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Neuromusculoskeletal disorders following SARS: a case series

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Objective: *To detail the presentation of three health care workers diagnosed with sudden acute respiratory syndrome (SARS) who later presented to a CMCC teaching clinic with neuromusculoskeletal sequelae and underwent conservative treatments. This case series aims to inform practitioners of the potential pathogenesis of these neuromuscular complaints and describes their treatment in a chiropractic practice.*

Clinical Features: *Three patients presented with a variety of neurological, muscular and joint findings. Conservative treatment was aimed at decreasing hypertonic muscles, increasing joint mobility, and improving ability to perform activities of daily living.*

Intervention and Outcome: *The conservative treatment approach utilized in these cases involved spinal manipulative therapy, soft tissue therapy, modalities, and rehabilitation. Outcome measures included subjective pain ratings, disability indices, and return to work.*

Conclusion: *Three patients previously diagnosed with SARS presented with neuromusculoskeletal complaints and subjectively experienced intermittent relief of pain and improvement in disability status after conservative treatments.*
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KEY WORDS: SARS, neuromusculoskeletal, manipulative therapy

Objectif : *Présenter en détail le cas de trois travailleurs de la santé chez qui on a diagnostiqué un syndrome respiratoire aigu sévère (SRAS). Ils se sont ensuite présentés à la clinique d'enseignement du CMCC avec des séquelles neuromusculosquelettiques et ont suivi des traitements conservateurs. La présente série de cas a pour objectif d'informer les praticiens sur la pathogenèse potentielle de ces douleurs neuromusculaires et de décrire leur traitement dans le cadre de la pratique de la chiropractie.*

Caractéristiques cliniques : *Trois patients présentaient une variété de troubles neurologiques, musculaires et articulaires. Les traitements conservateurs avaient pour objectif de réduire l'hypertonicité musculaire, d'augmenter la mobilité articulaire, et d'améliorer la capacité à réaliser des activités de la vie quotidienne.*

Intervention et résultat : *Les traitements conservateurs utilisés dans ces cas comprennent : des manipulations rachidiennes, manipulations des tissus mous, modalités et rééducation. La mesure des résultats comprend : évaluations subjectives de la douleur, taux d'invalidité et retour au travail.*

Conclusion : *Trois patients chez qui on avait diagnostiqué le SRAS présentaient des douleurs neuromusculosquelettiques et ont connu un soulagement subjectif intermittent de la douleur ainsi qu'une amélioration de l'état d'invalidité après des traitements conservateurs.*
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MOTS CLÉS : SRAS, neuromusculosquelettique, thérapie manuelle

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Introduction

Severe acute respiratory syndrome (SARS) is an infectious disease caused by the novel SARS-corona virus (SARS-CoV) that caused a global outbreak in 2003 and resulted in serious mortality and morbidity.[1–3] As a novel virus presenting as an atypical pneumonia,[4] SARS created a diagnostic challenge. To aid in disease management, the World Health Organization defined suspected cases as: "disease in a person with a documented fever (temperature >38°C), lower respiratory tract symptoms, and contact with a person believed to have had SARS or a history of travel to a geographic area where there has been documented transmission of the illness."[4] Further, a suspected case involving findings of pneumonia on chest radiograph, acute respiratory distress syndrome, or unexplained respiratory illness resulting in death with autopsy results demonstrating the pathology of acute respiratory distress syndrome without an identifiable cause was considered a "probable case."[4]

From November 1, 2002 to July 31, 2003, 8098 probable cases and 916 deaths were reported internationally.[5,6] In Canada, there were 438 suspected and probable cases resulting in 44 deaths, with the majority identified in Toronto, ON.[7] During the SARS outbreak, 769 health care workers (HCW) at nine Toronto hospitals had treated patients with suspected or probable SARS. HCW were particularly vulnerable during the early weeks of the outbreak as viral loads peaked at 10 days following symptom onset.[5] During this time, many HCW were exposed to virus via direct patient contact, particularly prior to a diagnosis of suspected SARS.[5,7–9]

The clinical course of SARS was characterised by fever, myalgia, and other systemic symptoms that generally improved after a few days, followed by a second phase with recurrence of fever, oxygen desaturation, and radiological progression of pneumonia.[1,2] Due to this clinical picture, much of the literature to date has focused on the respiratory effects and long-term sequelae of infection,[1,4,5,7] however, little is known about myopathic or neurological complications.[5–8]

Muscle weakness and an elevated serum creatine kinase (CK) level occurred in more than 30% of the SARS-infected patients.[10] Elevation of CK is an enzymatic indication of muscle damage, thus, an increased level may have indicated myopathy in affected patients. It was suspected skeletal myopathy was primarily responsible for the elevation of serum CK as the levels of the cardiac enzyme were normal.[2] It has been suggested serum CK level may also be a reflection of the severity of the myopathy, as patients with higher CK levels demonstrated more substantial necrosis on autopsy.[2] In a prospective case series by Lee et al., elevated CK levels were also correlated with the requirement for intensive care or death; however, this finding did not reach statistical significance.[10] As greater than 60% of patients in their study initially presented with myalgia and objective muscle weakness, the authors suggested myopathy in SARS may be quite common.[10] In a post-mortem case series, Leung et al. examined specimens from the psoas or quadriceps femoris of eight subjects who experienced progressive myalgia and symmetrical (typically truncal) weakness, and identified a spectrum of myopathy.[2] To date, this small sample represents the only preliminary evidence reporting SARS-associated myopathy.

Neurologic manifestations have rarely been described, and the relationship, if any, between the SARS-CoV and neuromuscular problems is still relatively unknown.[11] In a case report by Chao et al., a female patient presented with objective lower limb weakness, absent Achilles deep tendon reflex and a stocking pattern of paraesthesia continued to have neuromuscular symptoms at two month follow-up.[11] Tsai et al. presented a case series of four patients (with no concurrent medical conditions, systemic illness or symptoms prior to the diagnosis of SARS) with varied neuromuscular disorders with onset of up to three weeks following the onset of SARS.[12] Similar to the case report by Chao et al., these symptoms persisted at follow-up (up to two months).[12] These cases highlight how little is known about the long-term sequelae of SARS and the potential need for ongoing care.[8]

As one of the largest primary contact health care professions in Canada, it is reasonable to expect a patient previously diagnosed with SARS or other serious viral infections may present to a chiropractor for diagnosis and treatment of neuromusculoskeletal complaints. Chiropractors should be aware of this possibility and the potential effects on prognosis, as the course of novel diseases like SARS and resultant sequelae are currently not well understood.

The purpose of this case series is to describe the presentation of three health care workers diagnosed with SARS who later presented to a CMCC chiropractic teaching

clinic with a variety of neuromusculoskeletal complaints. Further, it aims to inform practitioners of the potential pathogenesis of these neuromuscular complaints. It describes their treatment in a chiropractic practice and includes follow-up periods up to six years following the onset of SARS.

Case Series

Case 1

This case report involved a 38 year old female nurse diagnosed with SARS in March 2003. Following her diagnosis, she developed constant left hip pain of three-year duration. She had presented to a physiotherapist in 2005 and experienced short-term relief, and later presented to a CMCC teaching clinic on March 24, 2006. The pain developed insidiously and was described as dull and achy in character. The patient reported the intensity as three out of ten on a numeric pain rating scale (NPRS). It was aggravated by weight-bearing on the left (could not walk for greater than five minutes or sit for greater than 15 minutes) and relieved by traction mobilization and therapeutic exercise (physiotherapy), oxycodone (10mg twice daily, 5 days per week), Percocet (as needed (prn)) and diazepam (5mg, one-four per day). The patient reported the following as complications of SARS: respiratory difficulty (associated with chronic cough and diaphragmatic dysfunction), vision changes, bilateral peripheral neuropathy affecting her hands and feet, and easy bruising. The patient reported great difficulty maintaining her quality of life, and noted minimal sleep, poor appetite and diminished activity levels. At the time of presentation, she had not returned to work. Imaging revealed mild joint degeneration of the left hip on radiographs, and increased uptake in the sacroiliac joints bilaterally and left proximal tibiofibular joint on bone scan. On initial presentation, visual analog scale (VAS) was marked at 3/10 (9/10 at worst) and Oswestry Back Disability Index (OBDI) score was 58%.

On physical examination, the patient presented with a right antalgic list and excessive left hip flexion. During gait analysis, the patient favoured her right leg and was unable to perform a squat due to pain. A lumbar spine screen caused pain in the left low back and thoracic cage with active and passive right rotation. Active hip range of motion demonstrated reduced motion with pain in all ranges on the left (right side was full and pain-free) and passive testing was not tolerated due to pain. Orthopaedic testing of the hips could not be performed due to pain. Weakness with manual muscle testing was revealed in the left psoas major, quadriceps, adductors, gluteus maximus and bilateral hamstrings. Pain was elicited with palpation of the left psoas, proximal rectus femoris, piriformis and bilateral gluteus medius. Neurological examination findings were reported as unremarkable in the file.

The patient was diagnosed with left hip dysfunction with associated psoas contracture (differential diagnoses included symptomatic left hip degenerative joint disease and left hip capsular irritation). The plan of management consisted of treatment twice weekly for four weeks and included hip mobilizations, Active Release Techniques® (ART®) to affected muscles, therapeutic ultrasound (100% duty cycle, 3 MHz, 1 W/cm^2 for 7 minutes), sacroiliac and lumbar manipulations, and monitoring of exercises prescribed by the physiotherapist. Education regarding proper exercise technique and sleep hygiene was also provided.

Re-evaluation was performed after eight treatments and the patient reported positive effects with the prescribed treatment plan. No clinically relevant changes were noted on objective outcome measures; however, as VAS was recorded at 4/10 and OBDI score was 56%. Due to the reported subjective improvements and lack of evidence regarding the prognosis of musculoskeletal complaints, the patient requested to continue treatment. In the following years, periods of withdrawl of care were attempted with reported exacerbations of pain, and thus treatment plans with durations ranging between eight and 16 visits were carried out at the patient's continued requests. Prior to initiating each new treatment plan, a report of findings was provided; importantly, the patient was counselled on alternatives and the lack of evidence regarding her prognosis. In addition to the conservative treatments, numerous meetings and interprofessional communications with other members of the patient's health care team occurred to discuss appropriate referrals and ongoing management options. In total, 151 treatments using a variety of treatment approaches were provided for a number of conditions and complaints. At the last visit (April 2009), the patient reported to have stable symptoms, but had not returned to full-time employment. She was resigned to the fact she would not likely be able to work full-time or without modified duties in the future.

Case 2

This case report involved a 25 year old female paramedic diagnosed with SARS in March 2004. The patient was hospitalized in May 2003, however, had returned to work full-time by June 7 of that year. In January 2004, she was unable to perform essential job tasks due to a constant cough, and was later diagnosed with SARS in March 2004. She developed left hip pain in April 2004 and presented to a CMCC teaching clinic on June 29, 2005. The pain was rated as 7/10 (NPRS) and described as a deep, dull ache localized to the left hip. It became sharp with hip flexion and/or external rotation. It was aggravated by running and skating and partially relieved with naproxen (prn). The patient reported associated difficulties with gait (stumbling), and left lateral knee and foot pain. Relevant systems review included respiratory difficulties (perceived 'tight' sensation in chest), difficulty sleeping (due to pain) and easy bruising following her SARS diagnosis. Radiographic and magnetic resonance imaging of the involved regions were read as normal. On initial presentation, VAS was scored as 4/10 and OBDI as 26%.

On physical exam, the patient presented with decreased thoracic kyphosis and bilateral genu valgum. Gait revealed left foot lag with toeing in and weight-bearing on the lateral aspect of the left foot only. A lumbar spine range of motion screen was full and pain-free. Left active and passive hip flexion caused sharp pain at 90°, internal and external rotation were limited to 30° and painful, active abduction was limited to 40° due to pain, active adduction was extremely painful at 30°, and passive adduction was full but painful at end range. All other ranges were full and pain-free. Manual muscle testing demonstrated full strength (5/5) but intense pain during left hip flexion, adduction, internal and external rotation. Further, extension and abduction were graded 4/5 (compared to the right) without pain. Trendelenberg sign was observed on the left. Single and double leg squat caused pain in the left hip. Thomas and FABER tests recreated pain in the left hip; all other relevant orthopaedic tests were unremarkable. Pain was caused during palpation of the left pectineus, obturator externus, psoas and proximal rectus femoris. Neurological examination revealed normal sensation to light touch and sharp/dull testing in the L1-S1 dermatomes bilaterally, 5/5 strength in L4-S1 myotomes bilaterally, and 3+ patellar reflexes bilaterally, 3+ Achilles reflex on the right, 1+ Achilles reflex on the left.

The patient was diagnosed with left psoas contracture/tendinopathy and a plan of management including active and passive stretching of left psoas and rectus femoris, manual facilitation of gluteus maximus, ART® of psoas, rectus femoris, obturator externus, sartorius and pectineus was proposed. The patient was treated two times per week for four weeks.

Following eight treatments, the patient reported short-term subjective improvement and believed treatment to be important in maintaining her quality of life during re-evaluation; however, no clinically relevant improvements were observed via VAS (4/10) or OBDI (26%) scores. Improvement in gait was observed (the left foot was no longer lagging) and hip range of motion had improved such that she was able to put on shoes and socks without increased pain. Due to these improvements and lack of evidence regarding the prognosis of musculoskeletal complaints in patients previously diagnosed with SARS, the patient requested to continue with treatment. In the following years, treatment plans with durations ranging between eight and 16 visits were carried out to deal with the patient's presenting symptoms and incorporated a variety of treatment modalities, including spinal mobilizations. During this time, the patient's gait continued to improve, and communications with the patient's medical doctor and physical therapist to discuss appropriate shared management were an important component of the patient's care. Further, referral to a chiropodist was made for custom orthotics to assist with gait normalization. Periods of withdrawl of care were attempted, and though not tolerated completely, the patient did consent to (and tolerated) decreasing the frequency of treatments. Prior to initiating each new treatment plan, a report of findings was provided; importantly, the patient was counselled on alternatives and the lack of evidence regarding her prognosis. In total, 124 treatments using a variety of treatment approaches were provided for a number of conditions and complaints. The patient was discharged in November 2008. At that time, she had resumed skating and running, and had returned to full-time employment.

Case 3

This case report involved a 39 year old female respiratory therapist diagnosed with SARS in April 2003. Following a two-week isolation period in hospital and a two-week isolation period at home, she developed "constant pain all

over" and presented to a CMCC teaching clinic on June 6, 2007. On initial presentation, the patient reported burning, shooting, sharp and stabbing pain originating in the cervical and thoracic spine that radiated to the fourth and fifth fingers bilaterally. The pain was typically rated as four out of 10 (NPRS), but became 9/10 with light exercise, work activities, and stressful events. Short-term relief was obtained with massage therapy, heat and home exercises (as prescribed by a physiotherapist). She had returned to modified duties (two 12-hour shifts per week). The patient also reported difficulty sleeping, headaches, fatigue and difficulty concentrating, and her relevant systems review revealed respiratory sensitivity, heart palpitations and easy bruising. Medications included naproxen, nortriptyline, oral contraceptive pills, vitamin C and calcium. On initial presentation, VAS was marked at 5/10 and Neck Disability Index (NDI) score was 22/50.

On physical examination, the patient presented with anterior head carriage and lateral protraction of the right scapula without winging. Cervical and thoracic ranges of motion were full but caused local pain at end-range. Jackson's, Spurling's, and cervical compression tests all caused shooting pain to the ipsilateral lower thoracic spine, and bilateral Kemps tests caused ipsilateral facet pain without radiation. Bilateral cervical doorbell test referred pain to the left anterior thoracic cage; the left cervical doorbell test also referred pain to the left posterior thoracic cage. EAST manoeuvre demonstrated a gradual increase in pain and numbness with failure to maintain the test at 45 seconds. Bilaterally, Adson's, Reverse Adson's, Eden's and Wright's tests demonstrated decreased radial pulse amplitude and tingling of the involved forearm, medial hand and fourth and fifth fingers. Jump signs were elicited with thoracic spine palpation, and palpation of the trapezius, rhomboids, levator scapulae, scalenes and erector spinae (thoracic) bilaterally. Neurological examination revealed normal sensation to light touch and sharp/dull testing in the C5-T12 dermatomes, 5/5 strength in C5-T1 myotomes, and 2+ biceps, brachioradialis and triceps reflexes bilaterally.

The patient was diagnosed with cervicothoracic dysfunction and thoracic outlet syndrome, and a plan of management including mobilizations of the cervical, thoracic and costovertebral articulations, ART® to affected muscles and microcurrent (acupuncture point LI4, setting: 30/300) was proposed. The patient was treated twice per week for six weeks. Exercises prescribed by a physiotherapist were reviewed and monitored.

The patient did not attend a number of scheduled visits; therefore re-evaluation occurred after the eighth treatment. She reported short-term subjective improvement, and believed treatment to be important in maintaining her quality of life and allowing for return to modified duties. No clinically relevant improvements were observed via VAS (6/10) or NDI (23/50) scores. Due to the subjective improvements and lack of evidence regarding the prognosis of musculoskeletal complaints, a similar treatment plan was proposed at the patient's request. In the following years, withdrawl of care was attempted with reported exacerbations of pain, and thus treatment plans with durations ranging between eight and 16 visits were carried out at the patient's continued requests. Prior to initiating each new treatment plan, a report of findings was provided; importantly, the patient was counselled on alternatives and the lack of evidence regarding her prognosis. In total, 84 treatments using a variety of treatment modalities, including spinal mobilizations were provided for a number of conditions and complaints. In January 2009, the patient had returned to full duties and was placed on PRN (return at own request).

Discussion

The number of chiropractors treating patients previously diagnosed with SARS is unknown, however, with over 8000 cases reported during the global outbreak, it is certainly possible these patients may present in a chiropractic office. Readers should be aware of the limited body of knowledge regarding neuromusculoskeletal complaints associated with SARS and therefore, the difficulties in the determination of prognosis. Furthermore, other viral infections may present as neuromuscular disorders, and practitioners should be educated regarding the potential mechanisms of pathogenesis including direct action (viral myositis or neuritis), inflammatory reaction (immune mimicry), or via a systemic inflammatory response syndrome.[12]

Myopathy

Muscle weakness and an elevated serum CK level have been documented in patients infected by the SARS-CoV, however, little is understood about the mechanism of injury.[2,10] Though clinical trials to examine the pathogenesis

of SARS-associated myopathy are currently not available in the literature, the findings of case reports and series suggest it may be a common sequela of the infection.[2,10] A number of potential causes have been identified and warrant further investigation.

Cachetic myopathy has been suspected due to disuse following bed rest.[2,5,13] Patients commonly suffered from acute respiratory failure during the second phase of SARS and required bed rest which may have lead to deconditioning and muscle wasting.[5] While disuse is likely to play a role in muscle atrophy, it does not fully explain the necrosis and histochemical changes reported in the literature.

Due to the number of patients presenting with myalgia and an elevated serum CK level, a viral-induced myositis has been suggested.[10] During in situ hybridization and viral culture for SARS-CoV, the negative findings suggest the necrosis may be due to cytokine release which caused immune damage rather than viral infection of the skeletal muscles.[13] This theory was reinforced by the absence of viral particles observed during electron microscopy.[13]

The use of systemic corticosteroids as treatment for acute respiratory failure during the second phase of SARS has also been suggested as a potential contribution to the development of myopathy.[2,5] Corticosteroids have been purported to alter electrical excitability of muscle fibres, decrease the number of thick filaments, and/or inhibit protein synthesis.[5] Interestingly, patients who did not receive steroid therapy were not found to experience myofiber atrophy, further indicating the potential role of corticosteroid therapy in the development of myopathy.[2] It must be noted however, authors believe three to 10 days of steroid therapy (typical dose) alone was not adequate to explain the pathogenesis of myopathy, and stressed the need for investigation of other (or combined) causes.[2]

One such cause may be the development of critical illness myopathy (CIM), an acquired myopathy following acute or chronic disease. This disorder has frequently been observed in conditions requiring mechanical ventilation and high-dose steroid treatment.[2] It is believed to be caused by activated leukocytes infiltrating skeletal muscle and causing the release of pro- and anti-inflammatory cytokines, leading to axonal degeneration with preservation of the myelin sheath.[13] This disorder is characterized by a normal cerebrospinal fluid protein level, preservation of cranial nerve and autonomic function and a lack of lymphocyte infiltration of neurons.[13] Clinically, patients maintain sensation (via peripheral nerves), and testing reveals elevated serum CK and decreased thick filaments with fiber atrophy and necrosis on biopsy.[2,13]

Neuropathy

Similarly, neurologic manifestations of SARS have not been well described in the literature.[12] A relationship between the SARS-CoV and neurological symptoms has not been established; it is currently unknown if the virus has the potential to damage peripheral nerves directly or if the observed neuropathy is an immune mediated process.[12]

Critical illness polyneuropathy (CIP) has been suggested most commonly to explain the neurologic presentation following a diagnosis of SARS.[11,13] CIP develops as an acute neuropathy during severe illness and typically remits when the underlying illness is controlled.[11] An illness such as SARS could have produced elevated levels of proinflammatory cytokines, platelet activating factor, arachidonic acid, free radicals and proteases.[13] These factors could create a neurotoxic environment and lead to neuropathy.[13] If acute, practitioners must ensure the underlying systemic inflammatory response (sepsis) is medically managed and other causes (neurotoxic drugs, poisoning and nutritional deficiencies) are ruled out as there is no specific treatment for CIP.[13] Prognosis is unknown and may vary depending on the severity of the disease. It has been suggested that symptoms (especially weakness) may persist in those patients with a long duration of sepsis or those requiring long-term care in intensive care, however, actual durations of illness or treatment are not defined.[13] Chao et al. reported rapid improvement in neurologic status following extubation in a patient with severe respiratory symptoms.[11]

Clinical considerations

In the cases one and two, the patients were diagnosed with a psoas contracture. The psoas major may be related to the respiratory system due to its anatomical relationship with diaphragm. The psoas originates on the transverse processes and lateral aspects of the vertebral bodies of T12-L5 (and associated intervertebral discs). At its most superior attachment, the psoas is related to the medial and lateral arcuate ligaments, and the central tendon of the diaphragm.[14] In case three, the jump sign elicited during palpation of the scalenes highlights a more apparent involvement of accessory respiratory muscles. Though this

has yet to be discussed in the literature, the involvement of muscles related with respiration in all three cases is an interesting finding following a respiratory illness. The respiratory difficulties reported by the patients in this case series may have required increased involvement of accessory muscles and resultant muscular pain. Future clinical or anatomical studies may be warranted to examine the relationship between viral respiratory infections and related muscular complaints.

Interestingly, all three patients complained of easy bruising following SARS. In a review by Yang et al., thrombocytopenia was a common haematological change reported in patients with SARS, though the exact cause was not well understood.[15] Increased destruction and/or decreased production of platelets in damaged lungs may be a mechanism resulting in thrombocytopenia in severe pulmonary conditions.[15] Clinicians should be aware of this possibility and educate patients and/or modify treatment plans accordingly.

Prognosis

As indicated above, the long-term prognosis of SARS and its associated complications are unknown. Practitioners must be prudent to re-evaluate frequently and ensure patients are improving or maintaining pain/disability status. Certainly, any deterioration in health status requires further investigation and co-management as appropriate.

Law et al. presented a case series to examine factors affecting return to work in 128 health care workers in Hong Kong with musculoskeletal complaints two years following in the SARS outbreak.[8] These authors noted patients continued to experience difficulties in performing activities of daily living and work tasks despite receiving acute treatment and rehabilitation. Return to work (RTW) has been suggested as an important measure of prognosis; however, it is known that pain does not correlate well with RTW.[8] A number of important considerations beyond pain and functional ability impact a worker's ability and desire to return.[7,8] Factors such as support in the workplace, feasibility of providing alternate duties, and the worker's beliefs on the effects of return-to-work on their injury progression must be considered.[8]

The lack of evidence regarding prognosis of neuromuscular complaints in patients previously diagnosed with SARS must be clearly communicated to patients, however, it does not preclude treatment of conditions within the chiropractic scope. The current case series suggests the importance of appropriate use of outcome measures, both generic and disease-specific. Although patients may report short-term pain relief and positive effects on health related quality of life, outcome measures (VAS, NDI, OBDI) may not demonstrate clinically relevant changes. The inclusion of an outcome measure that allows the patient to identify specific limitations (such as the MYMOP[16]) or addresses overall health related quality of life (such as the SF-36[17,18]) may allow for the measurement of subjective improvement.

Conclusion

Myopathic and neuropathic complications in patients diagnosed with SARS have been reported previously; however, to our knowledge, this is the first case series to describe patient presentation in a chiropractic clinic. Furthermore, we believe this case series represents a longer follow-up period (up to six years following SARS diagnosis) than was previously available in the literature. Little is known regarding the cause of these neuromuscular symptoms, and even less is known regarding treatment options for these patients, particularly after the acute illness has been controlled.[2,5,11,13] Follow-up research should be conducted to obtain more information about the long-term outcomes of SARS.

Clinicians should be aware of the proposed pathogenesis of neuromuscular complaints with a previous SARS or other severe respiratory infections and ensure any differential causes have been ruled out prior to commencing a plan of management focused on conservative therapies.

In this case series, three patients with varied neuromuscular complaints reported short-term subjective improvements in their pain experience and quality of life, and two were able to return to work. Future research should investigate the role of conservative care and manual therapies for this type of patient population using subjective outcome measures.

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