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Oral and craniofacial manifestations of multiple sclerosis: implications for the oral health care provider

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Abstract. – Multiple sclerosis is a complex neurological condition affecting sensory and motor nerve transmission. Its progression and symptoms are unpredictable and vary from person to person as well as over time. Symptoms of orofacial pain, trigeminal neuralgia, spasticity, spasms, tremor, fatigue, depression and progressive disability, impact on the individual's ability to maintain oral health, cope with dental treatment and access dental services. Also, many of the medications used in the symptomatic management of the condition have the potential to cause dry mouth and associated oral disease. There is no cure for multiple sclerosis, and treatment focuses on prevention of disability and maintenance of quality of life. The oral health care team plays an essential role in ensuring that oral health impacts positively on general health. This review highlights the epidemiology, etiology, pathophysiology, diagnosis, oral and craniofacial manifestations and their management, and oral health care considerations in patients with MS.

Key Words:
Multiple sclerosis, Oral manifestations, Craniofacial manifestations, Oral health care, Dental treatment.

Introduction

Multiple Sclerosis (MS), also known as disseminated sclerosis or encephalomyelitis disseminate, is a chronic, inflammatory, demyelinating disease of the central nervous system[1]. MS was first described in 1868 by Jean-Martin Charcot[2]. The name multiple sclerosis refers to scars (sclerae – better known as plaques or lesions) particularly in the white matter of the brain and spinal cord[2]. The presentation and course of the disease may vary significantly, but it is generally marked by recurrent attacks of physical, neurological and sometimes psychiatric dysfunction.[3] Multiple motor and sensory disturbances occur in MS and may present as painful conditions that affect the orofacial region[4]. The disease itself and the medications taken to manage it influence the oral health and oral health care in MS. The dentist plays an important role in the healthcare of MS patients as part of a multidisciplinary team (general physicians, neurologists, psychologists, etc.). The increasing prevalence of MS in the general population makes it imperative for oral health care providers to be able to recognize craniofacial and oral signs/symptoms of MS and distinguish them from those of dental origin.

We searched the Cochrane Database, MEDLINE (PubMed), Google Scholar and EMBASE, all up to 26 December 2013, using the key words multiple sclerosis, facial pain, oral pain, headache, dental considerations and oral health care. This review highlights the epidemiology, etiology, pathophysiology, diagnosis, craniofacial and oral manifestations, their management and oral health care considerations in patients with MS.

Epidemiology and Demographics

Signs of MS usually appear first in young adulthood, usually with a 2:1 ratio in favour of females[5]. Most individuals with MS are diagnosed between ages 20 and 45 years. It is generally found in countries in the northern hemisphere with an incidence of 55.2-110 cases for every 100,000 population in Canada, 85 cases in USA and 69 cases of world population[6]. MS is more common in people who live farther from the equator, although exceptions exist[7]. These exceptions include ethnic groups that are at low risk far from the equator such as the Samis, Amerindians, Canadian Hutterites, New Zealand Maori, and Canada's Inuit, as well as groups that have a relatively high risk close to the equator such as Sardinians, Palestinians and Parsis[7,8]. The cause of

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this geographical pattern is not clear. While the north-south gradient of incidence is decreasing as a result of migration, it is still present[5].

Etiology and Pathophysiology

The cause of MS is unknown; however, it is believed to occur as a result of a combination of geographic location, environmental factors such as infectious agents and genetics[9]. Geographic location may play a role with northern European populations being high risk groups and the geographic variation may simply reflect the global distribution of these high-risk populations[1].

Although MS is not a genetic disease, genetic linkages that may contribute to MS have been identified with alleles of the human leukocyte antigen (HLA) class II region of the major histocompatibility complex (MHC). Such linkages have been confirmed on chromosome 6p21, while suggestive linkages have been found on chromosomes 17q23 and 5q33[1,10]. However, genetic heterogenicity exists and different individuals may be affected by involvement of genes outside the HLA locus[10].

Environmental factors like decreased sunlight exposure resulting in decreased vitamin D production and cigarette smoking have been implicated as causative factors[9]. Infectious agents have been linked to MS, particularly the role of Epstein Barr virus (EBV)[11,12]. Early age (<15 years) at primary EBV infection is typically asymptomatic, but primary infection during adolescence (>15 years) or adulthood often manifests as infectious mononucleosis, which has been associated with a two- to threefold increased risk of MS[12]. This supports the widely accepted "hygiene hypotheses" which proposes that exposure to certain infectious agents early in life is protective, the disease being a response to a late encounter with such agents. Most importantly, MS risk is extremely low in individuals who are EBV negative, but it increases several folds following EBV infection. Additional evidence supporting a role for EBV in MS pathogenesis includes the observations of elevated antibodies to EBV antigens (especially EBV nuclear antigen-1) prior to the onset of MS, and an increased risk of MS among EBV-positive children[12]. Other viruses, such as human herpes virus type 6 and human endogenous retrovirus have been implicated as contributing to MS; however, further research is required before a confirmatory relationship between the viruses and the development of MS can be ascertained[13].

MS pathophysiology appears to involve a neurodegenerative or infectious process with secondary inflammation[14]. The inflammatory process results from the stimulation of T cells (CD4, CD8) which when activated release pro inflammatory cytokines such as interferon gamma, Interleukin (IL)-17, IL-21, IL-22 and IL-26. These activated cells and their cytokines selectively attack oligodendrocytes or the myelin sheath, promote vascular permeability and bring about demyelination and axonal loss[14]. B cell activation and antibody responses also appear to contribute to demyelination via direct damage or through their inflammatory mediators such as lymphotoxin and Tumour necrosis factor (TNF)-Alpha[14]. Regulatory T cells (T reg) are another CD4+ T cell type involved in the pathogenesis of MS. The role of T reg cells is to regulate the activated CD4+ cells. The number of T reg cells is the same between MS patients and controls; however, patients with MS have reduced T reg function[14]. The blood brain barrier becomes permeable of activated T and B cells following infection by a virus or bacteria, therefore providing access to the central nervous system.

Demyelination of a neuron ensures that it no longer can effectively conduct electrical signals. A repair process, called remyelination, takes place in early phases of the disease, but the oligodendrocytes are unable to completely rebuild the cell's myelin sheath[15]. Repeated attacks lead to successively less effective remyelinations, until a scar-like plaque is built up around the damaged axons. These scars (ranging from 1 or 2 mm to several centimetres) are found most commonly affecting the white matter in the optic nerve, brain stem, basal ganglia and spinal cord, or white matter tracts close to the lateral ventricles[1]. The peripheral nervous system is rarely involved (level of evidence IIa, Table I)[16].

Oral and Craniofacial Clinical Presentation

The orofacial region may be the site of initial signs and symptoms of neurologic disturbances associated with relapses of MS. These attacks typically last for at least 24 hours with an average frequency of 3 times a year[17]. The usual presenting symptoms are mild dysarthria (impaired ability to articulate words), Lhermitte sign (electrical sensation down the spine on neck flexion), monocular visual disturbances (partial vision loss, pain due to optic neuritis[18] and diplopia), intermittent unilateral facial numbness or pain

Table I. Description of the level of evidence supporting the use of a particular therapeutic intervention.

Ia - Evidence from meta-analysis of randomized controlled trials
Ib - Evidence from at least one randomized controlled trial
IIa - Evidence from at least one well designed controlled trial which is not randomized
IIb - Evidence from at least one well designed experimental trial
III - Evidence from case, correlation, and comparative studies
IV - Evidence from a panel of experts

(neuropathic or neuralgic pain), facial palsy or spasm. Danesh-Saini et al[19], in a clinical evaluation of 500 patients with MS reported a frequency of 88.6% for orofacial manifestations. Visual disorders (80.4%) were observed most frequently in patients with MS, followed by temporomandibular disorders (58.2%), dysarthria (42.1%), dysphagia (26.6%), facial palsy (19%), and trigeminal neuralgia (7.9%). A significant correlation with orofacial manifestations was found in patients with a longer duration of disease (>7 yr) compared with patients with a shorter duration. When such patients present to the oral health care provider, it is imperative that referral to a neurologist is sought for a thorough evaluation of MS.

Disease progression in MS may manifest as numerous sensory and motor disturbances in the orofacial region. Most of these symptoms are of pain, although paraesthesias, dysphagia, facial palsy, hemifacial spasm, Charcot triad (neurologic triad of nystagmus, intention tremor, and scanning or staccato speech) and tremor have been reported[4,20].

Cranial neuralgias (CNs) including trigeminal neuralgia (TN), glossopharyngeal neuralgia (GN), and occipital neuralgia (ON) are typical expressions of neuropathic pain in MS. Neuralgias are characterised by paroxysmal painful attacks of electric shock-like sensations that may be spontaneous or evoked by innocuous stimuli in specific trigger zones. In MS, demyelination is thought to play an important role in the origin of neuralgic pain in symptomatic cranial neuralgias[21]. This observation has been supported by the findings of histopathological studies of surgical specimens obtained from MS patients who were subjected to trigeminal rhizotomy, which show demyelination in the centrally myelinated part of the trigeminal root[22]. The painful syndromes arising in MS patients are considered "symptomatic", which is in contrast to classical CNs where no cause other than a neurovascular contact is identified[23].

The pain in TN is limited to the distribution of one or more divisions of the trigeminal nerve. The pain is usually intense and can be sharp, superficial and stabbing. In addition, the pain can last anywhere from a fraction of a second to 2 min. TN frequently attacks the second or third division and rarely (<5%) affects the ophthalmic branch of the nerve[24]. Between paroxysms, the patient is usually asymptomatic; however, a dull background pain may persist in some long-standing cases[24]. Pain is commonly evoked by trigger stimuli (e.g., washing, shaving, smoking, talking and/or brushing of the teeth), but pain may also occur spontaneously. Small areas in the nasolabial fold and/or the chin are well-known trigger areas[24]. The pain that is caused by symptomatic TN, which represents about 15% of all TNs, is indistinguishable from classical TN. However, the pain is caused by a demonstrable structural lesion other than a compression of the nerve root by a tortuous or aberrant vessel[23]. TN is relatively frequent in MS patients. In 0.3% of cases it is the first manifestation of MS[25]. The prevalence ranges from 1.9% to 4.9%, and MS patients have a 20-fold increased risk of TN compared with the general population[26]. Although symptomatic TN is mainly unilateral, bilateral involvement has been reported to occur with frequencies ranging from 11% to 31%[27]. Other clinical differences between symptomatic TN and classical TN are possible sensory impairments in the affected division of the nerve[23], the younger age at onset[23] and a lower frequency of involvement of the ophthalmic division than in non-MS patients[26]. The most likely cause of MS related TN is reported to be a pontine plaque damaging the intra axial primary trigeminal afferents[26]. Nevertheless, in some patients a neurovascular contact may act as a concurring mechanism[28], especially in older individuals with MS.

Glossopharyngeal neuralgia (GN) occurs in 0.5% of patients with MS[24]. GN refers to a unilateral pain that is located in the field of sensory distribution of the glossopharyngeal or vagus nerve (i.e., the ear, the base of the tongue, the tonsillar fossa or beneath the angle of the jaw). Pain is usually severe and stabbing and may remit and relapse similar to TN. In addition, pain is commonly provoked by stimuli such as swallowing, talking, coughing, yawning and eating spicy foods. Patients with GN report an abrupt severe pain in the throat or ear that lasts seconds to minutes.

Attacks are usually stereotyped in the same patient and can be associated with coughing paroxysms, excessive salivation, hoarseness and rarely syncope[24]. Pain that results from symptomatic GN is the same as classical GN. In addition, aching pain may persist between paroxysms, and sensory impairment may be found in the distribution of the glossopharyngeal nerve[29]. Symptomatic GN is secondary to inflammatory lesions of MS involving the root entry zone of cranial nerve IX in the brainstem[30], while in some individuals vascular compression of the glossopharyngeal nerve could independently cause glossopharyngeal neuralgia[31].

Occipital neuralgia (ON) is a paroxysmal pain syndrome that is characterised by jabbing pain in the distribution of the greater or lesser occipital nerves or in the third occipital nerve[24]. It can be accompanied by diminished sensation or dysesthesia in the affected area. The greater occipital nerve is involved in 90% of ON cases, whereas the lesser occipital nerve is only involved in 10% of cases. Interestingly, combined effects on both occipital nerves have been shown to be involved in 8.7% of ON cases[32]. MS can cause irritation of the occipital nerves and induce ON[32]. There are no data about the incidence or prevalence of ON in MS. In a retrospective study of 255 MS patients, 5 developed intermedius, retroauricular, or occipital neuralgia during a 25-year follow-up[33].

Demyelination of the motor nerves may present in the orofacial region as muscle weakness, tremor, hemi facial spasms and myokymia (involuntary facial muscle contractions)[34,35]. Sensory neuropathy secondary to MS may present as bilateral, progressive and often irreversible paresthesia commonly involving the second and third divisions of the trigeminal nerve. These may be accompanied by extra oral or intraoral numbness, tingling or pain[4]. Facial paralysis may occur in a quarter of MS patients in the advanced stages of disease[36]. There is a high prevalence of temporomandibular joint (TMJ) disorders symptomatology (pain on mouth opening, difficulty in mouth opening, TMJ sounds in MS which may be attributed to the underlying myofascial and neck pain[37].

Painful tonic spasms of the facial muscles are specific to MS[38]. These are unilateral or bilateral, stereotyped, involuntary muscle contractions that last less than 2 min and may manifest several times a day. They can be triggered by touch, movement, hyperventilation, or emotions, and are, though seldom, preceded by a "somesthesic aura". They may start from the face and spread to the adjacent part of the body. Their prevalence ranges from 6 to 11%. The spasms originate in the central nervous system from hyperactivity in the central motor fibres, caused by lesions in the internal capsule, cerebral peduncle, medulla, or spinal cord[38].

MS seems to be associated with headache, with studies reporting a frequency between 13 to 64%.The prevalence of migraine is three times higher in patients with MS than in the general population, 34 versus 10%, as estimated by the European Brain Council[39], whereas the prevalence of tension-type headache is similar, 21 versus 20-34%, as estimated by epidemiological studies in European Countries[40]. It is hypothesized that headache, and migraine in particular is believed to be a result of repeated demyelination of brainstem structures[41]. Treatment of MS with interferon beta may induce *de novo* headache and exacerbation of pre-existing headache[42].

Drugs used in the treatment of MS can lead to a variety of adverse effects on the orofacial tissues and these are listed in Table II[43-50]. Orofacial pain (acute/chronic) may significantly limit a MS patient in carrying out routine activities, reduce oral intake and affect the quality of life.

Diagnosis

Multiple sclerosis is diagnosed based on the presenting signs and symptoms, in combination with supporting medical imaging such as Magnetic Resonance Imaging (MRI) and laboratory testing from cerebrospinal fluid (oligoclonal bands and raised IgG index) and evoked potential studies (delayed evoked response with preserved waveform)[51]. It can be difficult to confirm, especially early on, since the signs and symptoms may be similar to other medical problems such as neuromyelitis optica (Devic disease), acute dissemi-

Table II. Medications used in MS with their possible oral side effects[43-50].

Class of drug	Possible side effects
Treatment of acute attacks	
Corticosteroids	Increased infection risk due to immunosuppression,
Prednisolone, methylprednisolone	Delayed wound healing
Immunosuppressants	
Methotrexate, Azathioprine, Mycophenolate, Cyclophosphamide	Mucositis, ulcerative stomatitis, opportunistic oral infection
Disease modifying therapies	
Interferon-β drugs	
IFN-1α (Avonex)	Glossitis, dysgeusia, xerostomia, headache,
IFN-1α (Rebif)	Increased risk of oral opportunistic infections
IFN 1β (Betaseron)	(Candidiasis), mucositis, ulcerative stomatitis
Glatiramer acetate (Copaxone)	Mucositis, ulcerative stomatitis, salivary gland Enlargement, increased risk of opportunistic oral infections,
Monoclonal antibody	Mucositis, ulcerative stomatitis, headache, risk of
Natalizumab (Tsyabri)	Opportunistic oral fungal/viral/bacterial infections
Fingoloimod (Gilenya)	Headache
Immunosuppressant	
Mitoxantrone (Novantrone)	Mucositis, ulcerative stomatitis, increased risk of Oral infections
Symptomatic therapy	
Tricyclic antidepressants	
Amitryptaline	Blurred vision, xerostomia,
Muscle relaxants	
Baclofen, Tizanidine	Visual hallucinations, xerostomia
Anticonvulsants	
Gabapentin, carbamazepin	Gingival hyperplasia, xerostomia, alveolar bone loss (level of evidence 1a)[50]
Anticholinergics	
Oxybutinin, tolterodine	Xerostomia

CNS: central nervous system.

nated encephalomyelitis, migraine, cerebral neoplasm, compression injuries of the spinal cord, infections (Human Immunodeficiency Virus, Syphilis), recurrent infarcts and paraneoplastic syndromes[51]. The McDonald et al[52] criteria (level of evidence IV) which focus on clinical, laboratory and radiologic evidence of lesions at different times and in different areas is the most commonly used method of diagnosis. While the McDonald et al criteria (most recently revised in 2010)[53] allow for a non-invasive diagnosis, some state that the only definitive proof is an autopsy or biopsy (level of evidence IV)[54] where lesions typical of MS are detected. However, biopsy is rarely utilized for this purpose. The cornerstone of diagnosis is involvement of disseminated plaques in time and space, indicating evidence of multiple lesions of the central nervous system (CNS) and the occurrence of distinct symptomatic episodes at least 30 days apart[1]. MRI is highly sensitive for detecting plaques in the CNS and therefore the most useful imaging test for confirmation of MS (level of evidence 1 a)[55]. Sensory evoked potential testing assesses function in CNS sensory pathways and is beneficial in demonstrating the presence of sub clinical lesions along these path-

Table III. Oral health care considerations in patients with multiple sclerosis (level of evidence IV)[88-91].

Time	Oral health care considerations
Before treatment	Dental clinics to be designed to accommodate wheel chairs Consult a physician for diagnosis of MS and treatment considerations Examine for TMJ dysfunction, dental caries, periodontal disease, bruxism, malocclusion Consider use of sedation/general anesthesia in patients with significant neurological symptoms or anxiety
During treatment	Appointment times and lengths to suit individual comforts Use mouth props to minimize the strain on masticatory muscles Avoid supine position Maintain a comfortable temperature in the outpatient setting to minimize heat intolerance Rule out dental causes for orofacial pain complaints before referral to experts for diagnosis and management
After treatment	Provide written as well as oral instructions to both patient and his/her accompanying person Avoid prescription of NSAID's in patients with peptic ulcers Decrease dosages of analgesics in patients with hepatic/renal impairment Antibiotic prophylaxis in patients on immunosuppressants, bone marrow suppression and those with risk of opportunistic oral infections
Home oral care	Use toothbrushes with built-up handles (or cut a small slit in the sides of a tennis ball and slide it onto the handle of the toothbrush); use flossing tools; consider electric toothbrushes and flossing devices. Brush twice daily Sit to brush and floss, if standing at the basin is tiring Floss at bed time without fail Allow a family member or personal assistant to help with tooth brushing/flossing Manage tremors by wearing a weighted glove while brushing The effects of xerostomia can be managed by sipping water or sugarless drinks Often, avoiding caffeine, tobacco and alcohol, using a small squirt of lemon in the mouth or sugarless lemon candies to stimulate parotid salivary flow, using a humidifier at night, using salivary substitutes to moisten the dry mouth Use fluoridated toothpaste Use a tongue scraper or brush to clean the tongue daily Eat a well-balanced diet without excessive sweets Replacing your toothbrush at least every 3 to 4 months Promptly reporting any bleeding gums, tooth or jaw pain or tooth sensitivity

NSAID: non steroidal anti-inflammatory drug.

ways or in providing objective evidence of lesions suspected on the basis of subjective symptoms[52]. Cerebrospinal fluid analysis is most useful in ruling out infections or neoplasms mimicking MS. Serologic testing may help rule out other conditions[56].

Recently, a set of 122 micoRNA (miRNA) biomarkers have been identified in blood for an improved diagnosis of MS as well as for monitoring of disease activity and treatment response[57]. The best single miRNA marker, hsa-miR-145, allowed discriminating MS from controls with a specificity of 89.5%, a sensitivity of 90.0%, and an accuracy of 89.7%[57]. Further, deregulation of miRNA's may play a role in the pathogenesis of MS.

Prognosis in Ms

The average life expectancy is 30 years from onset, being 5 to 10 years lower than that of unaffected people[1]. Female sex, relapsing-remitting subtype, optic neuritis or sensory symptoms at onset, few attacks in the initial years and especially early age at onset, are associated with a better course. Almost 40% of people with MS reach the seventh decade of life[58]. However, two-thirds of the deaths are directly related to the consequences of the disease. Suicide is more common, while death results from complications in a debilitated patient[1]. The signs and symptoms, particularly pain, contribute to an inability to complete daily routine activities, ambulation and/or mobility. Complications of MS may lead

to permanent impairment and/or disability. About 50% of MS patients remain ambulatory and can carry out daily activities unhindered while one third develop clinically significant paraparesis, paraplegia or tetraplegia[21]. Although most people lose the ability to walk before death, 90% are capable of independent walking at 10 years from onset and 75% at 15 years[58].

Management of Oral and Craniofacial Manifestations

Pharmacological therapy for TN shows good efficacy at the beginning of the disorder, and complete pain relief is generally observed in 80% of cases. Unfortunately, the drugs used to treat TN progressively lose efficacy over time[59]. In a systematic review of studies of drugs to treat trigeminal pain in MS from 1966 to 2010, Solaro and Uccelli[60] listed carbamazepine, lamotrigine combined with gabapentin, carbamazepine in combination with gabapentin, lamotrigine alone, gabapentin alone, topiramate, and misoprostol depending upon small open label (Class IV) studies. The authors state that the ability to draw conclusions and the evidence to treat with these medications are indeterminant due to small sample sizes and lack of randomized placebo controlled trials. Currently, the poor level of evidence for the treatment of GN and ON does not allow for the creation of guidelines. Interestingly, carbamazepine , phenytoin, gabapentin and baclofen (all with level of evidence III)[61,62] have all shown some efficacy in the treatment of GN. In addition, carbamazepine, gabapentin and pregabalin (all with level of evidence III)[63] play a major role in the conservative treatment of classical and symptomatic ON. The use of NSAIDS is not routinely recommended for treating neuralgic pain[64]. There are isolated reports of neuralgia responsive to NSAIDS and opioids. It is postulated that neuralgia responsive to NSAIDS is more likely to be due to some unknown acute inflammation. Opioids have been used as an adjuvant to the front-line neurogenic agents with limited success[65]. ON, which has recently been associated with the new onset of active inflammatory medullary cervical lesions (C2) during the course of myelitis and MS, has shown a satisfactory response to high-dose intravenous methyl prednisolone[66]. In addition, intravenous steroid therapy has been successfully administered in cases of GN that present at the onset of MS, and the steroid therapy has resulted in complete pain relief[66].

Non-responders or patients who are intolerant of drug side effects may be considered for a more interventional management, including surgery. Percutaneous procedures such as balloon microcompression of the gasserian ganglion (level of evidence III)[67], Gamma knife radiosurgery [GKR][68] (level of evidence III) and microvascular decompression (MVD)[69-71] (level of evidence III), percutaneous retrogasserian glycerol rhizotomy (PRGR) (level of evidence IIa)[72], are the most common surgical options for symptomatic TN in MS patients. Percutaneous procedures should be considered as suitable options in unhealthy or elderly patients. There are no reports regarding the application of neurosurgery for GN or ON in MS patients.

Painful tonic spasm of facial or pharyngeal muscles can be treated with antiepileptic agents (level of evidence Ib)[59], lidocaine (level of evidence Ib)[73], botulinum toxin (level of evidence Ib)[74] and cannabinoids (level of evidence Ib)[75].

Painful burning dysesthesias are treated with tricyclic antidepressants (TCAs) such as amitriptyline[76] or antiepileptics such as gabapentin[76]. While physiotherapy may ameliorate malposition-induced TM joint and muscle pain, additional drug treatment with paracetamol (acetaminophen) or NSAIDs may be useful[76].

Eye pain is treated with intravenous methyl prednisolone and nonsteroidal anti-inflammatory drugs (NSAIDs) such as ibuprofen[77]. Headache is managed with NSAIDs[59] initially and when non responsive with sumatriptan (level of evidence 1a)[78]. Prophylaxis is achieved with agents such as TCAs (amitriptyline, level of evidence 1b)[59] or antiepileptic drugs (topiramate, level of evidence 1b)[79]. Dysphagia is a result of oral and pharyngeal muscle fatigue and can be managed with changes in the nature of diet, pharyngeal electrical stimulation (level of evidence 1b)[80] or botulinium toxin (level of evidence III)[81].

Oral Healthcare Considerations

In the mid 1980's, a controversy brew that mercury present in amalgam restorations precipitated or exacerbated MS symptoms. This assumption was based on reports of miracle cures coincident with replacement of amalgam with non mercury containing restorative materials[82]. However, scientific evidence strongly refuted such an association[82-84]. The finding of such miracle cures may have been associated with incidental resolution of the underlying pathology or start of a remitting phase in the disease process[85].

Oral health in patients with MS may be compromised by difficulty in performing oral hygiene procedures (due to motor disturbances/weakness, spasticity, trigeminal neuralgia, facial pain, numbness/tingling) and in access to oral health care (patients with significant fatigue and/or mobility impairment)[86]. Studies have indicated a significantly greater occurence of periodontal disease[87] and dental caries[88] among MS patients when compared to the general population. This association may be indicative of the increased difficulty that MS patients face in cleaning their teeth and/or performing other oral hygiene procedures.

The focus of oral health care in MS is on prevention and/or reduction of dental or periodontal disease, maintenance of oral hygiene and facilitating access to stress free oral care. The oral health care professional must be aware of potential interaction of drugs used in MS and commonly prescribed medications in oral health care. Particular care must be exercised while prescribing non steroidal anti-inflammatory drugs, narcotics and acetaminophen where interactions between these drugs may result in hepatoxicity, cytotoxicity, alter the metabolism of certain drugs and/or amplify fatigue, myalgia and depression[4]. Knowledge of possible side effects from MS medications may aid the oral healthcare provider to minimize or manage these undesirable effects from both a systemic and oral health perspective. The most common side effects from MS medication that may pose a challenge to oral health care are xerostomia, mucositis/ulcerative stomatitis, gingival hyperplasia, dysgeusia and opportunistic infections like candidiasis, angular cheilitis and reactivation of herpes viruses[4].

Oral health care in individuals with MS must be modified to their special needs (Table III). The optimal period to treat MS patients is during periods of remission, as neurological symptoms are minimal and patients may be in their best physical and psychological health[89]. Patients with MS must not be placed in a supine position as there is a risk of pulmonary aspiration of dental materials/instruments/crowns secondary to dysphagia[37]. The habit of flossing at night must be reinforced as it eliminates the chance of bacterial multiplication while patients sleep[90,91]. The entire oral health care team at the out patient setting must participate in all features of oral care such as advice, skills, motivation, verbal reassurance, support throughout the treatment phase of the patient as well as after that to contribute to good oral health, improve patient attendance at oral health clinics[92] and enhance the general well being of the patient.

Conclusions

MS patients are all unique individuals with a wide variety of abilities and needs. Good oral health is extremely important, however, achieving this goal may present unique challenges. Considering the fact that there are an ever increasing number of patients diagnosed with MS, the onus is on oral health providers to recognize the condition and depending on the manifestations of MS and the related medications, create a treatment plan, educate, and make oral hygiene care recommendations accordingly. The mobility, dexterity, and spasticity of the patient will need to be considered when scheduling office visits and deciding on oral care routines with the patient. A multidisciplinary approach to treatment involves the neurologist, neurosurgeon and physiotherapist.

Conflict of Interest

The Authors declare that they have no conflict of interests.

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