a tissue which when compressed leads to local pain and if hypersensitive can lead to radiating pain in other regions of the face.[4] The MTrP is, in turn, defined as a palpable and hyperirritable nodule located in a taut band of muscle. Stimulation of this point produces two characteristic phenomena: Referred pain and sudden contractions of the taut band, called the local twitch response. Active MTrPs produce pain, and sometimes referred pain, spontaneously. Latent MTrPs produce referred pain as a response to pressure, but not spontaneously.[5-7]

MPDS is prevalent in regional musculoskeletal pain syndromes, either alone or in combination with other pain generators. It is a psychological disorder which involves the masticatory muscles and results in pain, limitation in jaw movement, joint noise, jaw deviation in closing and opening the mouth, and sensitivity in touching one or more masticatory muscles or their tendons. The main acceptable factors include occlusion disorders and psychological problems.[6,7-10]

Common etiologies of MPDS may be from direct or indirect trauma, spine pathology, exposure to cumulative and repetitive strain, postural dysfunction, and physical deconditioning. However, no signs of obvious pathogenesis causing the syndrome have been found, it relies significantly on the clinical examination.[8,9]

The definitive treatment of MPDS depends on the symptoms of the patient. Some require relief of symptoms while in some patient the treatment of causative factor is necessary.[11-13]

Here, we review the history, epidemiology, etiologic factors, clinical features, diagnosis, and the management of this syndrome.

HISTORY

Anatomical causes of MPDS were first postulated in 1918, when it was suggested that loss of vertical dimension resulted in damage to the meniscus, condyle, and glenoid fossa of temporomandibular joint (TMJ). In 1938, British rheumatologist Kellgren published a seminal paper describing specific referred pain patterns of many muscles and spinal ligaments following injections of hypertonic saline. We owe our present awareness of myofascial pain as an important clinical entity to the work of Travell (1901–1997) who injected local anesthetic instead of saline and later to the incredibly productive collaboration between Dr. Travell and Dr. Simons.[14]

Dr. Travell used the term "myofascial" to describe the involvement of both muscles and its covering tissue, the fascia, and "TrP" to convey the notion that pain initiated at one site in a particular muscle triggered pain felt at a site distant to the point of origin.

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Received: 14-11-2017 **Revised:** 24-11-2017 **Accepted:** 02-01-2018

Travell and Simons authored the classic two-volume text "myofascial pain and dysfunction - the TrP manual."[12]

Bruno (1971) found that the resulting pain in muscles will be concentrated in areas of fascia which on palpation demonstrated tenderness and pain and these areas were referred to as trigger areas.

According to Weinberg (1974), every patient has got adaptation to a situation which is determined by his psychological makeup. In a given patient, an occlusal interference may trigger the patient's acute symptoms, while another factor, such as emotional stress may sustain them.[12-14]

Evidence that nocturnal parafunction may be involved in MPDS stemmed from studies by Trenouth (1978) who observed that jaw pain and limitation of movement were often noted to be worse on awakening.

Christensen (1981) and Yemm (1979) demonstrated that in chronic cases of MPDS, an inflammatory stage occurs in affected muscles of mastication following the classic spasm. Several assessment and treatment approaches have emerged independently of each other both in Europe and in the United States, including MTrP therapy (USA), neuromuscular technique (NMT) (UK), neuromuscular therapy, also abbreviated as NMT (USA), and manual TrP therapy (Switzerland).[13-15]

Recent insights into the nature, etiology and neurophysiology of MTrPs and their associated symptoms have propelled the interest in the diagnosis and treatment of persons with MPS worldwide. Eversole and Machado subdivided TMJ dysfunction syndrome into two subgroups: (1) Myogenic facial pain and (2) TMJ internal derangements.[14,16]

EPIDEMIOLOGY AND DEMOGRAPHICS

TMJ symptoms are relatively common, occurring in 10–25% of the population; only about 5% of people with symptoms will seek treatment. However, the incidence of MPDS can be as high as 54% in women and 45% in men, although the prevalence of TrPs does not exceed 25%.[17]

Several studies show that MPDS is more common in females. It is most frequently seen in young unmarried females (married-to-unmarried ratio 1:2) and female-to-male ratio is 3:1. Since females are more exposed to psychological disorders and they have very limited tolerance to pain, these results might be reasonable.

The most common age at presentation is between 27.5 and 50 years, with preference in sedentary individuals.[14,16,18] In the studies achieved by other investigators such as Honarmand *et al.*, the average age was 32.4, 26.67, 33.5, and 31.3 years, respectively.[7,19-21]

ETIOPATHOGENESIS

MPDS has a multicausal etiology and the knowledge about the probable etiological factors seem to have improved over a period of time. Travell and Rinzler first suggested that skeletal muscles in spasm could be the source of pain. They described about the painful areas within the muscles and called them as "trigger areas" which were associated with pain, spasm, tenderness, and dysfunction.[22]

Schwartz (1955) adapted Travell's work and postulated the term TMJ pain-dysfunction syndrome. He reported that majority of patients with pain in the region of TMJ were suffering from functional disorders involving painful spasm of masticatory muscles. He hypothesized that stress was a significant cause of clenching and grinding habits which resulted in muscle spasm. Occlusal abnormalities were found to play a secondary role.[23]

The next significant development toward understanding this aspect of facial pain occurred when Laskin presented a comprehensive explanation of the problem and proposed his psychophysiological theory. He suggested that though mechanical factors related to occlusion may cause this condition by producing muscular overextension or overcontraction leading to muscle fatigue, psychophysiologically motivated oral habits is the frequent cause of painful spasm. To stress the role played by muscles, it was suggested that the term MPDS is a more accurate term to describe the condition than TMJ pain-dysfunction syndrome.[24]

One of the significant signs of MPDS is the presence of TrPs in a specific group of muscles. "TrPs are small exquisitely tender areas, which cause pain referred to a distant region, called the referred pain zone." They are activated by pressure, movement, change of barometric pressure and tension, be it physical or emotional and gives rise to referred pain and tenderness.[14,21]

Current controversy about etiology of MPDS is essentially between two major concepts occlusal disharmony and psychophysiologic factors and "several investigators" advocate a combination of both. The supporters of each of these concepts agree that most TMJ dysfunction problems are manifested primarily in the masticatory muscles, rather than in the TMJ itself.

MPDS can be caused by factors such as: [22-24]

A. Occlusal disturbances
B. Intracapsular disorders or
C. Emotional turmoil.

Etiologic factors may also include:

A. Whiplash injury from an auto accident,
B. Wrestling blow,
C. Trauma from falling, or
D. Unexpectedly biting into a hard object.

INTEGRATED TRP HYPOTHESIS

The presynaptic, synaptic, and postsynaptic mechanisms of abnormal depolarization i.e., excessive release of acetylcholine (Ach) and defects of acetylcholinesterase have been proposed as the possible etiological mechanisms by Simons. The excessive Ach maintains a sustained depolarization of the postjunctional membrane, which in turn results in an excessive release of calcium from the sarcoplasmic reticulum and sustained sarcomeric contractions. This, in turn, causes local hypoxia, reducing the available energy supply. This causes a combined decreased energy supply and increased metabolic demand stimulating the release of endogenous substances, resulting in hyperalgesia,

central sensitization, and stimulation of the autonomic nervous system, which has been shown to increase endplate potentials. Endogenous substances such as bradykinin, serotonin, prostaglandin, and calcitonin gene-related peptide, combined with lowered pH activates peripheral nociceptors and trigger capsaicin-sensitive afferents, causing pain [Figure 1].[9,14]

The masticatory muscle spasm is the primary factor responsible for signs and symptoms of pain-dysfunction syndrome. Spasm can be initiated in one of the three ways: [9,14]

A. Muscular overextension
B. Muscular overcontraction or
C. Muscle fatigue.

MPDS so produced not only causes pain and limitation of movement but also produces changes in jaw position so that teeth do not occlude properly (occlusal disharmony). In addition, it may also cause organic changes such as degenerative changes in the TMJ and muscle contraction which is a manifestation of long-term spasm. These organic changes result in an altered chewing pattern with attendant reinforcement of the original spasm and pain. The changes in neuromuscular control of mandible produced due to occlusal disharmony have been supported by many researchers.

Occlusal interferences, posterior bite collapse, deep overbite/overjet, and many other factors tend to restrict movement and predispose the patients to increased parafunctional activity resulting in overuse, and thus fatigue of muscles. Iron insufficiency, hypothyroidism, and Vitamin D deficiency are considered as perpetuating factors.[14-16]

CLINICAL FEATURES

A great diversity of criteria exists for defining myofascial pain. MPDS is a psychological disorder which involves the masticatory muscles and results in pain, limitation in jaw movement, joint noise, jaw deviation in closing and opening the mouth, and sensitivity in touching one or more masticatory muscles or their tendons. Other patient complaints include headache, earache, hypertrophy of masticatory muscles, and abnormal wear of masticatory surfaces.[25]

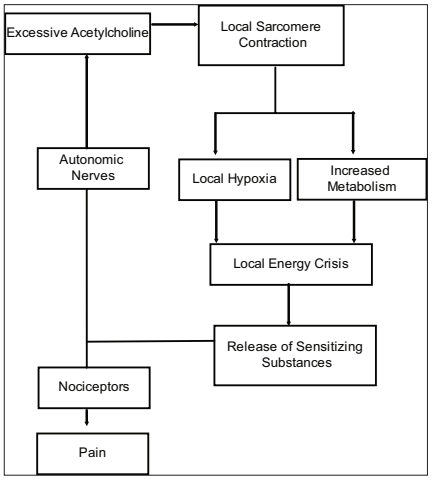

Figure 1: Integrated trigger point hypothesis

Pain

Pain associated with MPDS is usually unilateral. It may be bilateral in some, but if bilateral, it need not be symmetrical. The quality or character of the pain reported by the patient most often will fall into three gross categories: [26]

1. A dull aching pain,
2. A sharp shooting pain (burning), and
3. A tight-drawing sensation.

Patients with myofascial pain syndrome usually exhibit protective habits or reflexes to avoid activating the pain.[9]

Tenderness

It is present over the affected TMJ during normal opening and closing motions, best elicited by placing the examining fingers at the posterosuperior aspect of both the condyles and expressing pressure anteriorly during excursion. It is more common over the condyle, above the maxillary tuberosity, at the angle of the mandible and the temporal crest.

Clicking or Popping Noises in the TMJ

They are common and described as clicking, popping, or crepitus. The nature of the click is still uncertain. It is usually bilateral. It can occur at any point of jaw movement and there may be multiple clicks. It may be audible, palpable, or both and usually noted on simple palpation directly over the condylar head during the opening movement. Crepitus has been associated with perforations in the disk, which is usually followed by osteoarthritic change on the condylar surface followed by similar bony alterations on the opposing surface of the fossa.

Deviation of the Jaw to the Affected Side during Normal Opening Motion

It is a common finding since muscle spasm frequently accompanies joint dysfunction and as such contributes to the pain. This restricts the motion of the condyle, impairing or completely eliminating the forward gliding motion so that all that remains is a simple hinge action, with the condyle remaining in the fossa.[9,14,27,28]

Mortazavi *et al.* found that the most and the least common muscular involvements are related to medial pterygoid (87% of cases) and temporal (41% of cases) muscles, respectively. In the study of Darbandi, the most common muscular involvement is related to lateral pterygoid muscle (82.68%).[28]

They also found severe or slight limitation while opening the mouth in 71.8% of patients; whereas, Madani and Darbandi have reported 26% and 40.38%, respectively. These differences could be the result of difference in number of samples and also in measuring factors.[19,27,28]

Mortazavi *et al*. found jaw deviation and deflection were reported in 7.7% and 41% of patients in sequence; whereas, Madani has mentioned the total percentage of both of them as 45%.[19,28]

Varma et al. found occlusion Class I, to be the most common type in patients with MPDS. Their results were consistent with those reported by Darband *et al.* The most common malocclusions among patients with MPDS were cross bite, deep bite, and open bite.[14,19,27,29]

Bruxism is the most common habit observed in patients with MPDS. Long muscle contraction during bruxism prevents adequate

blood supply to the muscular tissue and results in accumulation of CO_2 and painful products in muscle and finally leads to pain, fatigue, and muscular spasm. 56.4% of cases were reported to have bruxism by Mortazavi *et al*.; whereas, the scales reported by Honarmand *et al*. were 45.6%, 38%, and 68.9% in sequence.[7,14]

Patients with MPDS may show symptoms other than pain in masticatory muscles region such as earache, neck pain, and particularly headache. Tension headache caused by long muscular contractions was the most common type of headache.[27]

The patients suffering from MPDS usually present with complaint of the following which have an interrelated mechanism [Figure 2]: [14,27-29]

1. Pain in a zone of reference (most important problem that causes patients to seek treatment).
2. TrPs in muscles which cause pain on stimulation.
3. Taut muscle band.
4. Limited jaw opening.
5. Associated symptoms.
6. Presence of contributing factors for onset of pain.
7. No tenderness in TMJ.

Signs and symptoms of myofascial pains are often accompanied by other pathological conditions and other problems such as: [9,14,30]

I. Neurologic: Tingling, numbness, blurred vision, and excess lacrimation.
II. Gastrointestinal: Nausea, constipation, and indigestion.
III. Musculoskeletal: Fatigue, tension, stiff joints, and muscle twitching.
IV. Otologic symptoms: Tinnitus, ear pain, and diminished hearing.
V. Other symptoms: Scratchy sensation, teeth sensitivity, increased salivation, increased sweating, and skin flushing.

DIAGNOSIS

Facial pain and its diagnosis have always posed a dilemma for the clinicians. The diagnosis of myofascial pain is based mainly on the history and clinical examination. Using the history, information is gathered regarding the type, intensity, duration, frequency, and location of the pain, as well as alleviating or accentuating factors [Table 1].

The essential part of the clinical examination is to locate the TrPs by manual palpation of the cervical and facial musculature. Localization of TrPs is based on three basic maneuvers: (a) Direct finger pressure, (b) flat palpation, and (c) pincer palpation.

Radiographs may be helpful in diagnosing the condition if it has affected the bony structure also. They include transcranial, transpharyngeal, panoramic views, computed tomography scans, and magnetic resonance imaging with arthroscopy. Arthrography can be useful in determining the position of meniscus (when internal derangement of TMJ is being considered).

Ultrasound, electromyography, algometry, and thermography are some of the complementary tests quoted in the literature for diagnosis.[32,33]

The development of criteria for the diagnosis of MPDS was the object of an attempt initiated at the 1998 International Myopain Congress in Italy. A multicenter study was developed, and the merged data evaluation of the 80 subjects in the study showed that local tenderness, referred pain, and a palpable taut band were useful. However, agreement on diagnosis for both centers was weak (Kappa = 0.32).[14,34]

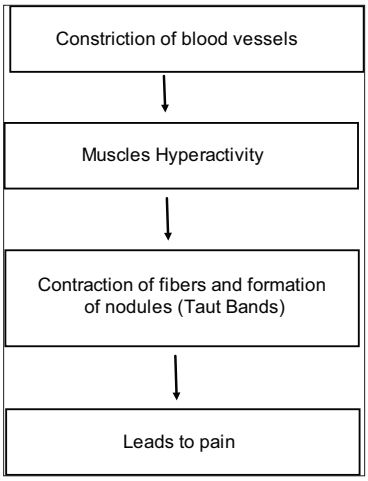

Figure 2: Mechanism to show how pain occurs[30]

Table 1: Factors which increase or decrease painful symptoms of TrPs,[17,31]

Aggravating factors	Moderating factors
Overuse of musculature	Rest
Active stretching	Passive stretching
Pressure on TrP	Specific myofascial therapy
Prolonged muscle contraction	Non-isometric contraction activity
Cold, damp, viral infections, tension	Local warming of TrP

TrP: Trigger point

DIFFERENTIAL DIAGNOSIS

The various disorders that are included in the differential diagnosis of MPDS can be categorized as follows:[14,34]

I. Non-articular conditions that mimic MPD syndrome: Pulpitis, pericoronitis, otitis media, parotitis, sinusitis, trigeminal neuralgia, atypical (vascular) neuralgia, temporal arthritis, trotter's syndrome, and Eagle's syndrome.
II. Non-articular conditions producing limitation of mandibular movement: Odontogenic infection, non-odontogenic infection, myositis, myositis ossificans, neoplasia, scleroderma, hysteria, tetanus, extrapyramidal reaction, depressed zygomatic arch, and osteochondroma.
III. Differential diagnosis of TMJ disease: Agenesis, condylar hypoplasia, condylar hyperplasia, neoplasia, infectious arthritis, rheumatoid arthritis, traumatic arthritis, degenerative arthritis, ankylosis, and internal disk derangement.

TREATMENT

Management of MPDS requires an intensive multidisciplinary treatment module. It is divided into two groups.[9,14,34]

1. Non-surgical management.
2. Surgical management.

Non-surgical Therapy

1. Initial therapy: It aims to bring the joint back to its normal healthy condition. It includes:
   A. Reassurance: Explanation to the patient about the nature and prognosis of the disorder and to reassure the patient about the treatment.
   B. Diet: Elimination of hard and chewy food helps to reduce loading forces on the joints and to rest hypertonic jaw muscles.
   C. Rest.
   D. Thermotherapy: Surface heat is applied by laying a hot moist towel, electric heating pad over the symptomatic area. This combination should remain in place for 10–15 min.
2. Supportive therapy: Two types of supportive therapy are there:
   a. Those directed toward the relief of pain. It includes pharmacologic therapy and physical therapy.
   b. Those directed toward the relief of dysfunction.

Pharmacologic therapy

A. Analgesics: Opioid analgesics depresses CNS, just relieve pain. Whereas, non-opioid analgesic relief pain without depressing CNS. For examples - morphine, pethidine, codeine, salicylates, and paracetamol. There is clear evidence that the analgesic properties of NSAIDs relieve pain in MPDS.[35] A number of RCT studies, case reports, and observational studies examining the efficacy of lidocaine patches on MPS have been conducted. These studies were congruent in showing that lidocaine patches had a statistically significant increased pain thresholds ($P < 0.001$), and increase in general activity ($P < 0.05$). Topical lidocaine has shown promise as a therapy for MPS and is especially appealing as it is not an oral systemic drug.[36,37]
   i. Anti-inflammatory agents: Commonly used are salicylates (aspirin), propionic acid (ibuprofen), acetic acid (indomethacin), fenamic acid, oxicam, and aryl-acetic acid derivatives (diclofenac sodium).
   ii. Anxiolytics agents: Benzodiazepines such as alprazolam, diazepam, lorazepam, and oxazepam are commonly used to alter the patient's perception or reaction to the supportive therapy.
   iii. Muscle relaxants: Reduces muscle strains. Commonly used are carisoprodol, chlorzoxazone, meprobamate, methocarbamol, and cyclobenzaprine. Tizanidine is a centrally acting alpha-2-adrenergic agonist, which decreases muscle spasticity.
   iv. Local anesthetics: Reduces the pain immediately, thus providing relief to the patient allowing complete muscular movement. Apart from therapeutic, it also acts like a diagnostic tool. Once the area is anesthetized, it is easy to diagnose the trigger zone and its radiating path.
   v. Herbal medicines: Lavender, lemon balm, rosemary, kava kava, and skullcap are some of the recommended medicines.[34,35]

Physical therapy

Many authors claim that physiotherapy is the main module of treatment and that injections are only of secondary purpose. Physiotherapy provides treatments to patients with physical limitations caused by disease or injury. Several modes of physiotherapy are available:

a. Spray and stretch
b. Ischemic pressure
c. Soft pressure and continuous massaging
d. Continuous suppleness exercises.

Physical therapy may also include electrical stimulation therapy which includes:

a. Electrogalvanic stimulation
b. Transcutaneous stimulation.

Furthermore, other techniques can be used such as:

i. Acupuncture
ii. Ultrasound
iii. Iontophoresis
iv. Cold or soft laser.

Therapy for relief of dysfunction

a. Restrictive use: Painful movements should be avoided to prevent damage to the structure. The patient should eat soft food and take smaller bites.
b. Exercises: All exercises programs involve the stretching of hypertonic muscles. Physiotherapy can be regarded as the first choice in select cases.
c. Biofeedback therapy: It gives patient voluntary control over automatically regulated body functions. It reduces bruxism and also reduces stress.
d. Occlusal appliances: Occlusal splints and night guards can be used which reduces muscle spasm and TMJ pain and tooth abrasion and also relieve clenching. They are best fabricated for the maxillary arch.[33-35]

RECENT TRENDS IN MANAGEMENT OF MPDS

1. Botulinum toxin an injection: Injection of BTX-A in the masseter and temporalis muscle fibers extraorally under electromyography guidance serves to prove effective in 9 out of 10 patients. These muscles are most commonly involved and radiate the pain to ear and temporal headache, respectively, which leads to the limitation of the mandibular motion and develop MPDS.[34-38]
2. Ultrasound: Ultrasound therapy uses the transmission of the sound waves through conducting gel into the tissue leads to the breakdown of scar tissue. It increases the cell membrane permeability by altering the sodium-potassium ion gradient. It increases the exchange of gases, which promotes healing and reduces inflammation.[34]
3. Iontophoresis: It is a procedure of passing low amperage current to the tissue of the area involved. A pad is placed over the skin of patient and electric current is passed through it into the tissue. Cold and soft laser therapy: Application of the low-level laser therapy has been sought to promote healing, reduce inflammation. It accelerates collagen synthesis, increases vascularity, and decreases the no of microorganism and pain.[24]

CONCLUSION

In the craniomandibular region, MTrPs are frequently responsible for local and referred pain patterns, causing headaches, TMJ pain and dysfunction, toothaches, and facial pains. MPDS is a psychophysiologically altered condition involving the muscles of mastication and cervical group of muscles. The condition is characterized by dull aching, radiating pain often results in muscle spasm and restricted movements. It is a self-limiting disorder if etiological factors are removed. Recent progress in experimental studies has provided a wealth of information that can be used to gain understanding of the syndrome. Only through improved understanding of the molecular and subcellular pathways behind this disorder, novel therapeutics can be discovered. Many details of the signaling pathways involved remain yet unclear and further studies are needed.

Treatment of MPDS finds that most interventions have demonstrated a limited body evidence for their use. This dearth of high-quality evidence is due to the heterogeneity of MPDS. The treatment should focus primarily on identifying and correcting the underlying cause of the symptoms. Surgical management should be considered only after reasonable non-surgical efforts have been tried.

Further, research is needed to better establish algorithmic and evidence-based treatment of MPDS. The advent of more sophisticated imaging allows new ways of evaluating both the muscle harboring a TrP and central responses to muscle TrP pain. Nevertheless, there are many areas that need more detailed or innovative studies to expand our knowledge about the fundamental nature of the TrP as well as to develop more effective ways of diagnosing and managing TrP - related pain.

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How to cite this Article: Nabhan AB. Enigma of myofascial pain-dysfunction syndrome - A revisit of review of literature. Asian Pac. J. Health Sci., 2017; 5(1):13-19.

Source of Support: Nil, **Conflict of Interest:** None declared.