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Orthopaedics

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The Cover Shot

Masonry bridge on old Stanley Road (赤柱古道)

Discovered by hikers in 2021, the bridge was part of the old Stanley Road, a bridleway built in the 1840s between Aberdeen and Stanley for horses and people on foot. In 1918, the new Stanley Gap Road, good for motorised vehicles, was built higher up the hill. Stanley Road was soon left abandoned and disappeared from maps. Due to the travel restrictions associated with COVID-19 in the past three years, local travel and hiking have become increasingly popular. Quite a number of similarly neglected historical architectures were rediscovered. This segment of the old Stanley Road is located between Chung Hom Kok Road and Ma Hang Prison on Carmel Road and is just a short walk from public transport stops.

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Editorial

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Editor

Dr WONG Yau-bun

On behalf of the Hong Kong Orthopaedic Association, I wish you a happy new year of 2023. Our association joined the Federation of Medical Societies of Hong Kong in the year 2022. We are delighted to participate in the publication of 2023 January issue of the Hong Kong Medical Diary.

There has been a lot of advancement and development in the field of orthopaedics in recent years. The use of robots in joint replacement surgery has become a recent trend. 3D printing technology has already been used in patient-specific implants, pre-op planning and intraoperative implant placement. 3D CT navigation technology has been used in minimal invasive surgery and treatment of fracture. We are keeping up with the advancement of technology and making use of these new tools to improve our surgical outcomes. We are also working closely with different specialties and various teams of rehabilitation to help patients get through their difficult time, especially for those patients with severe injury or complicated orthopaedic problems.

However, these newly developed surgeries and high-end technologies appear not to be so commonly seen in non-hospital-based practice. For medical practitioners of other specialties, the common orthopaedic problems encountered usually fall into the other end of the spectrum. Complaints such as back pain, knee pain, shoulder pain, and ankle pain probably are the mainstream of orthopaedic complaints faced by our first-line doctors in their daily practice.

We have invited orthopaedic doctors of various sub-specialties to write about those orthopaedic conditions that are commonly encountered in our community. I am particularly excited to have two renowned physiotherapy scholars from the Hong Kong Physiotherapy Association write us some updated physiotherapy treatments for shoulder problems. I hope you will enjoy this issue of the Hong Kong Medical Diary and find the information useful.

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Approach to Adult Scoliosis

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Dr WONG Kam-kwong

This article has been selected by the Editorial Board of the Hong Kong Medical Diary for participants in the CME programme of the Medical Council of Hong Kong (MCHK) to complete the following self-assessment questions in order to be awarded 1 CME credit under the programme upon returning the completed answer sheet to the Federation Secretariat on or before 31 January 2023.

INTRODUCTION

From time to time, we have encountered adult scoliosis in our daily practice. This article will describe the different types of adult scoliosis and the general approach to the management of adult degenerative scoliosis.

NON-STRUCTURAL AND STRUCTURAL SCOLIOSIS

In general, there are two categories of scoliosis, non-structural and structural. Non-structural scoliosis includes sciatic, postural, compensatory, inflammatory and hysterical types. Structural scoliosis includes congenital, neuromuscular, idiopathic, de novo, traumatic, and iatrogenic types.[1]

Adult scoliosis refers to all forms of scoliosis occurring in skeletally mature individuals with Cobb's angle of more than 10 degrees in the coronal plane.[1]

SCIATIC SCOLIOSIS

Sciatic scoliosis refers to non-structural scoliotic deformity in reaction to nerve root irritation.[3,4] It occurs more commonly in adolescents or young adults with lumbar disc prolapse, and the abnormal posture can be corrected once the painful stimulus is removed. Clinically, the patients present with uneven shoulder and trunk listing. Nerve root tension signs will be obvious. However, there is no obvious thoracic hump or loin prominence when the patient is asked to perform a forward bending test. Radiologically, it is characterised by listing the spine without obvious rotational deformity. The rotation measurement of the spine includes the position of the spinous process as well as the position and size of pedicles. (Fig. 1)

Although the mechanisms are not fully understood, these postural changes are believed to protect the spinal nerves from further damage.[4] In 1973, Finneson proposed that the trunk would list to the opposite side of the disc prolapse if the disc herniation is lateral to the nerve root, whereas the trunk would list to the same side of the disc prolapse if the herniation is medial to the nerve root. However, subsequent clinical studies by Matsui et al. and Suk et al. could not support the topographical position of disc herniation (medial or lateral to the nerve root) in relation to the direction of the sciatic scoliotic list.[4,6] In fact, there is a signification association (73 - 84%) of the coronal imbalance between trunk shift to the contralateral side and disc herniation. In other words, the symptomatic side with sciatica is more likely to be on the convexity side of scoliosis.[4,6-8] Matsui[4] stated that the intervertebral disc becomes wedge-shaped with the trunk shifting to the contralateral side, and the disc stretches and reduces in size resulting in lateral recess and foramen "expansion", which is later described as autonomic decompression attempted by the trunk posture.[6] All authors demonstrated satisfactory clinical outcomes and improvement of the sciatic scoliotic list after both traditional and endoscopic decompression.[4, 6-9]

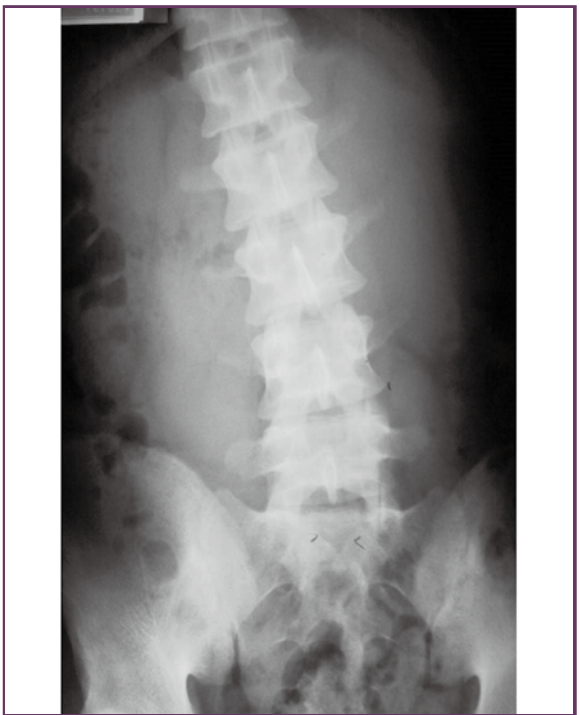

Fig. 1. Sciatic scoliosis: AP view of a lumbar spine showing listing to the right without obvious rotational deformity (Personal collection)

AEBI CLASSIFICATION BASED ON AETIOLOGICAL FOUNDATION TO UNDERSTAND THE NATURAL HISTORY OF ADULT SCOLIOSIS

Aebi Classification[10] is primarily based on aetiologies rather than on specific details of the deformity. There are four groups, namely Type I, II, IIIa and IIIb. The aetiologies may help predict the natural history of a deformity. The classification implies a generalisation of treatment approaches. Both Type 1 Primary degenerative scoliosis (de novo) and Type 2 adolescent idiopathic scoliosis (AIS) sequelae with the thoracic and/or lumbar spine that progresses into adulthood will be discussed in further details. Type III is further divided into two subgroups. Type IIIa consists of a secondary adult curve resulting from the disease either within the spine, such as an adjacent thoracic, thoracolumbar curve or lumbosacral transitional anomaly, or the disease outside of the spine, such as pelvic obliquity caused by leg length discrepancy. Type IIIb refers to the bone weakness, such as osteoporosis, as the more wedged or deformed vertebrae lead to deformity progression with kyphosis or scoliosis or in combination. However, Aebi Classification does not reflect the complexity of specific deformities for detailed surgical planning.[11] Schwab and SRS classification have included the spinopelvic parameters and regional/global spinal balance with functional measurement instruments, which in turn define the radiological realignment targets during surgical procedures.[12]

DISTINCTION BETWEEN DEGENERATIVE SCOLIOSIS AND AIS SEQUELAE

The primary pathology of Degenerative scoliosis is the asymmetric degeneration of the intervertebral disc and/or facet joints with secondary vertebral deformity, whereas the primary pathology of AIS sequelae lies in the vertebrae with secondary degenerative changes. In more detail, the pathogenesis of degenerative scoliosis started at asymmetric disc degeneration. The resulted hypertrophied but incompetent facet joints lead to multi-directional instability with reference to the translational and rotational plane. Asymmetric loading of the spinal segment causes three-dimensional deformity, and the presence of deformity triggers further asymmetric degeneration resulting in a vicious cycle. Finally, curve progression and ongoing degeneration result in central and foraminal stenosis.[10]

As for clinical features, Degenerative Scoliosis (de novo) more commonly presents with lumbar curve in late adulthood. Those patients tend to have more back pain, radicular symptoms and claudication when compared with AIS patients. Radiologically, it is associated with more degenerative changes at discs and facet joints or even kyphotic deformity. For those patients with AIS, they present with thoracic or thoracolumbar curves during adolescence. Their clinical presentations are less symptomatic except the deformity. It is associated with thoracic hypokyphosis and less degenerative changes at discs and facet joints.

SYMPTOMATOLOGY OF DEGENERATIVE SCOLIOSIS

There are four types of symptomatology, which can present alone or in combination.

Disabling Back Pain

Back pain is the most frequent complaint of adult degenerative scoliosis, but the pain generators are always difficult to be identified. The axial mechanical back pain becomes prominent with upright posture such as standing or sitting, and the symptom improves with lying down when the axial load is taken off from the spine; mechanically unstable segment should be suspected and dynamic imaging is needed.[13] Co-exiting lumbar kyphosis or significant sagittal imbalance should be looked for as possible pain generators. There are also concerns about the curve direction and asymmetric symptoms. Back pain at convexity may signify muscle fatigue or facet joint distraction pain, whereas back pain at concavity may represent facet joint destruction and disc space degeneration. S. Frank has demonstrated the statistical significant correlation between the severity of the axial back pain and thoracolumbar kyphosis, lateral listhesis, and the obliquity of specifically the L3 and L4 endplates, whereas no significant correlation was documented with patient age, Cobb's angle, the amount of sagittal imbalance and listhesis in his study.[14]

Lumbar Spinal Stenosis

With the asymmetric disc degeneration and facet joint hypertrophy, it is understandable that lateral recess stenosis and foraminal stenosis occur in the concavity side of the adult scoliosis. For the convexity side of the curve, the radicular symptoms can be produced by dynamic traction of the nerve roots.[13] Hence, bilateral leg pain can be accountable for central stenosis and possible bilateral radiculopathy in degenerative scoliosis.[15]

Acute Neurological Deficit

The acute neurological deficit with or without bowel and bladder sphincter problem is infrequent. The condition may happen when there is a sudden event due to a disc fragment herniation or acute curve decompensation.

Curve Progression

It has been said that degenerative scoliosis may progress at a rate of 3° or more per year.[13] In a prospective cohort study of adult degenerative scoliosis, statistically significant predictors of curve progression included curves with a Cobb's angle greater than 30 degrees, Grade 3 apical rotation, lateral listhesis of 6 mm or more and the prominence of L5 in relation to the intercrest line.[16]

CONSERVATIVE TREATMENT OF DEGENERATIVE SCOLIOSIS

Conservative treatment comprises lifestyle modification,

quitting smoking, weight control, core training exercises and a short period of bracing for pain relief. In several retrospective cohort systematic reviews, muscle deconditioning from chronic bracing could result in worsening curve progression rather than stabilisation of the deformity.[17,18,19] Pharmacological agents include non-steroidal anti-inflammatory drugs, opioid analgesics, muscle relaxants and neuropathic pain medications.[20] Osteoporosis treatment is another main concern as those patients who are elderly women suffering from vertebral collapse can trigger the curve progression. Interventional procedures, including epidural steroid injection, selective nerve root block and facet joint block, are found to be the short-term treatment modalities.[17,19,21]

SURGICAL MANAGEMENT OF DEGENERATIVE SCOLIOSIS

The goals of degenerative scoliosis surgery are (1) sagittal balance restoration, (2) symptomatic neural element decompression, (3) complication avoidance, and (4) improved quality of life.[22] The surgery will be challenging when the patients are of advanced age with multiple medical co-morbidities; hence patient-specific surgical treatment plan is required. Indications for surgery include disabling back pain despite conservative measures, new-onset or progressive neurological deficits, curve progression resulting in coronal and/or sagittal imbalance causing easy fatigue and worsened quality of life, cosmetic purpose requested by a fit surgical patient and radiographical parameters that predict curve progression.[17,19,23]

Surgical options comprise decompression alone, decompression with short segment fusion and long fusion to restore global and/or regional spinal alignment with or without decompression (Fig. 2 - 5), staged procedures, and posterior osteotomies. The decision of surgical plan should be dictated by the individual clinical presentation and patient factors. The challenges of surgery for adult degenerative scoliosis should be addressed. The conjoint care by orthopaedic surgeons, geriatricians and anesthetists is needed to minimise the high perioperative morbidity. The rigidity of deformity imposes difficulties of correction intraoperatively. Detailed preoperative imaging and planning are required. Deformed anatomy will be a difficulty for instrumentation, and navigation-guided surgery can play a role in facilitation. Intraoperative monitoring is always advised as there is a high incidence of neurological deficit.

CONCLUSION

Various types of adult scoliosis have been discussed, and various treatment options have been mentioned. Repeated assessments and in-depth consent discussions are required for the preparation of patient-specific treatment plans in order to address the individual symptoms with the corresponding pathoanatomy of the spinal deformity, expectations and realistic surgical outcomes and the risks and benefits of the surgical procedures.

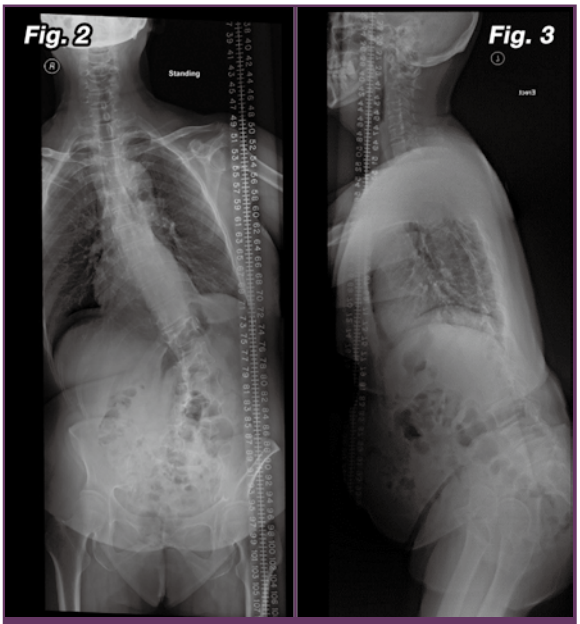

Fig. 2 and 3. Degenerative Scoliosis: Pre-op standing AP and Lateral view of the whole spine showed T12-L5 cobb's angle 44 degrees with convexity to left and sagittal imbalance. (Personal collection)

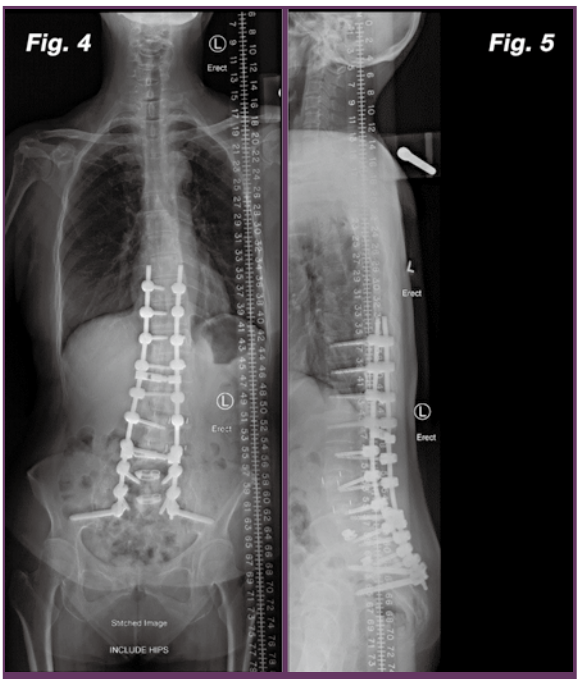

Fig. 4 and 5. Degenerative Scoliosis: T10-ilium posterior instrumentation and fusion with OLIF L2-4 was performed. Post-op standing AP and Lateral view of the whole spine showed the improvement in sagittal and coronal profile. (Personal collection)

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Dermatology Quiz

Dermatology Quiz

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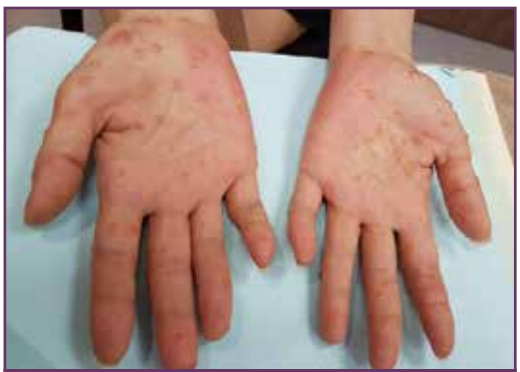

Fig. 1: Multiple small itchy vesicles on hands

This 48-year-old lady complained of having multiple small vesicles on both hands and fingers for months. They were very itchy. The lesions were precipitated by housework. Physical examination revealed multiple intact small vesicles on the hands and fingers with an erythematous rash on the palm (Fig. 1).

Questions

1. What is the diagnosis of her skin lesion?
2. What investigation(s) are you going to order?
3. How do you treat this patient?

(See P.32 for answers)

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MCHK CME Programme Self-assessment Questions

Please read the article entitled "Approach to Adult Scoliosis" by Dr WONG Kam-kwong and complete the following self-assessment questions. Participants in the MCHK CME Programme will be awarded CME credit under the Programme for returning completed answer sheets via fax (2865 0345) or by mail to the Federation Secretariat on or before 31 January 2023. Answers to questions will be provided in the next issue of The Hong Kong Medical Diary.

Questions 1-10: Please answer T (true) or F (false)

1. In general, scoliosis can be categorised into structural and non-structural types.
2. Sciatic scoliosis usually presents without nerve root tension signs.
3. Sciatic scoliosis is caused by the prolapsed intervertebral disc.
4. Significant improvement of sciatic scoliosis can be achieved by spinal decompression.
5. Non-structural causes include degenerative, neuromuscular and congenital types.
6. Aebi classification of adult scoliosis is primarily based on the aetiology.
7. Patients with degenerative scoliosis less commonly present with back pain and radicular symptoms when compared with those with adolescent idiopathic scoliosis sequelae.
8. To manage degenerative scoliosis, co-exiting lumbar kyphosis or significant sagittal imbalance secondary to possible pain generators should be looked for.
9. As for conservative management, muscle deconditioning with chronic bracing can cause worsening curve progression.
10. To manage degenerative scoliosis, osteoporosis treatment should be addressed as vertebral collapse in elderly women can trigger the curve progression.

ANSWER SHEET FOR JANUARY 2023

Please return the completed answer sheet to the Federation Secretariat on or before 31 January 2023 for documentation. 1 CME point will be awarded for answering the MCHK CME programme (for non-specialists) self-assessment questions.

Approach to Adult Scoliosis

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1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ 6 ☐ 7 ☐ 8 ☐ 9 ☐ 10 ☐

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Answers to December 2022 Issue

Immune Checkpoint Blockade in the Management of Haematological Malignancies

1. T 2. F 3. F 4. F 5. T 6. F 7. F 8. F 9. T 10. F

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Management of Achilles Pathology

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INTRODUCTION

Heel pain is a very common complaint encountered in primary care settings. In this article, a group of Achilles tendon disorders and their management will be reviewed.

ANATOMY

The Achilles tendon arises from the gastrocnemius and soles muscles, and is attached to the posterior aspect of the calcaneum. There is no true synovial sheath but only a single layer of paratenon. The paratenon is highly vascularised. The blood supply enters anteriorly. There is a hypovascular area about 2-6 cm proximal to the insertion, which is a common site of rupture.

PATHOLOGY

The aetiology of Achilles tendon pathology can be classified into internal and external factors. Internal factors include decreasing blood supply with advanced age, and corticosteroid or fluoroquinolone use. External factors include high-intensity plyometric exercise, training on unfamiliar surfaces and improper footwear.

INSERTIONAL ACHILLES TENDINOPATHY

As indicated in its name, the site of the pathology is located at the insertion of the Achilles tendon. The tendinous fibres at the insertion on the calcaneus start to degenerate. The prevalence of insertional Achilles tendinopathy ranges from 6.5% to 18% in runners and 9% in dancers[1]. It is an inflammatory process and is associated with Haglund deformity (Fig 1), which is a prominence located at the posterosuperior aspect of the calcaneal tuberosity, with overlying bursitis and tendinopathy. Symptoms include pain and stiffness at the back of the calcaneus, morning pain which worsens with activity, and painfulness at the mid-portion of the posterior aspect of the calcaneus.[1] MRI will show microtear, intrasubstance abnormalities, increased signal in the calcaneus, or enlarged retrocalcaneal bursa. MRI will also show calcaneal oedema, which is exclusively seen in symptomatic patients.[1] Hence MRI is useful to confirm the diagnosis. Non-operative management includes a brief period of immobilisation allowing for gradual integration of reduced load-bearing activities, physiotherapy, and intra-lesional injection using agents such as platelet-rich plasma. Surgical management involves removing the diseased portion of the tendon, osseous prominence which is irritating the tendon, and the inflamed bursa.

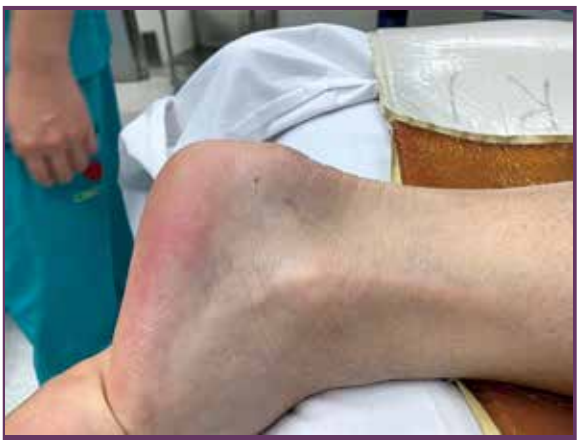

Fig. 1. Haglund deformity (Personal collection)

Retrocalcaneal bursitis is another entity of insertional tendinopathy. The bursa anterior to the Achilles tendon is inflamed, hypertrophied and adherent to the tendon, which will be irritated when compressed during ankle dorsiflexion. MRI will show partial Achilles tendon rupture, with peritendinous thickening, tendinosis, or ossification. Two-finger squeeze test[2] is a test for this condition. By applying medial and lateral pressure anterior to the Achilles tendon, the pain will be elicited. Non-operative treatment includes NSAIDs, modification of training regimens, eccentric strengthening, heel lift and immobilisation. Surgical procedures include debridement and decompression of the bursa, resection of the osseous prominence, and repair of the tendinous insertion. Up to 50% of the tendon attachment can be debrided without a high risk of rupture. The procedure can be done openly via resection of the Haglund prominence down to the insertion, or endoscopically (the latter being known as endoscopic calcaneoplasty).

NON-INSERTIONAL TENDINOPATHY

Non-insertional tendinopathy consists of paratendinopathy, paratenodinopathy with tendinosis and tendinosis alone. In paratendinopathy, the paratenon appears thickened and adherent to the normal tendon. Histology reveals inflammatory cell infiltration and capillary proliferation. Clinically the

patient complains of swelling and pain, with bedside findings of crepitus, local tenderness and warmth at the site. In paratendinopathy with tendinosis, the paratenon is inflamed together with intratendinous degeneration. There is failed healing response. Histology reveals additional loss of tendon collagen, fibre disorientation and scattered vascular ingrowth. Clinically there is a palpable asymptomatic tendon nodule. For tendinosis, it is signified by an early inflammatory infiltrate followed by a failed healing response and ultimately tendon degeneration. Histology reveals a non-inflamed appearance with collagen degeneration, hypocellularity, local necrosis, areas of calcification and minimal vascular ingrowth. It is often painless and without swelling. However, it is prone to injury.

Treatment of non-insertional tendinopathy is mainly non-operative, and includes modification in the training regimen, NSAID, heel lift or shock-absorbing orthotics, and night splint for heel cord tightness. There is moderate evidence for extracorporeal shock wave therapy, insufficient evidence for ultrasound or laser therapy, corticosteroid or platelet-rich plasma injection, and poor evidence for sclerosing injection or prolotherapy.[3] Surgical treatment is mainly for recalcitrant cases. The goals are to resect degenerative tissue and to augment the tendon if needed. The surgical approach can be percutaneous longitudinal tenotomy or minimally invasive stripping of the tendon.

ACHILLES TENDON RUPTURE

Achilles tendon rupture often happens in healthy, vigorous, young adults (mean age 37 - 43.5 years) or older, sedentary patients. It usually starts with prodromal calf or heel pain. Up to 90 - 100% of cases occur in active, forceful, unexpected foot plantar flexion e.g. pushing off with the weight-bearing foot while extending the knee, sudden unexpected dorsiflexion of the ankle, or violent ankle dorsiflexion of a plantarflexed foot. It most commonly ruptures at 3 - 4 cm proximal to its insertion on the calcaneus, as this is the avascular zone. Histology of the rupture site may show degenerative changes. Other factors contributing to rupture include age-related degeneration, poor tendon vascularity, gastrocnemius-soleus dysfunction, male sex, changes in training pattern, previous injury, choice of footwear and poorly conditioned individual being subjected to sudden overloading. Six percent to 26% of patients also rupture the contralateral tendon.

Basketball and racquetball sports consist of half of all injuries in the U.S. Patients may present with sudden snap in the heel region, pain with ankle plantar flexion, or difficulty with weight-bearing activity. Thirty-three percent of patients have prodromal symptoms. Physical examination shows a palpable gap and a positive Thompson test. However, it was reported that up to 25% of correct diagnosis was missed at the time of initial evaluation as a result of a large haematoma disguising the tendon defect. Plantar flexion power was retained secondary to extrinsic foot flexors. Positive Thompson test only occurs in 80% of chronic cases. X-ray can rule out concomitant fractures or detect calcific tendon changes. The disruption of the normal triangular pre-Achilles fat pad (Kager triangle) is a sign of rupture. Ultrasound is an inexpensive investigation, and can give a dynamic assessment. MRI can assess the extent of retraction and gapping and detect partial ruptures.

Non-operative management is usually reserved for the medically unfit patient. The tendon should be able to approximate as confirmed by ultrasound study. A cast or boot with an elevated heel wedge can be worn for two weeks to allow the haematoma to consolidate. A functional brace can be applied to allow plantar flexion exercise using variable-resistance bands and a dorsiflexion stop. Several reviews compare functional bracing with surgical repair. Results show that open surgical repair of acute Achilles tendon ruptures significantly reduced the risk of re-ruptures when compared with non-operative management.[4] There is no significant difference in the re-rupture rate between operative and non-operative treatment when similar functional postoperative mobilisation protocols were employed.[5] However, only 37% can return to the same level of the sport.

Operative management options include open repair (with or without augmentation), percutaneous repair, and limited open repair. The technique of open repair consists of a longitudinal incision along the medial aspect of the tendon, using modified Bunnel, Kessler, Krackow, triple-bundle suture method, and multilayer closure to prevent postoperative tendon adhesion to skin. Percutaneous repair is a less invasive method. There is no direct exposure to the tendon rupture site. There is a lower incidence of wound breakdown, or healing complications when compared with open repair. Mini-open repair (Fig. 2 & 3) involves a small incision that allows direct visualisation of the ruptured tendon ends and enables debridement of the diseased tendon at the same go. Patients can return to walking, stair climbing and sports in significantly shorter time compared with those undergoing standard open repair.[6] During the rehabilitation phase, a short period of immobilisation to minimise wound complications is required, followed by early protected weight-bearing and early mobilisation. This approach has decreased the time to return to activities, including work, sports and normal walking.

Fig. 2. The instrument used in mini-open Achilles tendon repair (Personal collection)

Fig. 3. The scar after mini-open repair is shown by the arrow. (Personal collection)

In conclusion, Achilles tendon pathology is common in athletes and non-athletes alike. The pathology is multifactorial, involving biomechanics and tendon degeneration. Non-operative management is the mainstay for Achilles tendon inflammation and degeneration. There is a large body of evidence addressing the management of acute and chronic Achilles tendon rupture. However, controversy remains.

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Non-Surgical Management of Osteoarthritis of the Knee

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INTRODUCTION

Knee osteoarthritis is one of the most common degenerative diseases in Hong Kong. The demand for joint replacement surgery is substantial, with over 30,000 patients waiting for surgery in the public sector; the 90th percentile for waiting time varies between 46-103 months.[1] Whilst surgical treatment is effective in end-stage osteoarthritis, there is abundant evidence to support the use of non-surgical management regimens in various stages of knee osteoarthritis, including non-pharmacological and pharmacological modalities. International organisations, including the American Academy of Orthopaedic Surgeons (AAOS), Osteoarthritis Research Society International (OARSI), European Society for Clinical and Economic Aspects of Osteoporosis, Osteoarthritis and Musculoskeletal Diseases (ESCEO) and American College of Rheumatology (ACR)/Arthritis Foundation, have published guidelines based on recommendations for various treatment modalities. These guidelines focus on individual treatment modalities via extensive literature review by international panels and via critical appraisal of evidence, with resultant recommendations based on the strength of available evidence. This review article summarises the recommendations from the above practice guidelines, reviews the latest literature and focuses on modalities available in Hong Kong.

NON-PHARMACOLOGICAL TREATMENT

Core Treatment

Patient education, exercise, weight reduction and self-management programmes are strongly recommended by all guidelines and should form the core of any OA knee treatment plan. Patient education should focus on education on the disease, joint protective measures, exercise regimen, medications, and side effects. Modern self-management programmes should be structured and group-based, facilitated by multidisciplinary input from physiotherapists, occupational therapists, nurses and physicians.[2] Exercise therapy can be supervised or un-supervised, land-based or water-based, but most importantly exercise therapy should be focused on the patient's preference and access. Land-based therapy comprises strength training, active range of motion exercise and aerobic exercise. It is worth noting that the ACR guidelines strongly recommend practising Tai Chi in OA knee for its benefit in strength, balance, fall prevention and psychological well-being.[2] Water-based exercise has shown small, short-term, clinically relevant improvement in pain, disability and quality of life in patients with OA knee.[3] Weight reduction in overweight patients is recommended in all guidelines with a target of 5% weight loss in 20 weeks for treatment to be efficacious.[4]

Biomechanical Interventions

Knee sleeves, braces and foot orthosis are non-invasive treatment options widely available on the market. While most guidelines offer a moderate recommendation for a tibiofemoral knee brace for improving function, pain, and quality of life in patients with OA knee[5,6], compliance remains an issue under the hot and humid local weather. Lateral wedge insoles, on the other hand, are strongly not recommended by AAOS and ACR guidelines due to limited supportive evidence. While clinical guidelines did not address soft knee bracing for OA knee, a recent meta-analysis has shown that soft knee braces exert a moderate effect on improving pain and self-reported physical function.[7]

Walking Aids

A cane or a walking aid could be used to reduce pain and improve function in knee OA. The ACR guidelines and AAOS guidelines have strong and moderate recommendations supporting the use of canes, respectively.[5,6]

Transcutaneous Electrical Nerve Stimulation (TENS)

While TENS has been proven to be a safe treatment modality, inconclusive evidence exists regarding the use of TENS in OA knee treatment. AAOS offers limited recommendation for its application in OA knee.[5]

Ultrasound

There is some evidence to suggest possible benefits of ultrasound therapy, but the quality of evidence is low.[8] The recommendation for its use in managing OA knee from OARSI is uncertain.[6]

Acupuncture

Despite the large volume of literature that tried to investigate whether acupuncture is helpful in patients with knee osteoarthritis, many trials have been criticised

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for inappropriate blinding, the validity of sham controls, sample size and effect size miscalculations. The ACR guidelines conditionally recommend its use in OA knee due to positive trials indicating effectiveness in analgesia with minimal risks.[2] On the other hand, due to inconclusive evidence, AAOS downgraded their recommendation to limited recommendation in the latest guideline.[5]

PHARMACOLOGICAL INTERVENTIONS

Topical NSAIDs

Topical non-steroidal anti-inflammatory drugs (NSAIDs), while being as comparably effective as oral NSAIDs[9], provides limited systemic exposure and thus are better tolerated. A recent meta-analysis showed topical NSAIDs were superior to placebo in reducing pain and improving function, amongst which diclofenac was most effective.[10] Topical NSAIDs have a lower risk of gastrointestinal (GI) side effects but have a higher risk of dermatological side effects. Topical NSAIDs are thus preferred to oral NSAIDs in OA patients ages ≥ 75 years and those with associated GI, cardiovascular or renal comorbidities. ESCEO recommends topical NSAIDs as a cyclical add-on in symptomatic patients despite core treatment prior to the use of oral NSAIDs.[11]

Oral Non-Selective NSAIDs

For patients unresponsive to core treatment, oral NSAIDs should form the mainstay of treatment for OA regardless of anatomical location and is the recommended first choice oral medication.[2] While their short-term efficacy is widely proven, consideration of patient's comorbidities and potential side effects on gastrointestinal, cardiovascular and renal system are to be accounted for.[9] All NSAIDs carry a higher risk of kidney injury, especially in the first month of initial treatment. While kidney injury rarely occurs in patients with normal preexisting kidney function, patients with diabetes, hypertension and congestive heart failure are more prone to complications.[12] All NSAIDs, therefore, should be taken at the lowest possible dose for the shortest possible duration. NSAIDs of any class are not recommended by OARSI in patients with cardiovascular risks and frailty.[6] All NSAIDs, selective or non-selective, increase cardiovascular risk. Compared with diclofenac and ibuprofen, Naproxen has lower cardiovascular risk due to suppression of platelet aggregation.[13]

Gastroprotective agents (e.g. proton pump inhibitors PPI) and H2 antagonists are proven to be effective in reducing gastric ulcers compared with placebo in non-selective NSAIDs[9]. PPIs are recommended to be prescribed concomitantly with NSAIDs by ESCEO, while OARSI recommends co-prescription only in patients with moderate risk.

Oral COX-II Inhibitors

COX-II inhibitors are associated with a lower chance of GI ulcers but carry a higher risk of cardiovascular events. While the risk of GI side effects is lower than non-selective NSAIDs, the risk is still higher than placebo.[13] Celecoxib and etoricoxib are the only two agents remaining in Hong Kong. ESCEO recommends celecoxib over non-selective NSAIDs due to its efficacy and safer GI side effects profile.

Oral Acetaminophen

Oral Acetaminophen (paracetamol) was often prescribed as a first-line medication treatment for OA knee. Emerging evidence suggests only a small effect size in managing OA pain, while side effects with long-term use, including GI side effects and multiorgan failure, were underestimated in the past.[6] Conservative dosing and treatment duration are thus recommended by OARSI and ACR.[2,6] AAOS is the only guideline that still strongly recommends its use.

Oral Narcotics

Oral narcotics, including tramadol, are associated with higher adverse events, particularly in the elderly aged over 60 years of age. Side effects include GI (dry mouth, oral ulcers, nausea, vomiting, dyspepsia and constipation), central nervous system (headache, dizziness and fatigue) and skin reactions. AAOS recommends against their use, while ACR and ESCEO conditionally recommend their use as the last pharmacological treatment before surgery or when NSAIDs are contraindicated. Tramadol is preferred over other opioids.

Oral/Dietary Supplements

Glucosamine and chondroitin are one of the most used dietary supplements for patients with knee OA. While many patients perceive them as effective, there is a considerable discrepancy in their efficacy between industry-sponsored and public-funded research, not to mention the placebo effect. Toxicity with glucosamine rarely occurs, although elevated serum glucose has been reported. It is worth noting that different preparations of glucosamine exist, including prescription crystalline glucosamine sulphate (pCGS) and glucosamine hydrochloride (GHCl) and studies demonstrating effects are on pCGS rather than GHCl.[14,15] The ESCEO concluded that despite a small effect size of pCGS on pain (0.27), it was higher than that of acetaminophen (ES=0.17); hence ESCEO strongly recommends the use of pCGS[11] and is against the use of non-pCGS glucosamine preparations. The ChONdroitin versus Celecoxib versus Placebo Trial (CONCEPT) found that chondroitin sulphate (800 mg/day) and celecoxib (200 mg/day) showed a significantly greater reduction in pain than placebo and such finding forms the basis of the strong recommendation by ESCEO.[16] Other dietary supplements investigated include vitamin D, fish oil and ginger extract, but the evidence is lacking to support their use. The strength of a recommendation from AAOS and OARSI on using dietary supplements for OA knee are limited and uncertain, respectively.[5,6] The ACR is strongly against using dietary supplements in OA knee due to limited evidence rather than safety concerns.[2]

INTRAARTICULAR INJECTIONS

Intraarticular Steroid

Clinical trials have demonstrated short-term efficacy of intraarticular steroid injection for symptomatic relief in OA knee. A recent randomised controlled trial has shown steroid injection resulted in greater cartilage volume loss in knee osteoarthritis compared with saline, though the clinical significance remains uncertain.[17] The ESCEO recommended its use, particularly in the setting of clinical effusion where aspiration is immediately followed by corticosteroid injection (methylprednisolone acetate or triamcinolone hexacetonide).[11] The ACR guideline strongly recommends its use, particularly when compared to other injection forms, such as hyaluronic acid.[2] The AAOS has a moderate recommendation for its use.

Intra-articular Hyaluronic Acid

The role of intraarticular hyaluronic acid in the treatment of knee osteoarthritis remains controversial. While most meta-analyses have shown small but significant benefits[18], others did not demonstrate improvement over saline.[19] The ESCEO guidelines suggest that injection of hyaluronic acid induces longer lasting effects compared with steroids, may have similar efficacy compared with oral NSAIDs and may delay joint replacement surgery. The ESCEO guidelines support their use and recognise their role in older patients contraindicated for NSAIDs since they are relatively safe.[11] The OARSI guidelines conditionally recommend their use over steroid injection for their longer-lasting effect beyond 12 weeks and for their more favourable long-term safety profile.[6] The AAOS and ACR guidelines do not recommend its routine use in symptomatic knee osteoarthritis due to a lack of evidence on efficacy.

Intraarticular Platelet Rich Plasma (PRP)

The lack of standardisation and heterogeneity of available preparations make it difficult to investigate what is actually being injected. This is also why OARSI and ACR guidelines strongly recommend against its use in knee osteoarthritis.[2,6] The AAOS guideline is the only guideline offering limited recommendations that PRP may reduce pain and improve function.[5]

Stem Cell Injections

Similar to PRP, lack of standardisation, heterogeneity of preparations and poor quality evidence have led the ACR, and OARSI guidelines to strongly recommend against the use of stem cell injections.

CONCLUSION

Non-surgical management of symptomatic knee osteoarthritis consists of non-pharmacological and pharmacological interventions which should be tailored to the patient's specific needs. The core non-pharmacological treatment includes exercise, weight loss, a walking aid and self-management programmes. Pharmacological treatment involves topical NSAIDs, oral NSAIDs or COX-II inhibitors. Intraarticular injection of corticosteroids is helpful in the short term, while hyaluronic acid is potentially helpful for a longer duration. Finally, if conservative treatment fails, modern knee replacement surgery offers good clinical outcomes and survivorship and provides hope to patients with end-stage knee osteoarthritis.

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14 February 2023	Complaint system The rights-, interest-, and power-based complaint system Complaint system design - with resolution and preventive focus	Dr. Ludwig TSOI
21 February 2023	Complaint – is somebody at fault? Complaint system of Medical Council and other regulatory bodies	Dr. Robert LAW
28 February 2023	Media in complaint Handling media in adverse events	Dr. Carl LEUNG
7 March 2023	Complaint – how-to Practical tips on handling complaints and how to survive a legal action	Ms. Suk-chong LEUNG Ms. Asha SHARMA
14 March 2023	Complaint – what's new Just culture, open disclosure and apology handling	Dr. Kai-ming CHOW
21 March 2023	Patients' complaint Patients' complaint avenue in HK What motivate patients to complain What they want and deserve	Dr. Kim-lian ONG

Date : 14, 21, 28 February & 7, 14, 21 March, 2023 (Tuesday)
Duration of session: 1.5 hours (6 sessions)
Time : 7:00 pm – 8:30 pm
Course Feature: Video lectures (with Q&A platform for participants to post the questions)
Quiz for doctors: DOCTORS are required to complete a quiz after the completion of each lecture
Language Media : Cantonese (Supplemented with English)
Course Fee : HK$1,000
Certificate : Awarded to participants with a minimum attendance of 70% (4 out of 6 sessions)
Deadline : 7 February 2023
Enquiry : The Secretariat of The Federation of Medical Societies of Hong Kong
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Common Orthopaedics Misconceptions

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INTRODUCTION

Many common orthopaedic misconceptions are seen in our day-to-day clinical practice. Frozen shoulder or, in local terms, "50-years-old shoulder/五十肩" is a common orthopaedic shoulder problem we encounter in our daily practice. The root of misconceptions arises from the fact that many conditions could mimic or even co-exist with a frozen shoulder, the former getting easily mixed up with the latter, resulting in wrong diagnosis and treatment, eventually leading to big consequences. The following paper hopefully clarifies some concepts and brings about a better treatment outcome for our patients.

FROZEN SHOULDER

The formal name is "adhesive capsulitis". There is tightening and thickening of the joint capsule, reducing the range of motion and causing pain. Under the microscope, the appearance of the shoulder joint capsule is very similar to the tissue which stops the fingers from moving in Dupuytren's contracture[1], a fairly common condition where the little finger curls into the palm.

Frozen shoulder can be mimicked in a variety of conditions: referred pain arising from the neck to the shoulder, shoulder impingement caused by rotator cuff tendinitis or even rotator cuff tear. Calcification found in the rotator cuff is one common condition that can easily be mixed up with a frozen shoulder. All these conditions represent different problems associated with different prognoses and treatments; hence they should be distinguished from each other.

The local term "50-year-old shoulder /五十肩", often missed, generally describes patients with shoulder problems at around 50 years old. This is a common term used to refer to frozen shoulders in Southeast Asia, including Japan, Korea, the Southern part of Mainland China, and Taiwan.

It is called "50-year-old shoulder/五十肩" because people get adhesive capsulitis most commonly at around this age. True frozen shoulder affects about 2% of the general population. It mostly affects people between 40 and 60 years of age, with no clear pattern of sex and arm dominance. It is more common in people with diabetes (up to 20%) and heart disease.

True frozen shoulder can be divided into three different stages:

Stage 1: "Painful or freezing" stage is often used in a patient who experiences shoulder pain of gradual onset without any predisposing injuries. The patient will still have a reasonable range of motions; it is especially characterised by pain at the end range in all directions, including forward flexion and abduction, and is particularly noticeably painful at external and internal rotations. The duration of this period can last from 2 - 9 months.

Stage 2: "Stiffening or Frozen" stage is when a patient starts to have more pain than usual and often, this pain is so severe that most of the time, it is unbearable, and hence patient will start to seek help from healthcare workers. It is especially characterised by pain which awakens the patient at night. During this phase, the shoulder range of motion will dramatically be diminished. It is more obvious on internal and external rotations. The patient will have a lot of limitations and be debilitated. For example, if patients are being told to perform internal rotation, they will often be able to reach the buttock only. They complained of difficulties in changing, especially doing and undoing their underwear. This phase usually lasts for 4 to 6 months.

Stage 3: "Resolution or Thawing phase", the beginning of which is seen when the patient's night symptoms are diminishing and resting pain improves. The patient's range of motion will improve eventually. This phase usually lasts for 1 to 2 years. Up to 95% of patients will have complete recovery. The natural history of this debilitating disease will resolve spontaneously in around two years.[2]

HOW IS FROZEN SHOULDER DIAGNOSED?

The emphasis on history and clinical examination in traditional clinical teaching works very well in helping us diagnose frozen shoulder most of the time. Most importantly, from the history, the patient will often complain of a gradual onset of shoulder pain without any previous injuries. A physical examination will show that there is global diminution in the range of motions in all directions.

Upon physical examination, if a patient not only presents with a diminished range of motions but demonstrates weakness of the rotator cuff function, then images at this juncture such as X-ray and MRI, can help us rule out other precipitating causes, such as a rotator cuff tear, calcific tendinitis, or even cervical radiculopathy.

WHAT ARE THE MOST COMMON MISCONCEPTIONS?

As said earlier, a proper history taking and physical examination will enable one to achieve the correct diagnosis. However, some other conditions can easily confound the picture, and if treatment of a frozen shoulder is offered alone without addressing other diagnoses, wrongly given treatment may sometimes lead to untoward consequences.

Here is a typical clinical scenario I would like to share with you. A 50-year-old patient presented with shoulder discomfort for six months after an outstretched hand injury. The injury itself did not cause many concerns for the patient: minor pain and slight difficulty in lifting the shoulder as a result of minor pain and impingement. Nonetheless, the patient was still able to perform most of the activities of daily living with minimal compensation. It was initially thought that the pain would eventually fade away, but after six months, because the pain was still there, they decided to seek help. When a healthcare worker attended to the patient, since the patient presented with a shoulder problem at the age of 50, they gave a diagnosis of a frozen shoulder. As a result, the patient was treated conservatively. Her symptoms did not improve after a year, and she noticed increasing weakness in shoulder movement. Finally, an MRI was performed, and she was found to have a massive rotator cuff tear with retraction and muscle atrophy. (Fig. 1) At this stage, it would be extremely difficult to treat the tear, and the patient could end up with debilitated function until the age of 70, when a reverse shoulder arthroplasty could be performed. If the problem had been correctly diagnosed at an earlier time, the patient would have been able to go through a minimally invasive procedure to restitch the rotator cuff back to the bone, with a likely improved outcome.

Fig. 1a. Normal Rotator cuff

Fig. 1b. Complete tear of Rotator cuff

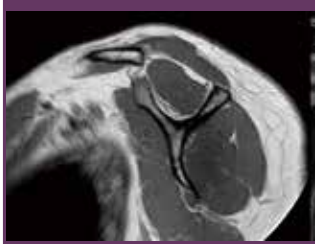

Fig 1c. Normal muscle bulk of rotator cuffs

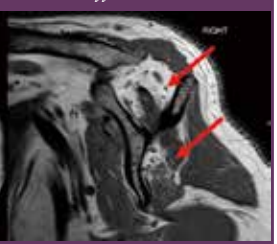

Fig 1d. Muscle wasting and atrophy of rotator cuffs

(Personal collection)

ADHESIVE CAPSULITIS TREATMENTS

By the natural healing process of the body, adhesive capsulitis will be mostly resolved after 1.5 - 2 years. This implies that even if the patient is not treated, their condition will improve. Hence, as medical providers, we aim to use symptomatic treatment to enhance the quality of life. There are two main factors that inform and guide the manner in which treatment takes place: the degree of pain and the extent to which the stiffness negatively affects the patient. If both factors are negligible, the patient should perform stretching exercises at home every day. That said, daily exercise at home may not be able to instantly resolve the condition; the aim is to prevent the patient's condition from deteriorating and to stretch out the tight capsule over time. If pain is the patient's main complaint, especially night pain that frequently disrupts sleep, but joint stiffness is not so debilitating, the patient should undergo a steroid/cortisone shot into the shoulder joint to suppress the active inflammation, dramatically alleviating the pain.

The majority of local patients will likely have concerns about steroid injections, but locally used steroids are effective, relatively safe to use, and have minimal side effects. If the patient is in a lot of pain and suffering from debilitating stiffness, surgical intervention through capsular release and MUA will dramatically improve their condition and enable them to be more active in daily life. Normally, patients are required to wait for 1.5 - 2 years for recovery, but surgery can lead to improvement in approximately one month after it has been carried out. However, this treatment should only be prescribed to those who struggle with severe stiffness, for example, an obese patient who is incapable of dressing and cleaning themselves due to their stiff joint.[3]

CONCLUSION

Frozen shoulder/adhesive capsulitis/五十肩 is an extremely common orthopaedic shoulder condition that is often found in patients. However, due to its prevalence, this condition is often overlooked. Medical history and physical examination remain the main diagnostic tools, and images are only needed to exclude associated conditions such as calcific tendinitis, rotator cuff tear, cervical radiculopathy, or fracture dislocation.

The main aim of treatment is to resolve the underlying cause of adhesive capsulitis. It is important to note that the patient's age should not cloud judgement when diagnosing this condition.

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Physiotherapy Management of Rotator Cuff-related Shoulder Pain

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INTRODUCTION

Rotator cuff-related shoulder pain, also known as rotator cuff tendinopathy/tendinitis/tendinosis and subacromial impingement syndrome, is a common musculoskeletal disorder. The patient mainly complains of pain and weakness during shoulder elevation and external rotation. It does not only affect participation in sports but also most of the activities of daily living. The pathology may range from symptomatic rotator cuff tendinopathy to partial- and full-thickness tears.

Physiotherapists play a major role in managing patients with rotator cuff-related shoulder pain.[1] Physiotherapy intervention has been shown to induce significant improvement in pain and function at three months compared to no physiotherapy.[2] A systematic review also found physiotherapy has similar effects as surgery both in the midterm and long term for pain, function and quality of life for certain groups of patients.[3,4] This article aims to summarise the evidence for selected treatments commonly used by physiotherapists in Hong Kong.

ULTRASOUND THERAPY

Thermal therapeutic effects of ultrasound may include improved circulation and increased enzymatic activity, increased collagen tissue extensibility and reduced muscle spasm (Fig. 1). Non-thermal effects may include affecting diffusional change between cell membranes to alter cell function/enhance healing and micro-massage effect. A number of studies have investigated the short-term and long-term effects of ultrasound in rotator cuff-related shoulder pain. Only low-quality studies demonstrated that therapeutic ultrasound therapy did not offer a better outcome than placebo intervention[5], while a 2016 Cochrane systematic review suggested that ultrasound may have only a short-term effect over placebo in treating rotator cuff tendinopathy, with low-quality evidence.[6]

ACUPUNCTURE

Acupuncture is commonly used by physiotherapists in treating musculoskeletal disorders. The research evidence on the effectiveness of treating musculoskeletal disorders in extremities is somewhat inconsistent.[7] A systematic review of 28 randomised trials concluded that acupuncture might have short-term benefits of reducing pain and enhancing shoulder function among individuals with rotator cuff disorders (low-quality evidence)[8], while a high-quality systematic review of 9 randomised controlled trials demonstrated that dry needling was effective in reducing pain and disability caused by subacromial syndrome.[9]

TRANSCUTANEOUS ELECTRICAL NERVE STIMULATION (TENS)

Transcutaneous electrical nerve stimulation (TENS) primarily stimulates sensory nerves to produce pain relief by either activating the pain gate mechanism or the opioid system (Fig. 2). Two systematic reviews published in 2015 and 2016 found that the effect of TENS in treating rotator cuff tendinopathy was inconclusive, because of the limited number of studies and low methodological quality of the reviewed studies.[6,10]

Fig. 1. Ultrasound therapy (Personal collection)

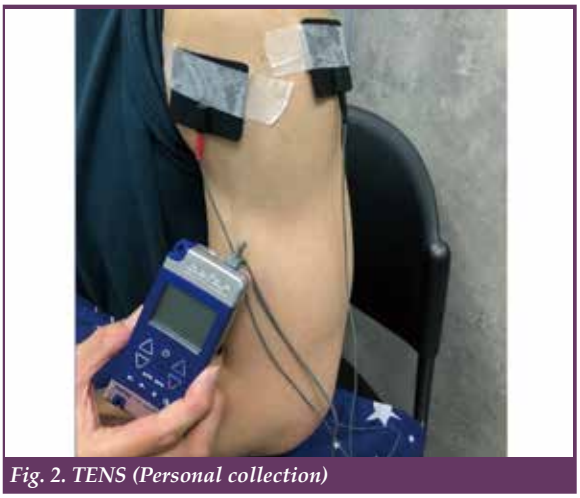

Fig. 2. TENS (Personal collection)

SHOCKWAVE THERAPY

Shockwave therapy is becoming more and more popular in treating musculoskeletal disorders in Hong Kong (Fig. 3). Physiological therapeutic effects may include stimulation of an inflammatory response to increase local blood flow and enhance tissue repair responses, increase in cellular activity, transient analgesic effect on afferent nerves, and breakdown of calcific deposits. The review by Moya et al.[11] found some evidence to support the use of shockwave therapy in treating rotator cuff tendinopathy. A systematic review from Harniman et al.[12] revealed moderate evidence to support the use of high-energy shockwaves in treating chronic calcific rotator cuff tendonitis but not non-calcific rotator tendon issue. Another systematic review of 17 studies by Huisstede et al.[13] also found that shock wave therapy has no evidence to treat non-calcific rotator cuff tendinopathy. However, the same review found moderate evidence to support the use of shockwave therapy to treat calcific rotator cuff tendinopathy.[13]

Fig. 3. Shockwave therapy (Personal collection)

TAPING

Taping is commonly used in sports physiotherapy to protect the injury structure, unload the irritating tissue, and correct the mechanical alignment. Taping has been demonstrated to have some efficacy in pain-free shoulder range, while the effect on pain reduction and improvement of functional movement in patients with rotator cuff tendinopathy was inconclusive.[14] The authors attributed the inconsistent findings to the substantial heterogeneity of subjects across studies.[14] A randomised controlled study of 60 subjects with rotator cuff injuries demonstrated that Kinesio tape could help to reduce pain and enhance the effect of an exercise programme.[15] In contrast, another randomised controlled study of 52 individuals found that adding kinesio tape to exercise therapy did not have a better outcome in pain reduction, functional ability, range of motion and disability level (as measured by the disabilities of the arm, shoulder and hand questionnaire or DASH), when compared with the exercise only treatment group among patients with rotator cuff related shoulder pain[16].

MANUAL THERAPY

Manual therapy is one of the core treatments used by physiotherapists in treating musculoskeletal disorders. Proposed effects involve mechanical stimulus on treating soft tissue, including muscle, ligament, tendon, joint capsule, fasciae and nerve. While the mechanical stimulus effect cannot explain all the treatment effects of manual therapy, neurophysiological mechanisms have also been proposed to explain the analgesic effect induced by manual therapy.[17] A systematic review and meta-analysis by Desjardins-Charbonneau et al. demonstrated that manual therapy could reduce pain among patients with rotator cuff tendinopathy (low to moderate evidence).[18] Another systematic review by Peiters et al.[19] found moderate to high-level evidence that exercise rehabilitation programmes with manual therapy had a better pain reduction effect in the short term than shoulder exercise therapy only.

EXERCISE THERAPY

The beneficial effects of exercise therapy in treating musculoskeletal pain disorders are well established (Fig. 4, 5 and 6). The proposed mechanisms may involve the modulation of the central and peripheral pain pathways, enhancement of immune function and reduction of pain-related fear.[20] A number of trials have reported the effectiveness of exercise therapy in people with subacromial pain. A systematic review by Haik et al.[21] suggested that exercise should be first prescribed for reducing pain and regaining range of motion and function. In a randomised controlled study involving 232 subjects, exercise therapy after corticosteroid injection for people who have subacromial impingement syndrome with moderate to severe shoulder pain showed a significantly greater improvement in pain and disability index (SPADI) at weeks 1 and 6, while the between-group difference was no longer apparent at week 24.[22] The authors suggested that the reduction of pain after corticosteroid injection may facilitate exercise therapy and achieve better pain relief and less disability. Another systematic review by Hanratty et al.[23] also found that exercises effectively treated subacromial impingement syndrome. Another systematic review by Littlewood et al.[24] suggested that exercise therapy was supportive in treating rotator cuff tendinopathy. Still, it was difficult to make a solid conclusion because the reviewed studies are of low-quality. However, different exercise intervention protocols were used in the reviewed studies. In a systematic review of 7 clinical trials, Gutierrez-Espinoza et al.[25] concluded that both supervised physiotherapy and home exercise programmes were equally effective in treating subacromial impingement syndrome in terms of improving shoulder range of motion and function, and pain relief.

Apart from pain relief and regaining range of motion, exercise therapy is also used to correct scapular dyskinesis. Excessive loading over the rotator cuff is always claimed to be one of the contributing factors to the development of rotator cuff tendinopathy.[26] Therefore, the correct kinematic pattern of the scapula and humeral movements, and thoracic mobility are very important to unload the symptomatic tendon.[26]

Fig. 4. Resistance exercise for muscle strengthening using dumbbell (Personal collection)

Fig. 5. Resistance exercise for muscle strengthening using pulley system (Personal collection)

Fig. 6. Mobilisation exercise (Personal collection)

A systematic review and meta-analysis about the effectiveness of scapular-focused approaches in patients with rotator cuff-related shoulder pain demonstrated significant improvement in pain and reduced disability in the first six weeks, but no difference was found after three months.[27] However, another systematic review of 15 studies revealed a lack of high-quality studies to examine the reliability of the tests used to evaluate scapular dyskinesis.[26] The systemic review highlights the need to develop a reliable and valid assessment to examine scapular dyskinesis, in order to accurately assess the interventional effects.[26]

CONCLUSION

Most conservative treatment methods used in physiotherapy are effective in treating rotator cuff-related disorders, but the quality of research evidence was only low to moderate. It was mainly due to the limited number of studies, the relatively low methodological quality of studies, and the heterogeneity of study samples. Nevertheless, available literature suggests that exercise rehabilitation combined with other modalities such as TENS, shockwave therapy or manual therapy has the most evidence support. Further research is required to investigate the therapeutic effects of different conservative treatments in managing rotator cuff-related disorders.

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Baseball - Pushing the Limits and Beyond

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Have you ever watched a baseball game? Do you have questions in mind like many others?

Why are some players using gloves and some using bats? Why are players standing still and appear not engaged most of the time? The ball is hit hard, but why is there no score? What is a home run? Is it similar to softball, or is it the same?

WHAT IS BASEBALL ABOUT?

The game of baseball is played between two teams of nine players, each team alternating between the offensive (batting) and defensive (fielding) roles. The goal of the team is to score more runs (points) than the opposing team. To score a run, a batter hits the baseball, then runs across the three bases and back to home. The defensive team tries to avoid the other team's runs by getting their opponents 'out' (e.g. force out, fly out and tag out). Once they obtain three outs, the half-inning is over, and it is the defensive team's chance to bat and score runs. The entire inning is over when both teams have batted; a total of nine innings are played in a professional game.

The game of baseball is complex (scan the QR code for an animated explanation), making the game difficult to interpret to many but fascinating to others.

Fig. 1. QR code for a four-minute animated explanation of the game of baseball (Reference: Metalhead Software, 13 Aug 2015, Rules of Baseball in 4 Minutes)

The essence of the game is often the battle between the pitcher and the batter. To start the game of baseball, the pitcher (i.e. the fielder in the front and centre of the diamond-shaped field) throws a baseball towards the batter. If the batter successfully hits that ball, he earns the right to run the bases and initiate his tough journey of scoring a run.

Hitting baseball is well known for being one of the hardest things in sports.

At the professional level, a thrown pitch can reach up to 100 miles per hour, allowing less than 400 milliseconds for the ball to reach the plate after its release. As there is a lag time for everyone's nervous system to process, the batter has less than 150 milliseconds to locate the ball, predict its location when it reaches the plate and react (to swing or not). A blink of an eye takes around the same time.

Further, the pitched ball seldom travels in a straight line. Each and every baseball has its cover sewed over its core with stitches; the slightly elevated stitches change the friction around the baseball when it travels in the air. With the correct velocity, spin and arm angle, the stitches create turbulence in the surrounding air. The baseball literally changes direction in mid-air due to the Magnus effect.

Pitchers usually have more than one type of breaking (direction-changing) balls in their arsenal; that is, they can throw pitches of different velocities, and they have different changes of directions. It might be a horizontal change, a vertical change or a mixture of both. Used appropriately, a sequence of different pitches targeting different corners of the strike zone will keep the batters off balance and disrupt their rhythm. The battle between the pitcher and batter is mental chess more than a competition of athleticism.

It is extremely hard to hit a baseball. A competent player must equip himself with a decisive intellectual mind and an agile, coordinated physique. Even the elites fail more often than they succeed. When given a chance to hit a baseball, the best baseball players only have a success average of 30%. Thus baseball is also known as the game of failure, or a game built around coping with failure.

In the game of baseball, players are required to make decisions within a very limited time; failures and frustrations are inevitable. The beauty is that, due to the complexity of the game, failure (or multiple repeated failures) does not keep you from winning the game. For instance, you may misjudge twenty consecutive pitches resulting in three consecutive strikeouts, but if you are able to move forward and execute one precise swing, you may hit a home run and win the game for the team.

The failure rate is very high in baseball, as it is always similarly high among your opponents; embracing the

adjustment is the key to winning. The team which manages to analyse the cause of failure under pressure, and to cope and work through failure usually ends up on top. Unlike many other team sports, baseball game has no time limitation, any adjustments in adversity prior to the last 'out' might be the game changer. No matter how improbable, any comeback is theoretically possible, as stated by one of the best catchers Yogi Berra's, 'It ain't over till it's over'.

Fig. 2. Hong Kong Women's Baseball Team after a win against Cuba in Women's Baseball World Cup 2018. Hong Kong was down two runs in the last inning with two outs; they scored five runs for a comeback win. 'It ain't over till it's over'. (Personal collection)

Personally, I have found the complexity of baseball enchanting. Physically it demands agility, mobility, strength and endurance; intellectually, it involves statistical analysis and decisiveness; psychologically, it demands resilience.

Many lessons learnt in baseball can be translated into my career as an orthopaedic surgeon. The training of a focused mind under stress can be utilised in any operation; perseverance in team sports also makes me a more resilient team member while I am at the table. On the other hand, my orthopaedic knowledge better equips me as an athlete, especially in strength, conditioning and injury prevention.

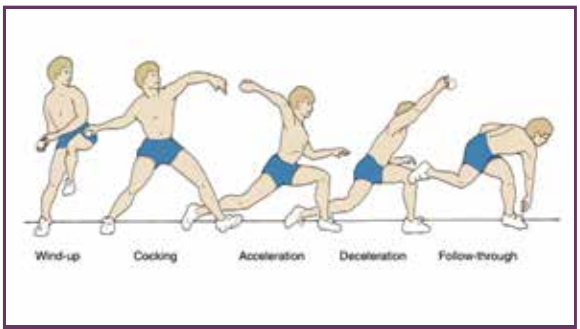

Fig. 3. The five phases of throwing a baseball. (Excerpted from Miller MD, Cooper DE, Warner JJP: Review of sports medicine and arthroscopy, Philadelphia, 1995, WB Saunders, p 123)

I hope you too would find a sport that brings sparkles to your life, and in case it is yet to be found, baseball would be a good start.

Fig. 4. Hong Kong Women's Baseball Team in 2018. Hong Kong is currently ranked No. 11th in the Women's Baseball World Rankings. (Personal collection)

Q and A's

Q: Why are some players using gloves and some using bats?
A: A player uses a glove during defence and a bat during the offense. A player uses both, but not simultaneously.

Q: Why are players standing still and appear not engaged most of the time?
A: It is a game of mental chess; players need time to analyse the situation and plan the subsequent move(s).

Q: The ball is hit hard, but why is there no score?
A: A player must touch all bases sequentially before scoring a run. One hard-hit ball rarely allows adequate time for such unless it is a home run.

Q. What is a home run?
A: A home run is a hit ball that allows the batter to circle the bases and reach home to score a run, usually by hitting the ball out of the field.

Q: Is it similar to softball, or is it the same?
A: Baseball and softball have a lot in common; most of the rules are similar. The obvious differences are the ball, size of the field and pitching style (Table 1).

Table 1. A comparison of baseball vis-a-vis softball (Developed by author)

	Baseball	Softball
Ball	Smaller and lighter - 9 inches (5 ounces) - White with red stitches	Larger and heavier - 12 inches (7 ounces) - Yellow with red stitches
Field size	Larger - Pitching distance 60 feet 6 inches - Baseline 90 feet	Smaller - Pitching distance 43 feet - Baseline 60 feet
Pitching	Overhand throw on pitching mound	Underhand throw on level ground

Despite the similarities in rules, baseball and softball are very different from each other and they demand different skill set and tactics. A baseball is smaller and lighter; it travels faster and is more influenced by friction in the air. Softball is pitched underhand, and breaking balls non-existence in baseball, like a rise ball (a ball thrown with a backspin that travels 'upwards' when it reaches the batter) is possible. A softball field is smaller, making it 'easier' to advance to the next base, but at the same time allowing less margin for defensive errors. Baseball and softball are both unique and fascinating in their own ways.

Sunday	Monday	Tuesday	Wednesday	Thursday	Friday	Saturday
1	2	★In-person/Zoom HKMA-HKSH CME Programme 2022-2023 (Physical Lecture + Online) Topic: Hypertension - From Drug To Device ★Certificate Course on Mental Health 2022 (Video Lectures) 3	★Zoom From Date to Practice - Managing Heart Failure Patients in Hong Kong - Online 4	★Certificate Course on Common Diseases in Otorhinolaryngology, Head & Neck Surgery 2023 (Video Lectures) 5	★Zoom Management of Nasal Polyps and the Latest Advance - Online 6	7
8	9	10	★Zoom SGLT2 Inhibition - From Antidiabetic Treatment to Chronic Kidney Disease Management - Online 11	★Zoom Timely Diabetes Management with Insulin ★Certificate Course on Common Diseases in Otorhinolaryngology, Head & Neck Surgery 2023 (Video Lectures) ★FMSHK Executive Committee Meeting 12	13	14
15	16	★In-person / Zoom HKMA-GHK CME Programme 2023 - Helicobacter infection in Hong Kong 17	18	★Certificate Course on Common Diseases in Otorhinolaryngology, Head & Neck Surgery 2023 (Video Lectures) 19	20	21
22	23	24	25	★Certificate Course on Common Diseases in Otorhinolaryngology, Head & Neck Surgery 2023 (Video Lectures) 26	27	28
29	★Zoom Overactive bladder? Time to look outside the bladder for causes and treatment - Online 30	★Zoom Pancreatic Exocrine Insufficiency (PEI) in Patients with Diabetic Mellitus - Online 31				

Date / Time	Function	Enquiry / Remarks
3 TUE 2:00 PM	**In-person/Zoom** **HKMA-HKSH CME Programme 2022-2023 (Physical Lecture + Online)** **Topic: Hypertension - From Drug To Device** Organiser: Hong Kong Medical Association and Hong Kong Sanatorium & Hospital Speaker: Dr Anthony Yiu-tung WONG Venue: HKMA Dr. Li Shu Pui Professional Education Centre, 2/F, Chinese Club Building, 21-22 Connaught Road, Central, Hong Kong	HKMA CME Dept. Tel: 3108 2507 1 CME Point
7:00 PM	**Certificate Course on Mental Health 2022 (Video Lectures)** Organiser: The Federation of Medical Societies of Hong Kong Speaker: Dr William WH CHUI	Ms Vienna LAM Tel: 2527 8898
4 WED 2:00 PM	**Zoom** **From Date to Practice - Managing Heart Failure Patients in Hong Kong - Online** Organiser: HKMA-Central, Western & Southern Community Network Speaker: Dr LEUNG Sai-chau	Ms. Candice Tong Tel: 3108 2513 1 CME Point
5 THU 7:00 PM	**Certificate Course on Common Diseases in Otorhinolaryngology, Head & Neck Surgery 2023 (Video Lectures)** Organiser: The Federation of Medical Societies of Hong Kong Speaker: Dr Alcie KY SIU	Ms Vienna LAM Tel: 2527 8898
6 FRI 2:00 PM	**Zoom** **Management of Nasal Polyps and the Latest Advance - Online** Organiser: HKMA-KLN City Community Network Speaker: Dr Henry Chuen-kwong LAM	Ms. Candice Tong 3108 2513 1 CME Point
11 WED 2:00 PM	**Zoom** **SGLT2 Inhibition - From Antidiabetic Treatment to Chronic Kidney Disease Management - Online** Organiser: HKMA-Shatin Community Network Speaker: Dr LAM Chung-man	Ms. Candice Tong Tel: 3108 2513 1 CME Point
12 THU 2:00 PM	**Zoom** **Timely Diabetes Management with Insulin** Organiser: HKMA-Kowloon East Community Network Speaker: Dr Joseph See-yui LAM	Ms. Candice Tong Tel: 3108 2513 1 CME Point
7:00 PM	**Certificate Course on Common Diseases in Otorhinolaryngology, Head & Neck Surgery 2023 (Video Lectures)** Organiser: The Federation of Medical Societies of Hong Kong Speaker: Dr Eddy WONG	Ms Vienna LAM Tel: 2527 8898
8:00 PM	**FMSHK Executive Committee Meeting** Organiser: The Federation of Medical Societies of Hong Kong; Venue: Council Chamber, 4/F, Duke of Windor Social Service Building, 15 Hennessy Road, Wanchai, Hong Kong	Ms Nancy CHAN Tel: 2527 8898
17 TUE 2:00 PM	**In-person / Zoom** **HKMA-GHK CME Programme 2023 - Helicobacter infection in Hong Kong** Organiser: Hong Kong Medical Association and Gleneagles Hong Kong Hospital Speaker: Dr LEE Ting-lam Venue: HKMA Dr. Li Shu Pui Professional Education Centre, 2/F, Chinese Club Building, 21-22 Connaught Road Central, Hong Kong	HKMA CME Dept Tel: 3108 2507 1 CME Point
19 THU 7:00 PM	**Certificate Course on Common Diseases in Otorhinolaryngology, Head & Neck Surgery 2023 (Video Lectures)** Organiser: The Federation of Medical Societies of Hong Kong Speaker: Dr Fergus WONG	Ms Vienna LAM Tel: 2527 8898
26 THU 7:00 PM	**Certificate Course on Common Diseases in Otorhinolaryngology, Head & Neck Surgery 2023 (Video Lectures)** Organiser: The Federation of Medical Societies of Hong Kong Speaker: Dr CHANG Wai-tsz	Ms Vienna LAM Tel: 2527 8898
30 MON 2:00 PM	**Zoom** **Overactive bladder? Time to look outside the bladder for causes and treatment - Online** Organiser: Hong Kong Medical Association Speaker: Dr Raymond Wai-man KAN	HKMA CME Dept. Tel: 3108 2507 1 CME Point
31 TUE 2:00 PM	**Zoom** **Pancreatic Exocrine Insufficiency (PEI) in Patients with Diabetic Mellitus - Online** Organiser: Hong Kong Medical Association Speaker: Dr CHEUNG Sai-wah	HKMA CME Dept. Tel: 3108 2507 1 CME Point

Answers to Dermatology Quiz

Answers:

1. The diagnosis is dyshidrotic eczema (pompholyx), and the possible differential diagnoses include fungal infection, palmoplantar pustular psoriasis, bullous impetigo, allergic or irritant contact dermatitis, subcorneal pustular dermatosis, erythema multiforme and immunobullous diseases such as bullous pemphigoid or linear Ig-A diseases.

2. Dyshidrotic eczema is diagnosed by its characteristic clinical features. No investigation is needed to confirm it. Sometimes fungal smear or culture may be necessary to rule out dermatophytes infection, and a patch test is needed for suspected allergic contact dermatitis. Skin biopsy is reserved for difficult cases rarely.

3. Treatment of dyshidrotic eczema is sometimes difficult. Typical first-line treatment includes potent and even ultra-potent topical corticosteroids. Short course systemic prednisolone is often necessary, especially for acute flares. Other immunosuppressants may be tried in recalcitrant patients. Some case reports suggest calcineurin inhibitors may be helpful. Phototherapy of Soak PUVA (topical application of psoralen and UVA) or Narrow-band UVB has demonstrated beneficial effects on dyshidrotic eczema.

Dr KWAN Chi-keung
MBBS(HK), MRCP (UK), FRCP(Lond, Glasg, Edin), Dip Derm(Glasg), PDipID(HK), FHKCP, FHKAM(Medicine)
Specialist in Dermatology and Venereology

TAKE ON THE CHALLENGES OF COVID-19[1]

TEST. TREAT. TAKE CHARGE. molnupiravir

You can NOW PURCHASE Molnupiravir from us!

For more information,
please contact MSD Professional sales representatives.

Reference: 1. molnupiravir US EUA Product Insert.

MOLNUPIRAVIR Selected Safety Information

Authorized Use

1. Molnupiravir is authorized for use under an Emergency Use Authorization (EUA) for the treatment of mild-to-moderate coronavirus disease 2019 (COVID-19) in adults:
   - with positive results of direct SARS-CoV-2 viral testing, and
   - who are at high risk for progression to severe COVID-19, including hospitalization or death, and
   - for whom alternative COVID-19 treatment options approved or authorized by FDA are not accessible or clinically appropriate
2. Molnupiravir is not approved for any use, including the treatment of COVID-19, but is authorized for emergency use by the FDA under an Emergency Use Authorization (EUA).
3. The emergency use of molnupiravir is only authorized for the duration of the declaration that circumstances exist justifying the authorization of the emergency use of drugs and biological products during the COVID-19 pandemic under Section 564(b)(1) of the Federal Food, Drug, and Cosmetic Act, 21 U.S.C. § 360bbb-3(b)(1) unless the declaration is terminated or authorization revoked sooner.

Limitations of Authorized Use

4. Molnupiravir is not authorized:
   - for use in patients who are less than 18 years of age
   - for initiation of treatment in patients hospitalized due to COVID-19. Benefit of treatment with molnupiravir has not been observed in subjects when treatment was initiated after hospitalization due to COVID-19
   - for use for longer than 5 consecutive days
   - or pre-exposure or post-exposure prophylaxis for prevention of COVID-19
5. Molnupiravir may only be prescribed for an individual patient by physicians, advanced practice registered nurses, and physician assistants that are licensed or authorized under state law to prescribe drugs in the therapeutic class to which molnupiravir belongs (i.e., anti-infectives).

Contraindications

6. No contraindications have been identified based on the limited available data on the emergency use of molnupiravir authorized under this EUA.

Warnings and Precautions

7. There are limited clinical data available for molnupiravir. Serious and unexpected adverse events may occur that have not been previously reported with molnupiravir use.
8. Molnupiravir is not recommended for use during pregnancy. Based on findings from animal reproduction studies, molnupiravir may cause fetal harm when administered to pregnant individuals. There are no available human data on the use of molnupiravir in pregnant individuals to evaluate the risk of major birth defects, miscarriage or adverse maternal or fetal outcomes.
9. Molnupiravir is authorized to be prescribed to a pregnant individual only after the healthcare provider has determined that the benefits would outweigh the risks for that individual patient. If the decision is made to use molnupiravir during pregnancy, the prescribing healthcare provider must document that the known and potential benefits and the potential risks of using molnupiravir during pregnancy were communicated to the pregnant individual.
10. Advise individuals of childbearing potential of the potential risk to a fetus and to use an effective method of contraception correctly and consistently during treatment with molnupiravir and for 4 days after the final dose.
11. Prior to initiating treatment with molnupiravir, assess whether an individual of childbearing potential is pregnant or not, if clinically indicated.
12. Hypersensitivity reactions, including anaphylaxis, have been reported with molnupiravir. If signs and symptoms of a clinically significant hypersensitivity reaction or anaphylaxis occur, immediately discontinue molnupiravir and initiate appropriate medications and/or supportive care.
13. Molnupiravir is not authorized for use in patients less than 18 years of age because it may affect bone and cartilage growth. The safety and efficacy of molnupiravir have not been established in pediatric patients.

Adverse Reactions

14. The most common adverse reactions occurring in ≥1% of subjects in the molnupiravir treatment group in the Phase 3 double-blind MOVe-OUT study were diarrhea (2% versus placebo at 2%),nausea (1% versus placebo at 1%), and dizziness (1% versus placebo at 1%) all of which were Grade 1 (mild) or Grade 2 (moderate).
    Serious adverse events occurred in 7% of subjects receiving molnupiravir and 10% receiving placebo; most serious adverse events were COVID-19 related. Adverse events leading to death occurred in 2 (<1%) of the subjects receiving molnupiravir and 12 (2%) of subjects receiving placebo.

Drug Interactions

15. No drug interactions have been identified based on the limited available data on the emergency use of molnupiravir. No clinical drug-drug interaction trials of molnupiravir with concomitant medications, including other treatments for mild to moderate COVID-19, have been conducted.

Breastfeeding

16. There are no data on the presence of molnupiravir or its metabolites in human milk. It is unknown whether molnupiravir has an effect on the breastfed infant or effects on milk production. Based on the potential for adverse reactions in the infant from molnupiravir, breastfeeding is not recommended during treatment with molnupiravir and for 4 days after the final dose. A lactating individual may consider interrupting breastfeeding and may consider pumping and discarding breast milk during treatment and for 4 days after the last dose of molnupiravir.

Males of Reproductive Potential

17. Nonclinical studies to fully assess the potential for molnupiravir to affect offspring of treated males have not been completed. Advise sexually active individuals with partners of childbearing potential to use a reliable method of contraception correctly and consistently during treatment and for at least 3 months after the last dose of molnupiravir. The risk beyond three months after the last dose of molnupiravir is unknown.

Before prescribing, please consult the full prescribing information.

MSD

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HK-ANV-00809 May/2022

Kyprolis® (carfilzomib) for Injection

WHEN MULTIPLE MYELOMA RELAPSES,

LOOK TO KYPROLIS® FOR A WAY FORWARD

Around 4 in 10 patients with MM who start second-line therapy never start a third[1]
Your choice of treatment at first relapse matters.

KdD

KdD as dual-target approach[2]

mPFS 28.6 months*
vs. 15.2 months with Kd

mOS data not mature
At a median follow up of 27.8 months

* HR=0.59; 95% CI: 0.45-0.78; $p<0.0001$

KRd

KRd as triplet therapy[3-5]

mPFS 26.3 months†
vs. 17.6 months with Rd

mOS 48.3 months‡
vs. 40.4 months with Rd

† HR=0.69; 95% CI: 0.57-0.83; $p=0.0001$
‡ HR=0.79; 95% CI: 0.67-0.95; $p=0.0091$

Kd

Kd as doublet therapy[6]

mPFS 18.7 months§
vs. 9.4 months with Vd

mOS 47.6 Months‖
vs. 40.0 months with Vd

§ HR=0.53; 95% CI: 0.44-0.65; $p<0.0001$
‖ HR=0.79; 95% CI: 0.648-0.964; $p=0.01$

KYPROLIS® + dexamethasone (Kd) and KYPROLIS® + dexamethasone + daratumumab (KdD) regimens are available in ONCE-WEEKLY and TWICE-WEEKLY dosing[5]*

*Once-weekly KRd has not been approved.

INDICATION:
KYPROLIS® is indicated for the treatment of adult patients with relapsed or refractory multiple myeloma who have received one to three lines of therapy in combination with lenalidomide and dexamethasone; or dexamethasone; or daratumumab and dexamethasone.[3]

Abbreviations: CI: Confidence interval; HR: Hazard ratio; Kd: Carfilzomib and dexamethasone; KdD: Carfilzomib, dexamethasone and daratumumab; KRd: Carfilzomib, lenalidomide, and dexamethasone MM: Multiple myeloma; mOS: Median overall survival; mPFS: Median progression-free survival; Rd: lenalidomide and dexamethasone; Vd: bortezomib and dexamethasone

References: 1. Yong K, et al. Multiple myeloma: patient outcomes in real-world practice. *Br J Haematol*. 2016; 175 (2): 252-264. **2.** Usmani SZ, et al. Carfilzomib, dexamethasone, and daratumumab versus carfilzomib and dexamethasone for patients with relapsed or refractory multiple myeloma (CANDOR): updated outcomes from a randomised, multicentre, open-label, phase 3 study. *Lancet Oncol* 2022;23:65–76. **3.** Moreau P, et al. Impact of prior treatment on patients with relapsed multiple myeloma treated with carfilzomib and dexamethasone vs bortezomib and dexamethasone in the phase 3 ENDEAVOR study. *Leukemia*. 2017;31:115–122. **4.** Siegel DS, et al. Improvement in Overall Survival With Carfilzomib, Lenalidomide, and Dexamethasone in Patients With Relapsed or Refractory Multiple Myeloma. *J Clin Oncol*. 2018;36:728-734. **5.** KYPROLIS® (carfilzomib) Hong Kong Prescribing Information. June 2021. **6.** Dimopoulos MA, et al. Carfilzomib or bortezomib in relapsed or refractory multiple myeloma (ENDEAVOR): an interim overall survival analysis of an open-label, randomised, phase 3 trial. *Lancet Oncol*. 2017;18(10):1327-1337.

Kyprolis® (Carfilzomib) Abbreviated Prescribing Information
Indications: Kyprolis is indicated for the treatment of adult patients with relapsed or refractory multiple myeloma who have received one to three lines of therapy in combination with lenalidomide and dexamethasone; or with dexamethasone; or with daratumumab and dexamethasone. **Dosage & Administration:** Administration Precautions: Dose based on BSA to max 2.2 m^2; dose adjust for weight changes >20%. See full PI for dosing & administration information. Ensure adequate hydration before Cycle 1, Day 1 with oral fluids (30 mL per kg) and prior to each dose in Cycle 1 with IV fluids (250-500 mL). Premedicate with the recommended dose of dexamethasone administered as part of the combination therapy; administer dexamethasone PO or IV 30 mins to 4 hrs before Kyprolis during Cycle 1. Administer Kyprolis after hemodialysis procedure for patients on hemodialysis. In combination with lenalidomide and dexamethasone: Administer Kyprolis as a 10min IV infusion on Days 1, 2, 8, 9, 15, and 16 of each 28-day cycle in combination with lenalidomide and dexamethasone until Cycle 12. The recommended starting dose of Kyprolis is 20 mg/m^2 on Cycle 1, Days 1 and 2. If tolerated, escalate the dose to 27 mg/m^2 on Cycle 1, Day 8. From Cycle 13, administer Kyprolis on Days 1, 2, 15 and 16. Continue treatment until disease progression or unacceptable toxicity occurs. Treatment for longer than 18 cycles should be based on an individual benefit/risk assessment. In combination with dexamethasone: Administer dexamethasone 30 mins to 4 hrs before Kyprolis. *Twice weekly:* Administer Kyprolis as a 30-min IV infusion on Days 1, 2, 8, 9, 15, and 16 of each 28-day cycle in combination with dexamethasone until disease progression or unacceptable toxicity. The recommended starting dose of Kyprolis is 20 mg/m^2 on Cycle 1, Days 1 and 2. If tolerated, escalate the dose to 56 mg/m^2 on Cycle 1, Day 8. *Once weekly:* Administer Kyprolis as a 30-min IV infusion on Days 1, 8, and 15 of each 28-day cycle in combination with dexamethasone until disease progression or unacceptable toxicity. The recommended starting dose of Kyprolis is 20 mg/m^2 on Cycle 1, Day 1. If tolerated, escalate the dose to 70 mg/m^2 on Cycle 1, Day 8. In combination with IV daratumumab and dexamethasone: Administer dexamethasone 30 mins to 4 hrs before Kyprolis and 1 to 3 hrs before IV daratumumab. *Twice weekly:* Administer Kyprolis as a 30-min IV infusion on Days 1, 2, 8, 9, 15 and 16 of each 28-day cycle in combination the IV daratumumab and dexamethasone until disease progression or unacceptable toxicity. The recommended starting dose of Kyprolis is 20 mg/m^2 on Cycle 1, Days 1 and 2. If tolerated, escalate the dose to 56 mg/m^2 on Cycle 1, Day 8 and thereafter. *Once weekly:* Administer Kyprolis as a 30-min IV infusion on Days 1, 8 and 15 of each 28-day cycle in combination with IV daratumumab and dexamethasone until disease progression or unacceptable toxicity. The recommended starting dose of Kyprolis is 20 mg/m^2 on Cycle 1, Day 1. If tolerated, escalate the dose to 70 mg/m^2 on Cycle 1, Day 8 and thereafter. **Contraindications:** None. **Warnings & Precautions:** Consider: Uric acid-lowering drugs in patients at risk for tumor lysis syndrome (TLS), prophylaxis with antivirals for patients who are hepatitis B virus (HBV) carriers. Monitor: Clinical signs or symptoms of cardiac failure or cardiac ischemia (cardiac toxicities), blood pressure (hypertension), blood loss (hemorrhage), thrombotic thrombocytopenic purpura/hemolytic uremic syndrome (TTP/HUS) (thrombotic microangiopathy), renal function with regular measurement of the serum creatinine and/or estimated clearance (acute renal failure), TLS, platelet counts (thrombocytopenia), liver enzymes (hepatic toxicity and failure), serum potassium levels. Discontinue Kyprolis: In the event of drug-induced pulmonary toxicity, if Posterior Reversible Encephalopathy Syndrome (PRES) is suspected or if Progressive Multifocal Leukoencephalopathy (PML) is suspected. Others: Evaluate promptly if cardiac toxicity is suspected. Withhold Kyprolis for Grade 3/4 cardiac adverse reactions until recovery. Withhold for pulmonary hypertension until resolved or returned to baseline. Stop Kyprolis for Grade 3/4 dyspnea until resolved or returned to baseline. Optimize blood pressure prior to starting Kyprolis; withhold Kyprolis and evaluate if hypertension cannot be adequately controlled. Provide thromboprophylaxis (venous thrombosis). Infusion-related reactions up to 24 hrs after administration of Kyprolis. HBV reactivation. Kyprolis in combination with melphalan and prednisone is not indicated for transplant-ineligible patients with newly diagnosed multiple myeloma. **Pregnancy:** Kyprolis can cause fetal harm when administered to a pregnant woman. Use effective contraception during & after treatment (females: 6 mo; males 3 mo); consider non-hormonal contraception during treatment when Kyprolis is administered in combination. Do not administer to breastfeeding women. **Adverse Reactions:** Common: Anemia, neutropenia, thrombocytopenia, diarrhea, constipation, nausea, vomiting; fatigue, pyrexia, edema peripheral, asthenia, upper respiratory tract infection, bronchitis, viral upper respiratory tract infection, respiratory tract infection, pneumonia, hypokalemia, hypocalcemia, hyperglycemia, muscle spasms, back pain, peripheral neuropathies, headache, insomnia; cough, dyspnea, rash, embolic and thrombotic events, hypertension. **Please read full prescribing information prior to administration (available upon request).** Kyprolis® is a registered trademark owned or licensed by Amgen Inc., its subsidiaries, or affiliates. Version: HKKYPPI04

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