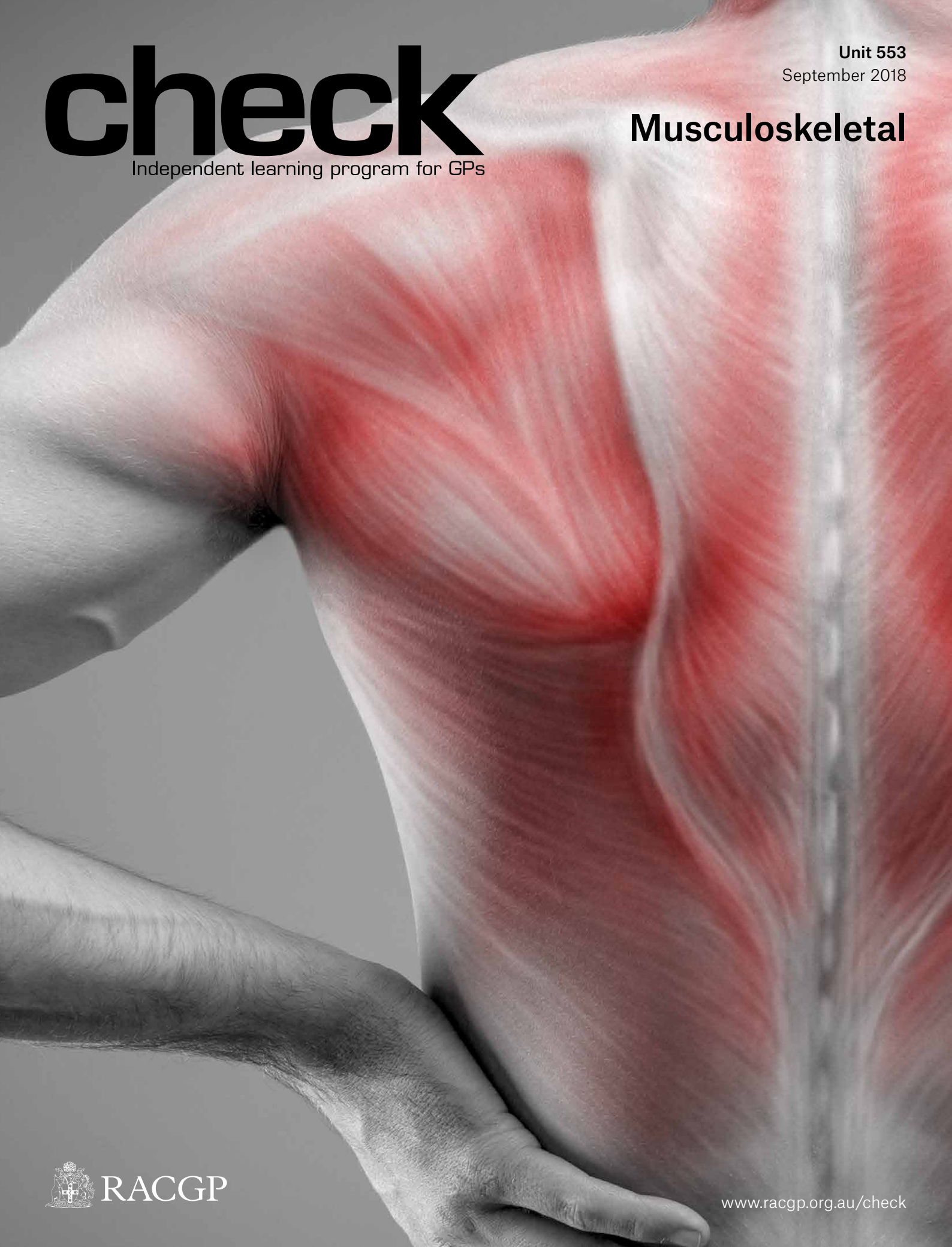
check

Independent learning program for GPs

Unit 553
September 2018

Musculoskeletal



RACGP

www.racgp.org.au/check

Disclaimer

The information set out in this publication is current at the date of first publication and is intended for use as a guide of a general nature only and may or may not be relevant to particular patients or circumstances. Nor is this publication exhaustive of the subject matter. Persons implementing any recommendations contained in this publication must exercise their own independent skill or judgement or seek appropriate professional advice relevant to their own particular circumstances when so doing. Compliance with any recommendations cannot of itself guarantee discharge of the duty of care owed to patients and others coming into contact with the health professional and the premises from which the health professional operates.

Whilst the text is directed to health professionals possessing appropriate qualifications and skills in ascertaining and discharging their professional (including legal) duties, it is not to be regarded as clinical advice and, in particular, is no substitute for a full examination and consideration of medical history in reaching a diagnosis and treatment based on accepted clinical practices.

Accordingly, The Royal Australian College of General Practitioners Ltd (RACGP) and its employees and agents shall have no liability (including without limitation liability by reason of negligence) to any users of the information contained in this publication for any loss or damage (consequential or otherwise), cost or expense incurred or arising by reason of any person using or relying on the information contained in this publication and whether caused by reason of any error, negligent act, omission or misrepresentation in the information.

Subscriptions

For subscriptions and enquiries please call 1800 331 626 or email check@racgp.org.au

Published by

The Royal Australian College of General Practitioners Ltd
100 Wellington Parade
East Melbourne, Victoria 3002, Australia

Telephone 03 8699 0414
Facsimile 03 8699 0400
www.racgp.org.au

ABN 34 000 223 807
ISSN 0812-9630

We acknowledge the Traditional Custodians of the lands and seas on which we work and live, and pay our respects to Elders, past, present and future.

check
Independent learning program for GPs

Musculoskeletal

Unit 553 September 2018

The five domains of general practice

- Communication skills and the patient–doctor relationship
- Applied professional knowledge and skills
- Population health and the context of general practice
- Professional and ethical role
- Organisational and legal dimensions

RACGP

About this activity

Almost one in five (18.4 per 100) general practice encounters relate to musculoskeletal problems,[1] and almost a quarter of these occur as a result of injury.[2] Musculoskeletal problems account for 56.9% of work-related issues.[1] Osteoarthritis, the most common form of arthritis, accounted for 3.9% of general practice consultations and 26.9% of orthopaedic surgeon referrals in 2013–14.[1] Approximately 2.1 million Australians have osteoarthritis; this includes 21% of people over the age of 45 years and 35% of people over the age of 80 years.[3] Osteoporosis affected 3.3% of Australians in 2011–12, and just over half (53.2%) of those patients had visited a general practitioner (GP) for their condition in the past 12 months.[4] Heel pain is a common presentation in general practice; the prevalence of heel pain is estimated to be 3.6% in the general population.[5] In patients aged 40–60 years, a common cause of heel pain is plantar fasciitis.[6] A recent study found that approximately 15% of Australian adults live with chronic pain. Of this group, 12% of males and 13.4% of females were prescribed opioid analgesics.[7] Past expert-based guidelines have encouraged GPs to initially and regularly assess the risk of addiction when providing opioid analgesics.[8] However, this more effectively addresses prescriber barriers than it prevents the range of opioid-related harms.

References

1. Britt H, Miller GC, Henderson J, et al. General practice activity in Australia 2013–14. General practice series no. 36. Sydney: Sydney University Press, 2014.
2. Pollack AJ, Bayram C, Miller GC. Musculoskeletal injury in Australian general practice: 2000 to 2015. Aus Fam Physician 2016;45(7):462–65.
3. Australian Institute of Health and Welfare. Osteoarthritis snapshot. Canberra: AIHW, 2018. Available at www.aihw.gov.au/reports/chronic-musculoskeletal-conditions/osteoarthritis/formats [Accessed 7 August 2018].
4. Australian Bureau of Statistics. Australian Health Survey: Health Service Usage and Health Related Actions, 2011-2012. ABS cat no. 4364.0.55.002. Belconnen, ACT: ABS, 2013.
5. Pollack AJ, Britt H. Plantar fasciitis in Australian general practice. Aus Fam Physician 2005;44(3):90–91.
6. BMJ Best practice. Plantar fasciitis. London: BMJ Publishing Group. Available at https://bestpractice.bmj.com/topics/en-us/487 [Accessed 23 July 2017].
7. Miller A, Sanderson K, Bruno R, Breslin M, Neil AL. The prevalence of pain and analgesia use in the Australian population: Findings from the 2011 to 2012 Australian National Health Survey. PDS 2017;26(11):1403–10.
8. The Royal Australasian College of Physicians. Prescription opioid policy: Improving management of chronic non-malignant pain and prevention of problems associated with prescription opioid use. Sydney, RACP: 2009.

Learning outcomes

At the end of this activity, participants will be able to:

- describe the approach to managing osteoarthritis
- summarise the assessment and management of osteoporosis
- identify signs and symptoms of plantar fasciitis
- discuss issues around prescription of opioids for management of chronic non-cancer pain

Authors

Kathryn Gibson (Case 2) BA, BMBCh, FRACP, PhD is a senior staff specialist in Rheumatology at Liverpool Hospital Sydney. She has a special interest in patient-reported outcome measures in rheumatic diseases and in clinical informatics. She is a member of the Musculoskeletal Clinical Academic Group of the Sydney Partnership for Health, Education, Research and Enterprise.

Geraldine Hassett (Case 3) MBBS, FRACP, MMed (Clin Epi), PhD is a full-time consultant rheumatologist and director of the Rheumatology Department at Liverpool Hospital, South Western Sydney Local Health District, and conjoint senior lecturer, University of New South Wales. She is focused on ensuring that the burden of musculoskeletal disease in the community is reduced by promoting best evidence patient-centred care through translation of evidence into practice. She is the leader of the Musculoskeletal Clinical Academic Group of the Sydney Partnership for Health, Education, Research and Enterprise.

Simon Holliday (Case 4) BMed, FAChAM, FRACGP, FACRRM, DA, DipRACOG, GradDipA&DSt, GCertClinEpid is a general practitioner and Addiction Physician at Albert St Medical Centre, Taree, NSW.

Kenneth de Jong (Case 1) MBBS (Hons), GradDipSurgAnat is a hospital medical officer with Eastern Health, Victoria. His interests include orthopaedics, anatomy education and medical education.

Rachel de Jong (Case 1) BBioMedSci is a medical student at Monash University.

Narainraj Kamalaraj (Case 3) MBBS (Hons) is a rheumatology advanced trainee at Concord Repatriation General Hospital and Conjoint Associate Lecturer at Western Sydney University. He is currently in his second year of subspecialty training, having completed previous appointments at Campbelltown and Liverpool Hospitals. He has published research in a number of areas including osteoporosis prevalence, management of acute gout in hospital and lupus. He is currently on scholarship, undertaking a Masters of Internal Medicine through the University of Edinburgh. His current interests relate to interventional cervical spine care, particularly in osteoarthritis.

Ayano Kelly (Case 3) MBBS, BSc (Med), BA, FRACP is a rheumatologist and a PhD candidate at the Australian National University. She has a research interest in medication adherence and osteoporosis. She is a member of the Musculoskeletal Clinical Academic Group of the Sydney Partnership for Health, Education, Research and Enterprise.

Deepa Mahananda (Case 3) MBBS, DCH is a part-time general practice registrar in Inner West Sydney, and Casual Academic Tutor in the Department of General Practice, Western Sydney University. She has a particular interest in reproductive health, child health and preventive health. Additionally, she is passionate about using low-carbohydrate nutrition for both optimal health and chronic disease management.

Justine Naylor (Case 2) PhD, BAppSc (Phty) is the Senior Principal Research Fellow in Orthopaedics and Director of the Whitlam Orthopaedic Research Centre, South West Sydney Local Health District. She has research expertise in arthroplasty for osteoarthritis including rehabilitation, acute-care interventions and unwarranted practice variation. She is a member of the Musculoskeletal Clinical Academic Group of the Sydney Partnership for Health, Education, Research and Enterprise.

Siobhan Schabrun (Case 2) PhD, BPhysio (Hons) is a National Health and Medical Research Council Career Development Fellow and Senior Research Scientist at Neuroscience Research Australia. She has a special interest in the role of neuroplasticity in chronic musculoskeletal pain conditions.

Fabian Schwarz (Case 1) BSc, MBBS, MPH, DCH, FRACGP, FARGP, CCFP is a rural generalist working for the Department of Health in the Northern Territory. Dr Schwarz also has a special interest in people and their stories, and bringing personal aspects into contemporary medical education – going beyond mere medical facts.

Lawrence Tan (Cases 2 and 3) MBBS, DCH, DRCOG, MPH, FRACGP is a part-time general practitioner in Southwest Sydney, and senior lecturer in the Department of General Practice, Western Sydney University. He enjoys teaching medical students and registrars. Dr Tan's research interests are in cultural and linguistic diversity and how this affects the delivery of primary health care. He is a member of the Musculoskeletal Clinical Academic Group of the Sydney Partnership for Health, Education, Research and Enterprise (SPHERE).

Vivek Thakkar (Case 3) BSc, MBBS, DMedSc, FRACP is an associate professor, Faculty of Medicine and Health Sciences, Macquarie University, staff specialist rheumatologist at Liverpool Hospital, and is active in clinical practice in South West Sydney. Dr Thakkar is particularly interested in medical education and cardiopulmonary complications of autoimmune connective tissues diseases.

Peer reviewers

Diana Chessman MBBS, BMedSci, PhD, FRACP is a staff specialist in rheumatology at Liverpool Hospital and works privately in Chatswood NSW. She has a PhD in immunology and has a special interest in autoimmune inflammatory arthritides. She is the coordinator for the Australian Rheumatology Association (ARA) NSW Advanced Trainee Teaching Program and is a member of the ARA NSW council. She is also a member of the ARA National Education, Training and Workforce Committee. She is a conjoint senior lecturer of the University of New South Wales.

Greg Harris MBBS (Hons), FACSEP is a sport and exercise medicine physician at Ballarat Orthopaedics and Sports Medicine. Dr Harris was the deputy medical director for the 2018 Australian Olympic Winter Games team, is the Chief Medical Officer for the Australian National Motocross and Supercross Championships and the director of the Elite Motorsport Medical Clinic. He also consults at Absolute Health and Performance in Melbourne.

Leesa Huguenin MBBS, MSports Med, FACSEP is the founder and director of MP Sports Physicians, a specialist sports medicine practice in Victoria with clinics in Mornington, Frankston and Berwick. She is actively involved in registrar teaching, providing several supervised training positions each year. She currently provides medical advice to the Frankston Dolphins VFL and St Kilda Saints VFLW teams.

Abbreviations

BMD	bone mineral density
BMI	body mass index
CBT-i	cognitive behavioural therapy for insomnia
COX	cyclooxygenase
CT	computed tomography
DXA	dual energy X-ray absorptiometry
GP	general practitioner
LtOT	long-term opioid therapy
MBS	Medicare Benefits Schedule
MSC	mesenchymal stem cells
MRI	magnetic resonance imaging
NSAIDs	non-steroidal anti-inflammatory drugs
OACCP	Osteoarthritis Clinical Care Pathway
OARSI	Osteoarthritis Research Society International
ONJ	osteonecrosis of the jaw
ORP	osteoporosis re-fracture prevention
PEG	pain, enjoyment, general activity
PRP	platelet rich plasma
PTH	parathyroid hormone
RCT	randomised controlled trial
RACGP	Royal Australian College of General Practitioners
SNRI	serotonin–noradrenaline reuptake inhibitor
WHO	World Health Organization

CASE

1 George has a sore heel

George is a store assistant aged 45 years who presents to you with a painful left heel. After struggling with his weight for some time, he decided to take up running. On completion of his first five-kilometre run, however, he felt some discomfort in his left heel, which was self-limiting at first, but now seems to be constant. He tried some over-the-counter medications (paracetamol and ibuprofen), with moderate effect. George is otherwise feeling well and does not have any medical conditions of note. His weight is 90 kg and he is 180 cm tall. George is wondering if this is plantar fasciitis.

Question 1

How will you answer George?

Question 2

What is your approach to diagnosing George's heel pain? What are the differential diagnoses?

Further information

George points to his plantar fascia region and describes pain when walking or running.

Question 3

Considering your differential diagnoses, what else would you like to know from George?

Question 4

What objective tests would you do to further clarify your differential diagnosis?

Further information

Your examination of George reveals no skin or muscle wasting. On gait assessment, you notice a slightly antalgic gait. George is able to complete calf raises. Tinel's sign is negative.

Question 5

What investigations, if any, would be indicated for George?

Question 6

What are important diagnoses that should not be missed? What other symptoms would these conditions present with?

Question 7

What would be an initial management plan for George? Is there a role for surgical referral?

CASE 1 Answers

Answer 1

Heel pain can be the result of various aetiologies. One of the most common causes is plantar fasciitis, which affects sedentary and active people, mostly between the ages of 40 and 60 years.[1] It is thought to be caused by mechanical overload from lifestyle or exercise and often affects individuals who are overweight or obese.[1] More specifically, non-resolving inflammation and degeneration of the plantar fascia under the foot is often found to accompany the characteristic heel pain. It has been suggested, however, that the cause of pain is more likely to be degeneration rather than inflammation.[2] Other risk factors include poor biomechanics, prolonged barefoot walking and weight-bearing, and abnormal foot arches.[2] Nevertheless, the exact causes and mechanisms underlying the condition are poorly understood.

Answer 2

A practical approach to heel pain, based on anatomical structure, has been previously described (Table 1, Figure 1).[3]

A thorough history and examination are integral in the diagnosis of plantar fasciitis. Classically, a history delineates symptoms of severe heel pain in the morning or after rest, which subsides with movement but is further aggravated following extended periods of weight-bearing. On physical examination, common findings are tenderness over the medial calcaneal tubercle along with pain on dorsiflexion of the first toe. [2]

Table 1. Possible structures and sites of pain

Site/type of pain	Common sources of pain	Less common sources of pain
Posterior	• Achilles tendon insertion • Superficial calcaneal bursa • Posterior impingement of soft-tissues/os trigonum in active people • Calcaneal apophysis in adolescents	• Sural nerve
Inferior	• Plantar fascia • Calcaneal fat pad	• Medial or lateral calcaneal nerve, especially as they split from the tibial branch
Medial	• Tibialis posterior tendon and sheath • Tibialis posterior insertion and apophysis in adolescents	• FHL and sheath • Abductor hallucis • Deltoid and spring ligaments • Posterior tibial nerve in tarsal tunnel (is associated with neural symptoms such as tingling) • Bone: medial malleolus
Lateral	• Lateral ligaments of the ankle • Sinus tarsi	• Peroneal tendinopathy or tenosynovitis associated with subluxation • Cubometatarsal joint • Peroneus brevis insertion/apophysis of base of 5th metatarsal in adolescents or after ankle sprain
Deep, vague pain	• Subtalar joint	• Bone pain: calcaneus, talus, navicular

FHL, flexor hallucis longus

Reproduced with permission from The Royal Australian College of General Practitioners, from Rio E, Mayes S, Cook J. Heel pain: A practical approach. Aust Fam Physician 2015;44(3):96–101.

Differential diagnoses include:[1]

- calcaneal contusion
- medial calcaneal nerve entrapment
- calcaneal stress fracture
- rupture of plantar fascia
- inferior calcaneal bursitis
- abductor hallucis tendonitis
- tarsal tunnel syndrome
- inflammatory arthropathy (eg ankylosing spondylitis, Reiter's disease)
- sacroiliac nerve entrapment (L5 or S1 radiculopathy)
- rheumatoid arthritis.

However, inflammatory arthropathy, sacroiliac nerve entrapment and rheumatoid arthritis would be expected to produce symptoms beyond the heel and are not common causes of isolated heel pain.

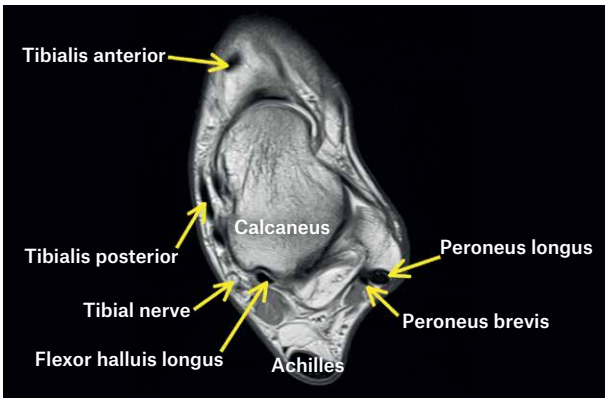


Figure 1. Axial magnetic resonance imaging (T1) of the calcaneus and surrounding structures.

Reproduced with permission from The Royal Australian College of General Practitioners, from Rio E, Mayes S, Cook J. Heel pain: A practical approach. Aust Fam Physician 2015;44(3):96–101.

Table 2. Subjective assessment questions to direct clinical reasoning

Question	Common responses	Diagnoses to consider
Age	Young	Common: Sever's disease (calcaneal apophysitis) Uncommon: calcaneal stress or tumour
	Middle-aged	Common: Achilles insertion tendinopathy, plantar fascia pain
	Older	Common: tibialis posterior tendinopathy and lengthening
What aggravates the pain?	Mechanical causes (eg walking, running)	Insertional Achilles tendinopathy (note, a warm-up phenomenon with activity is reported) Pain that increases with activity may indicate: • involvement of sheath (paratendinitis) • bone (stress reaction or stress fracture) • sinus tarsi or neural sources (including tarsal tunnel); prolonged standing can irritate tarsal tunnel.
	Non-mechanical	Pain at rest should be questioned further in terms of positioning and neural symptoms Tendon pain at rest is uncommon but pain on rising after sitting is a hallmark sign of tendinopathy
Was the onset of pain associated with an incident?	Yes: change to weight-bearing load (eg running or footwear)	Achilles tendinopathy Calcaneal bone stress
	No	Consider arthritic causes
Pain behaviour	Morning pain and stiffness	Achilles tendinopathy, flexor hallucis longus tenosynovitis, plantar fascia pain Long time to warm up (>60 minutes): consider rheumatological cause
	Night pain	Bone stress (eg calcaneal stress or more sinister causes)
General health questions and red flags	Night pain Other flags such as loss of weight, night sweats, joint pain and swelling	Tumour Consider non-musculoskeletal cause and include rheumatoid arthritis, gout, spondyarthopathies, infection
	Past injury	Repeated ankle sprains can commonly cause posterior impingement and sinus tarsi syndromes
Other	Cramping (calf and feet)	Vascular claudication Can be an early indicator of bone stress
	Neural symptoms	Sharp pain, burning, 'pins and needles' or numbness indicate neural involvement (eg tarsal tunnel syndrome [posterior tibial nerve and branches] present with symptoms in posteromedial ankle and heel and may extend to distal sole and toes)

Reproduced with permission from The Royal Australian College of General Practitioners, from Rio E, Mayes S, Cook J. Heel pain: A practical approach. Aust Fam Physician 2015;44(3):96–101.

Answer 3

The differential diagnoses can be further guided by considering George's age, asking general questions about his health, covering red flag conditions and other specific questions (Table 2):[3]

- What aggravates the pain?
- Was the onset of pain associated with a specific incident?
- What are the characteristics of the pain?

Red flag symptoms to be particularly aware of include:

- unrelenting pain including at night
- associated fevers, night sweats and weight loss
- other joint pain and swelling.

Answer 4

A number of objective examination tests are required to differentiate plantar fasciitis from other common causes of heel pain. These include:[3]

- functional tests such as walking
- calf raises, single and double
- tests of muscle strength against resistance
- special tests:
  - Simmonds' calf squeeze test for Achilles tendon rupture
  - posterior impingement test
  - calcaneal squeeze test for calcaneal stress fracture

Other tests are listed in Table 3.

Answer 5

Plantar fasciitis is diagnosed clinically, with a thorough history and examination. Laboratory tests and imaging are not usually necessary, although X-rays may be useful to rule out other potential causes of heel pain.[4]

When the diagnosis is unclear, however, plain weight-bearing radiographs of the foot are a reasonable first-line investigation. Modalities such as bone scans are useful for diagnosing calcaneal stress fractures; magnetic resonance imaging (MRI) is useful for the diagnosis of tumours, plantar fascia tears, infections and nerve entrapment.[3]

Management strategies are listed in Table 4. Patient handouts describing stretching exercises are available at The Royal Australian College of General Practitioners' website (refer to 'Resources for patients and doctors').

Answer 6

Important diagnoses to consider include:[2,5]

- inflammatory arthropathy – polyarthropathy would be expected peripheral neuropathy – plantar parasthaesia would be expected

Table 3. Objective assessment

Site/type of pain	Common sources of pain
Observation	Skin – colour, bruising, swelling, abrasions, rashes, Achilles tendon swelling / thickening or obvious deformity, muscle wasting
Functional – walking	Limping, avoiding joint movement or loading
Calf raise – double or single	Should be capable of lifting body weight on each leg at least 10 times. If there is an inability to do this, consider Achilles tendon rupture or tibialis posterior tear
Palpation	The sural nerve can be easily palpated lateral to the Achilles tendon with the ankle in dorsiflexion Sinus tarsi pain can indicate local and subtalar joint synovitis Plantar fascia attachment on the medial process of the calcaneal tuberosity Tibialis posterior and flexor hallucis longus (FHL) tendons posteromedial to medial malleolus Apophyses such as calcaneal, navicular, base of 5th metatarsal • Note the Achilles tendon squeeze can be painful when the tendon is not the source of heel pain and is a poor diagnostic test Sites of bone stress: calcaneal squeeze for calcaneus, dorsal navicular and talar neck
Muscle strength	Resisted inversion in plantarflexion for tibialis posterior Resisted eversion for peroneals. Observe peroneal tendon does not sublux around lateral malleolus
Other – special tests	Specific tests such as the Simmonds' calf squeeze test for suspected Achilles tendon rupture Posterior impingement test and FHL testing should reproduce symptoms Crepitus and clicking not always associated with symptoms Gentle palpation of the Achilles during active plantarflexion and dorsiflexion may demonstrate crepitus consistent with Achilles paratendinitis (sheath inflammation) Tinel's sign is well described and involves 4–6 taps over the nerve (such as sural or tibial) and should elicit 'pins and needles' or tingling
Neural testing	Straight leg raise with bias for peroneal nerve (add adduction, ankle plantar flexion and inversion) or tibial nerve (add dorsiflexion and eversion) Commonly – this may not reproduce the pain but an asymmetry can be noted Seated slump with lumbar kyphosis or lordosis

Reproduced with permission from The Royal Australian College of General Practitioners, from Rio E, Mayes S, Cook J. Heel pain: A practical approach. Aust Fam Physician 2015;44(3):96–101.

- stress fracture – more likely in long-distance runners and patients with osteoporosis and osteopenia, but could be relevant in this case.

Answer 7

Nonsurgical treatment is the standard of care for acute heel pain, although 10% of cases are recalcitrant and unresponsive to nonsurgical care after 12 months. In recalcitrant cases, surgery has been shown to be generally effective.

Conservative treatment involves combination therapy. This includes:[6,7]

- avoiding activities that aggravate the condition, or use of ice after aggravating activities that cannot be avoided
- avoiding walking in flat shoes or bare feet
- exercises to stretch and strengthen the calf muscles
- stretching the fascia
- use of a heel cup or cushion
- pharmacotherapy for pain relief
  - oral non-steroidal anti-inflammatory drugs
  - corticosteroid injections into the region of attachment.

The use of orthoses, night splints and low-dye taping have little evidence to support efficacy, and extracorporeal shockwave therapy has not been shown to be beneficial.[6,7]

Table 4. Evidence-based and clinical opinion for management by cause

Cause of pain	Recommendations	No evidence or not advised
Achilles tendon insertion pain or retrocalcaneal bursitis	Heel raises in shoes or added externally to shoes (to reduce compression at the Haglund prominence)	Avoid stretching and the eccentric heel drop program (due to compression). May be completed to plantargrade
	Graded strength rehabilitation from flat ground into plantar flexion	Intra-tendinous injections
	Other: polypill (ibuprofen, epigallocatechin gallate and doxycycline)[18]	Rest/ice/anti-inflammatory medications have limited efficacy: there is no evidence of inflammation in chronic tendinopathy
Posterior impingement syndrome	Calf strengthening – single leg heel raises, 25+ repetitions in a painfree range of motion in younger people Manual techniques that involve sub-talar joint distraction	Compressive positions or forced ankle joint plantar flexion
FHL (usually tenosynovitis)	Calf strengthening – single leg heel raises, 25+ repetitions in a pain-free range of movement in younger people Hirrudoid/diclofenac gel wrap – good clinical results with physiological justification (heparin-based treatments block the formation of fibrin associated with crepitus[17]	Eccentric exercises and stretches in full dorsiflexion or heel off step
Neural – entrapment (including tarsal tunnel)	Neural mobilisation	Neural tension exercise
	Check for direct compression (eg footware)	
	Treat underlying pathology of FHL	
Plantar fascia pain	Taping and orthotics may offer relief Strengthening of the foot intrinsics, calf and kinetic chain Low height isometric heel raise sustained hold	
Tibialis posterior tendinopathy (may be tenosynovitis)	Taping and orthotics Heel raise Strengthening in good ankle alignment	Eccentric exercise often increases the friction around the medial malleolus and exacerbates symptoms
Calcaneal bone stress **Talar stress fracture** **Navicular stress fracture**	Reduction in load and may require non-weight bearing	
Apophysitis	Reduction in load May benefit from a heel cup with heel raise in shoes to reduce traction of the apophysis Taping and orthotics Graded strengthening program Improving muscle compliance of the gastrocnemius, soleus and tibialis posterior	Though stretching is regularly recommended, initially it is painful especially if felt at the insertion

FHL, flexor hallucis longus

Reproduced with permission from The Royal Australian College of General Practitioners, from Rio E, Mayes S, Cook J. Heel pain: A practical approach. Aust Fam Physician 2015;44(3):96–101.

If George does not respond to conservative management, he may need to be referred to a specialist.

Resources for patients and doctors

- HANDI – Making non-drug interventions easier to find and use – Stretching exercises for plantar fasciitis, www.racgp.org.au/your-practice/guidelines/handi/interventions/other/stretching-exercises-for-plantar-fasciitis/
- Rio E, Mayes S, Cook J. Heel pain: A practical approach. Aust Fam Physician 2015;44(3):96–101 www.racgp.org.au/afp/2015/march/heel-pain-a-practical-approach
- Schwartz EN, Su J. Plantar Fasciitis: A Concise Review. The Permanente Journal. 2014;18(1):e105-e107. doi: 10.7812/TPP/13-113. Available at www.ncbi.nlm.nih.gov/pubmed/24626080/

References

1. BMJ Best practice. Plantar fasciitis. London: BMJ Publishing Group. Available at https://bestpractice.bmj.com/topics/en-us/487 [Accessed 23 July 2017].
2. Schwartz EN, Su J. Plantar fasciitis: A concise review. Perm J 2014;18(1):e105-07. doi: 10.7812/TPP/13-113.
3. Rio E, Mayes S, Cook J. Heel pain: A practical approach. Aust Fam Physician 2015;44(3):96–101. Available at www.racgp.org.au/afp/2015/march/heel-pain-a-practical-approach [Accessed 23 July 2018].
4. Cole C, Seto C, Gazewood J. Plantar fasciitis: Evidence-based review of diagnosis and therapy. Am Fam Physician 2005;72(11):2237–42.
5. Bower G, Uglow M. The ankle and foot. In: Blom A, Warwick D, Whitehouse MR, editors. Apley and Solomon's System of orthopaedics and trauma. 8th edn. Boca Raton, Florida: CRC Press, 2018; p. 609–51.
6. McPoil T, Martin R, Cornwall M, Wukich D, Irrgang J, Godges J. Heel Pain Plantar Fasciitis: Clinical Practice Guidelines Linked to the International Classification of Functioning, Disability, and Health from the Orthopaedic Section of the American Physical Therapy Association. J Orthop Sports Phys Ther 2008;38:2–18.
7. Expert Group for Rheumatology. Rheumatology: Limb conditions: Plantar fasciitis. In: eTG complete [Internet]. Melbourne: Therapeutic Guidelines Limited, 2018.

CASE

2 Jeffrey has joint pain

Jeffrey, aged 69 years, presents to you with a painful, swollen right knee. He has had pain for a few years, but it became worse after playing soccer with his grandson two weeks ago. Jeffrey has hypertension and type 2 diabetes. He takes perindopril 5 mg with amlodipine 5 mg daily, and metformin 1000 mg twice a day.

Question 1

What differential diagnoses would you consider? What information would you elicit on history-taking and examination?

Further information

Jeffrey's history and examination findings are consistent with uncomplicated osteoarthritis. His blood pressure is 150/95 mmHg, his height is 1.75 m, weight 86 kg and body mass index (BMI) 28 kg/m^2. Jeffrey requests magnetic resonance imaging (MRI) of his knee because his friend from the bowling club had a scan and ended up having 'key-hole' surgery to his knee.

Question 2

What is the role of imaging for osteoarthritis of the knee?

Further information

You advise Jeffrey that in many middle-aged people, changes are seen with MRI, but these changes are often not the cause of their symptoms. You add that after examining him, you are confident that his knee pain is caused by osteoarthritis and that MRI could lead to unnecessary and costly surgery and further complications. You recommend starting with some basic treatment and consider doing an X-ray if his symptoms do not improve within the next couple of months. Jeffrey agrees and asks what you think is wrong with his knee.

Question 3

How would you explain the underlying pathophysiology of his knee pain to Jeffrey?

Further information

You tell Jeffrey that osteoarthritis occurs when the joint surface (cartilage) becomes worn and that around 50% of people experience some form of osteoarthritis by age 65 years. You add that there is good evidence that exercise and weight loss can improve symptoms. Jeffrey asks what can be done for his knee pain.

Question 4

What is the evidence behind non-drug management of knee osteoarthritis?

Question 5

What pharmacological options could be considered for managing Jeffrey's knee pain?

Question 6

When would referral be indicated for Jeffrey's knee osteoarthritis?

CASE 2 Answers

Answer 1

Osteoarthritis is the most common form of arthritis, presenting with joint pain, stiffness and limitation of function.[1] It usually affects one or a few joints, but multiple joints can be involved. Osteoarthritis usually has an onset in patients aged >40 years; however, the age of onset may be earlier if there is a predisposing condition or injury. Overweight and obesity also predispose to osteoarthritis.

The main symptoms of osteoarthritis are pain and stiffness. The pain has an insidious onset, progresses over years, varies in intensity and may be intermittent; it is typically increased by activity and relieved by rest, although patients with severe osteoarthritis may have pain at rest.

Osteoarthritis usually affects both knees, but the pain may be worse in one knee.[2] Stiffness is generally short-lived (<30 minutes) and occurs early in the morning or with inactivity.

Physical examination findings characteristic of osteoarthritis include:

- bony enlargement and weakness/wasting of the muscles around the joint
- crepitus
- minimal or no redness, warmth or swelling
- periarticular and joint line tenderness
- reduced range of motion.

Instability in the knee can be due to muscle weakness but is sometimes due to true joint instability. Anterior knee pain and difficulty with activities, such as negotiating stairs or rising from a chair, suggest patellofemoral osteoarthritis.

Other causes of knee pain include gout, pseudogout, haemarthrosis, joint sepsis, referred pain, fracture and malignancy. Autoimmune inflammatory arthritis, such as psoriatic arthritis, can present with pain and inflammation in a single joint such as the knee. Inflammatory joint pain tends to improve with exercise and is associated with prolonged morning stiffness. The prolonged nature of the morning stiffness sometimes assists in differentiation between inflammatory arthritis and osteoarthritis but not in all cases. Crystal arthritis may co-exist with osteoarthritis but is generally associated with acute pain, swelling and warmth, with significant restriction of mobility, fever and raised inflammatory markers. Fractures of the tibial plateau have a relatively acute onset and may be related to a low-trauma event in elderly people or those with osteoporosis. Joint infection and spontaneous haemarthrosis can present with similar symptoms, but the latter is unusual in the absence of an underlying bleeding disorder, anticoagulation or severe trauma. If infection is suspected, a joint aspirate is indicated to confirm organisms and/or crystals. Referred pain to the knee can come from the hip or the lumbosacral spine. Malignancy should be considered if the pain is constant, increasing in severity, disturbing sleep or associated with systemic features such as weight loss.

Answer 2

A confident diagnosis of knee osteoarthritis can be made without imaging when typical features are present,[3] but imaging may be required when there is diagnostic uncertainty or to assess benefit from surgery.[4]

The Medicare Benefits Schedule (MBS) rebates for MRI of the knee are currently available on referral by general practitioners (GPs) for patients aged 16 years or older with acute knee trauma and with:[5]

- inability to extend the knee, suggesting the possibility of acute meniscal tear; or
- clinical findings suggesting acute anterior cruciate ligament tear.

MRI is a key imaging tool for knee osteoarthritis research,[6] but is unhelpful for decision making in most cases because around 90% of middle-aged and elderly people have lesions on MRI, whether or not knee pain is present. MRI has a false-positive rate of 65% for medial tears and 43% for lateral meniscus tear.[7] MRI could reduce the need for arthroscopy, but even if anterior cruciate ligament or meniscal tears are present, the latter may improve without surgery.[8] MRI could be considered when the patient has not responded to 6–8 weeks of conservative management and the diagnosis is unclear, or when there is suspicion of complex or unusual pathology.[9]

First-line imaging would usually be a plain anteroposterior weight-bearing X-ray of the knee, particularly when fracture is a possibility. Changes seen on an X-ray, such as marginal osteophyte formation, subchondral sclerosis and cysts, help to confirm the diagnosis of osteoarthritis, and assessment of joint space narrowing can suggest progression of osteoarthritis,[6] although X-ray changes do not always correlate with the severity of symptoms and progression is slow and changes are not seen in everyone.[4,10]

Further investigations, such as ultrasonography, computed tomography (CT) or bone scans, may be required if the X-ray is normal or suggests an alternative underlying process. Ultrasonography is generally unhelpful in the investigation of osteoarthritis and would only be indicated if questioning tendon injury. CT can be useful in evaluating tibial stress fractures in acute worsening of pain, but would probably only be used to

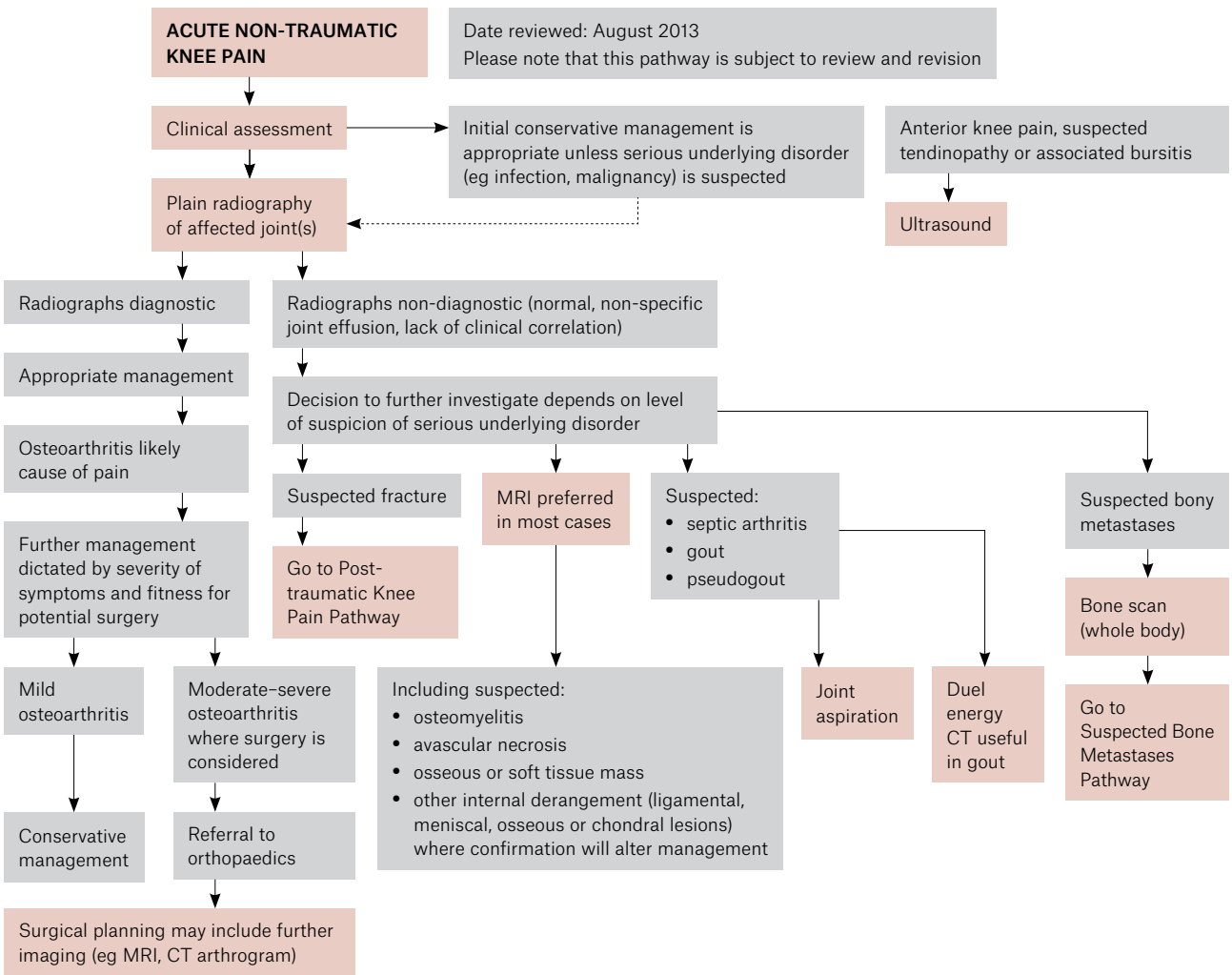


Figure 1. Flowchart for the use of imaging in osteoarthritis[47]

Reproduced with permission from Diagnostic Imaging Pathways, Western Australia Department of Health

define the affected area after an MRI. Bone scans can indicate subchondral bone pathology, but in weighing up risks and benefits, X-ray and MRI would be the two most valuable tools clinically with CT if further information is needed. Joint aspiration, inflammatory markers and screening for gout and inflammatory arthritis may be necessary (Figure 1).

Answer 3

Osteoarthritis is a degenerative disease of articulating joints, characterised by articular cartilage degeneration, subchondral bone sclerosis and osteophyte formation. Changes are also observed in the menisci, synovium, ligaments and periarticular muscle. Although the importance of local joint pathology, loading and biomechanics is well recognised, pain associated with knee osteoarthritis does not always correlate with radiographic severity[11] – 30–50% of patients with severe osteoarthritis and joint damage are asymptomatic, whereas about 10% with moderate-to-severe knee pain have normal X-rays.[12,13] The disconnect between peripheral structural damage and pain has been attributed to other dimensions of the patient's experience, including psychological distress, high comorbidity load and pain sensitisation in the central nervous system (CNS).[14]

Pain sensitisation can explain why patients complain of more pain than can be attributed to structural changes.[15] For this reason, pain neuroscience education is an important component of treatment in knee osteoarthritis. Education regarding pain biology and the role of the CNS reduces fear of structural damage, fear of movement and exercise, and pain catastrophising, which are factors linked to pain and disability in knee osteoarthritis.[16] Improved pain-related knowledge reduces the severity of musculoskeletal pain.[17] Useful resources for pain education and improving health literacy include the book *Explain pain*[18] and web-based services.[19]

Table 2. Summary of recommendations for non-surgical, non-drug options investigated for the management of knee osteoarthritis

	Knee osteoarthritis only – no comorbidity	Knee osteoarthritis only – plus comorbidity	Multi-joint osteoarthritis – no comorbidity	Multi-joint osteoarthritis – plus comorbidity	Quality of evidence	Effect size: minor; small; moderate; large*
Land-based exercise	**Appropriate**	**Appropriate**	**Appropriate**	**Appropriate**	Good	Small – short-term benefits for pain and physical function
Water-based exercise	**Appropriate**	**Appropriate**	**Appropriate**	**Appropriate**	Good	Small to moderate – short-term benefits for function and quality of life Minor – pain
Lower limb strength training	**Appropriate**	**Appropriate**	**Appropriate**	**Appropriate**	Good	Moderate – pain, physical function
Walking aid – cane	**Appropriate**	**Appropriate**	Uncertain	Uncertain	Fair	Not available
Weight management	**Appropriate**	**Appropriate**	**Appropriate**	**Appropriate**	Good	Not categorised
Self-management and education	**Appropriate**	**Appropriate**	**Appropriate**	**Appropriate**	Good	Moderate – pain and disability
Biomechanical interventions such as knee braces, knee sleeves and orthoses	**Appropriate**	**Appropriate**	**Appropriate**	**Appropriate**	Fair	Not available
Spa/balneotherapy	Uncertain	Uncertain	Uncertain		Fair	Not available
Acupuncture	Uncertain	Uncertain	Uncertain	Uncertain	Good	Not categorised
Walking aid – crutches	Uncertain	Uncertain	Uncertain	Uncertain	No trials	Not available
Electrotherapy/ neuromuscular electrical stimulation	Not appropriate	Not appropriate	Not appropriate	Not appropriate	Fair	Not available
Transcutaneous electrical nerve stimulation (TNS)	Uncertain	Uncertain	Not appropriate	Not appropriate	Good	No effect size reported as a primary result
Ultrasound	Uncertain	Uncertain	Not appropriate	Not appropriate	Good	Not categorised

Adapted from 2014 OARSI Guideline.

Note 1: 'Comorbidity' specifically includes diabetes, hypertension, cardiovascular disease, renal failure, gastrointestinal bleeding, depression or physical impairment limiting activity, including obesity.

**Where pooling was performed and effect size categorised.*

Answer 4

Nonsurgical, non-drug options can be used alone or in combination with medication. These options are recommended in the Osteoarthritis Research Society International (OARSI) guidelines[20] (Table 2) and discussed below.

Weight loss

Weight management is recommended when BMI is ≥25 kg/m^2.[20,21] Weight loss of >7% body weight is required to achieve a meaningful change in symptoms.[22] A combination of diet and exercise has the greatest effect on pain and function.[23] This combination is most desirable given the importance of preserving lean muscle mass – potentially undermined by caloric restriction[23] – and lower limb strength in the presence of osteoarthritis.

Exercise

The Royal Australian College of General Practitioners (RACGP) guidelines[21] recommend exercise as a 'core treatment for

Table 3. Summary of recommendations for pharmacologic options investigated for the management of knee osteoarthritis

	Knee osteoarthritis only – no comorbidity	Knee osteoarthritis only – plus comorbidity	Multi-joint osteoarthritis – no comorbidity	Multi-joint osteoarthritis – plus comorbidity	Quality of evidence	Effect size: standardised mean difference for pain*
Paracetamol (Acetaminophen)	**Appropriate**	Uncertain	**Appropriate**	Uncertain	Good	0.18 (0.11–0.25)
Capsaicin	**Appropriate**	Uncertain	Uncertain	Uncertain	Good	Not available
Corticosteroids (intra-articular injection)	**Appropriate**	**Appropriate**	**Appropriate**	**Appropriate**	Good	Not available
Chondroitin (for symptom relief)	Uncertain	Uncertain	Uncertain	Uncertain	Good	0.13 (0.00– 0.27) to 0.75 (0.50–0.99)
Chondroitin (for disease modification)	Not Appropriate	Not Appropriate	Not Appropriate	Not Appropriate	Good	0.26 (0.14–0.38) to 0.30 (0.00–0.59)
Duloxetine	**Appropriate**	Uncertain	**Appropriate**	**Appropriate**	Fair	Not available
Glucosamine (for symptom relief)	Uncertain	Uncertain	Uncertain	Uncertain	Good	0.17 (0.05–0.28) to 0.47 (0.23–0.72)
Glucosamine (for disease modification)	Not Appropriate	Not Appropriate	Not Appropriate	Not Appropriate	Good	0.08 (0.12-0.27)
Hyaluronic acid (intra-articular injection)	Uncertain	Uncertain	Not Appropriate	Not Appropriate	Good	0.37 (0.28–0.46) to 0.46 (0.28–0.65)
Non-steroidal anti-inflammatory drugs (oral non-selective)	**Appropriate**	Not Appropriate#	**Appropriate**	Not Appropriate#	Good	0.37 (0.26–0.49)
Non-steroidal anti-inflammatory drugs (oral cyclooxygenase-2 inhibitors)	**Appropriate**	Not Appropriate#	**Appropriate**	Not Appropriate#	Good	0.44 (0.33–0.55)
Non-steroidal anti-inflammatory drugs (topical)	**Appropriate**	**Appropriate**	Uncertain	Uncertain	Good	Not available
Opioids (transdermal)	Uncertain	Uncertain	Uncertain	Uncertain	Good	0.22 (0.03– 0.42) to 0.36 (0.26–0.47)
Opioids (oral)	Uncertain	Uncertain	Uncertain	Uncertain	Good	0.36 (0.26–0.47) to 0.51 (0.01–1.01)
Risedronate	Not Appropriate	Not Appropriate	Not Appropriate	Not Appropriate	Poor	Not available
Avocado soybean unsaponfiables	Uncertain	Uncertain	Uncertain	Uncertain	Good	0.39 (0.01–0.76)
Rosehip	Uncertain	Uncertain	Uncertain	Uncertain	Good	0.37 (0.13–0.60)

Adapted from 2014 OARSI Guideline

Note 1: 'Comorbidity' specifically includes diabetes, hypertension, cardiovascular disease, renal failure, gastrointestinal bleeding, depression or physical impairment limiting activity, including obesity.

**If the level of evidence included a meta-analysis, the Estimated Effect Size for pain versus control is stated from that meta-analysis. Only pooled effect sizes reported as a standardized mean difference (SMD) are reported. #Assessed as not appropriate if high comorbidity risk; in moderate comorbidity risk deemed uncertain appropriateness.*

osteoarthritis in all clinical guidelines regardless of patient age, pain levels or disease severity.' Land-based and/or water-based programs are recommended for improvement in pain and function (Table 2). Programs focusing on one fitness domain (eg strength) or multiple (eg strength, aerobic) are effective.[24] Programs should be supervised or monitored, carried out three times a week[25] and maintained for a minimum of 8–12 weeks if large effects are to be obtained.[26] Booster sessions by physiotherapists, motivational strategies and behavioural graded exercise may help improve long-term adherence.[27]

Interdisciplinary management and coordinated care

The reliance on multiple treatment options provided by different healthcare practitioners necessitates a coordinated care approach for patients with osteoarthritis. Coordinated, evidence-based interdisciplinary management would optimise the use of nonsurgical treatment options; however, low referral rates for nonsurgical, non-drug treatments are seen among Australian GPs.[28,29]

Models such as the NSW Osteoarthritis Clinical Care Pathway (OACCP)[30] include a musculoskeletal care coordinator based in a public hospital, leading a team of physiotherapists, dietitians, occupational therapists and psychologists. Most patients access this service via the orthopaedic waiting list, but some services accept direct GP referral. Evaluation of models such as the OACCP showed some patients withdrew from surgical waiting lists because they no longer required surgery.[31] The GP Management Plan could be used to refer patients for up to five sessions per year for private allied health services subsidised through the MBS. In the UK, physiotherapists in private clinics have led coordinated care approaches,[32] although this avenue has not been well explored in Australia.

Answer 5

Osteoarthritis often co-exists with conditions associated with ageing and obesity, including diabetes, hypertension and renal disease. Poor vision, mood disturbance and social isolation also need to be considered when managing osteoarthritis in older people. Medication should be prescribed as part of a holistic management plan for the patient. Table 3 summarises the OARSI recommendations for pharmacological management of knee osteoarthritis.

Non-steroidal anti-inflammatory drugs (NSAIDs)

Topical NSAIDs can be a short-term adjunct to other treatment strategies in people with osteoarthritis of the knee. Oral NSAIDs are more effective than paracetamol but have a greater potential for harm. A trial of an oral NSAID may be considered as first-line treatment for patients at low risk of harm. Factors that increase the risk of harm from NSAIDs include older age, history of gastrointestinal bleeding or peptic ulcer, *Helicobacter pylori* infection, concomitant use of drugs that increase bleeding risk, and smoking. NSAIDs are not recommended for people with cardiovascular risk factors, people at risk of volume depletion and people with significant hypertension or renal impairment.[33,34] NSAIDs selective for cyclooxygenase (COX)-2 have a lower risk of gastrointestinal toxicity than non-selective NSAIDs, but concomitant use of low-dose aspirin eliminates any upper gastrointestinal safety advantage of COX-2 inhibitors.

Intra-articular corticosteroids

The use of intra-articular steroids for symptomatic management of knee osteoarthritis is supported by two systematic reviews.[35,36] However, they should not be used in people in whom intra-articular or skin infection/ulceration is known or suspected to be present, and are generally not given more than once every three months. If the patient has very severe osteoarthritis, intra-articular steroids are likely to be less effective. A recent systematic review concluded that the available studies of intra-articular infective complications following joint replacement in people who had previously received intra-articular steroid were underpowered and at risk of selection bias. No level 1 or 2 studies were available for review, and three of the four studies that were available showed no evidence of increased intra-articular infection risk, while one showed a significantly increased infection risk.[37]

Opioids

The OARSI guidelines concluded that both transdermal and oral opioid analgesia have an 'uncertain' role in the management of osteoarthritis of the knee[20] because, although data support small-to-moderate improvements in pain with opioid analgesia,[38,39] there is also clear evidence of a significantly increased risk of adverse events, especially in elderly people and those with comorbidities. The decision to use opioids should be weighed carefully by GPs and by patients in relation to their specific circumstances.

Other therapies

A recent review has found that studies examining platelet-rich plasma (PRP) are of low-to-moderate methodological quality.[40] In general, PRP was found to be safe and had the potential to provide symptomatic benefit in the short term (up to 12 months). However, no conclusion could be drawn about the effect of PRP on structural changes in osteoarthritis, or the effects of PRP in osteoarthritis generally. This treatment can be costly and, in the absence of high-quality data, does not have a clear role in the management of knee osteoarthritis at this time.

Mesenchymal stem cells (MSCs) are pluripotent stem cells found in numerous adult human tissues, including adipose tissue and bone marrow. They are treated then re-injected into the damaged joint, or bound to a scaffold and embedded into a cartilage defect. A recent systematic review found trials to be at high risk of bias. The studies reported improvement in pain and function and improved radiological and histological outcomes when compared with controls at 24 and 48 months of follow-up, but because of the low-quality evidence, it was concluded that MSC therapy currently cannot be recommended for the treatment of knee osteoarthritis.[41]

There is some evidence of pain reduction with low-dose fish oil of about 1.5 standard capsules a day;[42] however, the

additional calories in fish oil may make losing weight more difficult. Glucosamine and chondroitin sulphate have shown variable results in the management of knee osteoarthritis and cannot be recommended. There is insufficient evidence to support the use of turmeric in knee osteoarthritis.

Answer 6

First-line management for moderate-to-severe knee osteoarthritis should include referral for multidisciplinary care, as mentioned above.

GPs can play a key role in preventing unsuitable referral. Guidelines generally recommend rheumatology referral when the diagnosis is unclear, to provide a second opinion and to assist with pain management, including intra-articular injections if the GP is unable to administer them.[21,43,44] There is insufficient evidence to support arthroscopy and meniscectomy as first-line management, even in symptomatic meniscal tears.[45] Referral for joint replacement is generally recommended when there is pain, stiffness and impaired function that reduces the patient's quality of life,[46] after having tried more conservative management.[20] Factors that may affect the outcome, including the age of the patient and presence of centralised pain, should be taken into consideration when deciding whether referral for knee replacement is appropriate. Referral should be made before there is established functional restriction and severe pain.[46] Localised health pathways are being developed at a Primary Health Network level that can assist in determining the criteria for referrals to pain clinics when pain becomes chronic and refractory to conservative measures, and surgery is contraindicated.

Resources for doctors

- Australian Commission on Safety and Quality in Health Care. Osteoarthritis of the Knee Clinical Care Standard. Sydney: ACSQHC; 2017; www.safetyandquality.gov.au/wp-content/uploads/2017/05/Osteoarthritis-of-the-Knee-Clinical-Care-Standard-Booklet.pdf
- NPS MedicineWise. Physical Examination of acute ankle and knee injuries (video); www.nps.org.au/_scrivito/physical-examination-of-acute-ankle-and-knee-injuries-8508d0c1755fafcb
- The Royal Australian College of General Practitioners. Guideline for the non-surgical management of hip and knee osteoarthritis. RACGP; 2009; www.racgp.org.au/download/documents/Guidelines/Musculoskeletal/racgp_oa_guideline.pdf

References

1. National Collaborating Centre for Chronic Conditions, National Institute for Health and Clinical Excellence: Guidance. Osteoarthritis: National Clinical Guideline for Care and Management in Adults. London: Royal College of Physicians (UK), 2008.
2. Doherty M, Abishek A. Clinical manifestations and diagnosis of osteoarthritis. In: Post T, ed. UpToDate. Waltham, MA: UpToDate Inc, 2017.
3. Australian Commission on Safety and Quality in Health Care. Osteoarthritis of the Knee Clinical Care Standard. Sydney: ACSQHC, 2017. Available at www.safetyandquality.gov.au/wp-content/uploads/2017/05/Osteoarthritis-of-the-Knee-Clinical-Care-Standard-Booklet.pdf [Accessed 25 July 2018].
4. Mathiessen A, Cimmino MA, Hammer HB, Haugen IK, Iagnocco A, Conaghan PG. Imaging of osteoarthritis (OA): What is new? Best Pract Res Clin Rheumatol 2016;30(4):653–69. doi: 10.1016/j.berh.2016.09.007.
5. Australian Government Department of Health. MBS Online Medicare Benefits Schedule. Canberra, ACT: Australian Government Department of Health. Available at www.mbsonline.gov.au [Accessed 25 July 2018].
6. Roemer FW, Eckstein F, Hayashi D, Guermazi A. The role of imaging in osteoarthritis. Best Pract Res Clin Rheumatol 2014;28(1):31–60. doi: 10.1016/j.berh.2014.02.002.
7. Guermazi A, Niu J, Hayashi D, et al. Prevalence of abnormalities in knees detected by MRI in adults without knee osteoarthritis: population based observational study (Framingham Osteoarthritis Study). BMJ 2012;345:e5339. doi: 10.1136/bmj.e5339.
8. The Royal Australian College of General Practitioners. Clinical guidance for MRI referral. Melbourne: RACGP, 2013. Available at www.racgp.org.au/your-practice/guidelines/mri-referral/mri-of-the-knee/ [Accessed 25 July 2018].
9. National Prescribing Service MedicineWise. Acute knee pain presentations in middle-aged patients: what is the role of MRI? Canberra, ACT: NPS MedicineWise, 2016. Available at www.nps.org.au/medical-info/clinical-topics/news/acute-knee-pain-presentations-in-middle-aged-patients-what-is-the-role-of-mri [Accessed 25 July 2018].
10. Lawrence JS, Bremner JM, Bier F. Osteo-arthritis. Prevalence in the population and relationship between symptoms and X-ray changes. Ann Rheum Dis 1966;25(1):1–24.
11. Finan PH, Buenaver LF, Bounds SC, et al. Discordance between pain and radiographic severity in knee osteoarthritis: findings from quantitative sensory testing of central sensitization. Arthritis Rheum 2013;65(2):363–72. doi: 10.1002/art.34646.
12. Hannan MT, Felson DT, Pincus T. Analysis of the discordance between radiographic changes and knee pain in osteoarthritis of the knee. J Rheumatol 2000;27(6):1513–17.
13. Creamer P, Hochberg MC. Why does osteoarthritis of the knee hurt--sometimes? Br J Rheumatol 1997;36(7):726–28.
14. Dell'Isola A, Allan R, Smith SL, Marreiros SS, Steultjens M. Identification of clinical phenotypes in knee osteoarthritis: a systematic review of the literature. BMC Musculoskeletal Disord 2016;17(1):425. doi: 10.1186/s12891-016-1286-2.
15. Arendt-Nielsen L. Pain sensitisation in osteoarthritis. Clin Exp Rheumatol 2017;35:Suppl 107(5):68–74.
16. Somers TJ, Keefe FJ, Godiwala N, Hoyler GH. Psychosocial factors and the pain experience of osteoarthritis patients: new findings and new directions. Curr Opin Rheumatol 2009;21(5):501–06. doi: 10.1097/BOR.0b013e32832ed704.
17. Lee H, McAuley JH, Hubscher M, Kamper SJ, Traeger AC, Moseley GL. Does changing pain-related knowledge reduce pain and improve function through changes in catastrophizing? Pain 2016;157(4):922–30. doi: 10.1097/j.pain.0000000000000472.
18. Butler DS, Moseley L. Explain pain. 2nd ed. Adelaide: NOI Group Publishing, 2013.
19. Osteoarthritis Healthy Weight for Life. Available at https://oa.hwfl.com.au/ [Accessed 1 May 2018].
20. McAlindon TE, Bannuru RR, Sullivan MC, et al. OARSI guidelines for the non-surgical management of knee osteoarthritis. Osteoarthritis Cartilage 2014;22(3):363–88. doi: 10.1016/j.joca.2014.01.003.
21. The Royal Australian College of General Practitioners. Guideline for the non-surgical management of hip and knee osteoarthritis.

Melbourne: RACGP, 2013. Available at www.racgp.org.au/your-practice/guidelines/musculoskeletal/hipandkneeosteoarthritis/ [Accessed 25 July 2018].

22. Atukorala I, Makovey J, Lawler L, Messier SP, Bennell K, Hunter DJ. Is there a dose-response relationship between weight loss and symptom improvement in persons with knee osteoarthritis? Arthritis Care Res (Hoboken) 2016;68(8):1106–14.
23. Messier SP, Mihalko SL, Legault C, et al. Effects of intensive diet and exercise on knee joint loads, inflammation, and clinical outcomes among overweight and obese adults with knee osteoarthritis: The IDEA randomized clinical trial. JAMA 2013;310(12):1263–73. doi: 10.1001/jama.2013.277669.
24. Uthman OA, van der Windt DA, Jordan JL, et al. Exercise for lower limb osteoarthritis: systematic review incorporating trial sequential analysis and network meta-analysis. BMJ 2014;48(21):1579. doi: 10.1136/bmj.f5555.
25. Juhl C, Christensen R, Roos EM, Zhang W, Lund H. Impact of exercise type and dose on pain and disability in knee osteoarthritis: a systematic review and meta-regression analysis of randomized controlled trials. Send to Arthritis Rheumatol 2014;66(3):622–36. doi: 10.1002/art.38290.
26. Young JL, Rhon DI, Cleland JA, Snodgrass SJ. The influence of exercise dosing on outcomes in patients with knee disorders: A systematic review. J Orthop Sports Phys Ther 2018;48(3):146–61. doi: 10.2519/jospt.2018.7637.
27. Nicolson PJA, Bennell KL, Dobson FL, Van Ginckel A, Holden MA, Hinman RS. Interventions to increase adherence to therapeutic exercise in older adults with low back pain and/or hip/knee osteoarthritis: a systematic review and meta-analysis. Br J Sports Med 2017;51(10):791–99. doi: 10.1136/bjsports-2016-096458.
28. Basedow M, Williams H, Shanahan EM, Runciman WB, Esterman A. Australian GP management of osteoarthritis following the release of the RACGP guideline for the non-surgical management of hip and knee osteoarthritis. BMC Res Notes 2015;8:536. doi: 10.1186/s13104-015-1531-z.
29. Brand CA, Harrison C, Tropea J, Hinman RS, Britt H, Bennell K. Management of osteoarthritis in general practice in Australia. Arthritis Care Res (Hoboken) 2014;66(4):551–58. doi: 10.1002/acr.22197.
30. Agency for Clinical Innovation Musculoskeletal Network. Osteoarthritis Chronic Care Program Model of Care. Chatwood, NSW: Agency for Clinical Innovation, 2012.
31. Deloitte Access Economics. Osteoarthritis Chronic Care Program evaluation. Kingston, ACT: Deloitte Access Economics, 2014. Available at www.aci.health.nsw.gov.au/__data/assets/pdf_file/0009/259794/oaccp-evaluation-feb-2015.pdf [Accessed 25 July 2018].
32. Walker A, Williams R, Sibley F, Stamp D, Carter A, Hurley M. Improving access to better care for people with knee and/or hip pain: service evaluation of allied health professional-led primary care. Musculoskeletal Care 2018;16(1):222–32. doi: 10.1002/msc.1189.
33. Nissen SE, Yeomans ND, Solomon DH, et al. Cardiovascular safety of celecoxib, naproxen, or ibuprofen for arthritis. N Eng J Med 2016;375(26):2519–29. doi: 10.1056/NEJMoa1611593.
34. Expert Group for Rheumatology. Rheumatology: Principles of nonsteroidal anti-inflammatory drugs use for musculoskeletal conditions in adults. In: eTG complete [Internet]. Melbourne: Therapeutic Limited, 2018.
35. Bannuru RR, Natov NS, Obadan IE, Price LL, Schmid CH, McAlindon TE. Therapeutic trajectory of hyaluronic acid versus corticosteroids in the treatment of knee osteoarthritis: a systematic review and meta-analysis. Arthritis Rheum 2009;61(12):1704e11.
36. Bellamy N, Campbell J, Robinson V, Gee T, Bourne R, Wells G. Intraarticular corticosteroid for treatment of osteoarthritis of the knee. Cochrane Database Syst Rev 2006;(2):CD005328.
37. Marsland, Daniel; Mumith, Aadil; Barlow, Ian W. Systematic review: The safety of intra-articular corticosteroid injection prior to total knee arthroplasty. Knee 2014;21(1):6–11. doi: 10.1016/j.knee.2013.07.003.
38. Nuesch E, Rutjes AW, Husni E, Welch V, Juni P. Oral or transdermal opioids for osteoarthritis of the knee or hip. Cochrane Database Syst Rev.2009(4):Cd003115. doi: 10.1002/14651858.CD003115.pub3.
39. Cepeda MS, Camargo F, Zea C, Valencia L. Tramadol for osteoarthritis: A systematic review and metaanalysis. J Rheumatol 2007;34(3):543–55.
40. Bennell KL, Hunter DJ, Paterson KL. Platelet-rich plasma for the management of hip and knee osteoarthritis. Curr Rheumatol Rep 2017;19(5):24. doi: 10.1007/s11926-017-0652-x.
41. Pas HI, Winters M, Haisma HJ, Koenis MJ, Tol JL, Moen MH. Stem cell injections in knee osteoarthritis: a systematic review of the literature. Br J Sports Med 2017;51(15):1125–33. doi: 10.1136/bjsports-2016-096793.
42. Hill CL, March LM, Aitken D, et al. Fish oil in knee osteoarthritis: a randomised clinical trial of low dose versus high dose. Ann Rheum Dis 2016;75(1):23–29. doi: 10.1136/annrheumdis-2014-207169.
43. American College of Rheumatology. Referral Guidelines. Atlanta, GA: American College of Rheumatology, 2015. Available at www.rheumatology.org/Portals/0/Files/Referral%20Guidelines.pdf [Accessed 25 July 2018].
44. Government of Western Australia Department of Health. Rheumatology Referral Guidelines – Specialist Adult Clinical Priority Access Criteria. Perth, WA: WA Health, 2014. Available at www.gp.health.wa.gov.au/CPAC/speciality/guidelines/Rheumatology%20CPAC%20Referral%20Recommendations.pdf [Accessed 25 July 2018].
45. Buchbinder R, Harris I. Treatment of meniscal year. Anna Intern Med 2016;165(8):602–03. doi: 10.7326/L16-0327.
46. Conaghan PG, Dickson J, Grant RL. Care and management of osteoarthritis in adults: summary of NICE guidance. BMJ 2008;336(7642):502–03. doi: 10.1136/bmj.39490.608009.AD.
47. Diagnostic Imaging Pathways, Western Australia Department of Health. Diagnostic Imaging Pathways - Knee Pain (Non-Traumatic). Perth, WA: WA Heath, 2013. Available at www.imagingpathways.health.wa.gov.au/index.php/image-galleries/medical-images/musculoskeletal-trauma?id=314#pathway [Accessed July 25 2018].

CASE

3 Colin has a Colles'

Colin, 55 years of age, presents to you with a letter from the local hospital and a cast on his left arm. He had tripped over the dog, falling onto his outstretched hand. The discharge letter describes a minimally displaced fracture of the distal radius, which was plastered by the physiotherapist.

Question 1

What further information would you seek from Colin?

Further information

This was Colin's first fall causing an injury; he sustained no other injuries. His pain is currently well controlled and he does not have any altered sensation in his left hand. He has not had any previous fractures; however, his mother recently fractured her hip. He has no significant past illnesses and takes no regular medications. He has never taken glucocorticoids, selective serotonin reuptake inhibitors or proton pump inhibitors. Colin works as a software engineer. He does not undertake any regular physical activity. He smokes 10–12 cigarettes per day and consumes no alcohol. He lives alone with his dog. Colin reports gastrointestinal upset when having milk, so he minimises his consumption of dairy products. Colin usually maintains a balance of fruits, vegetables and meat. His weight has been stable. On examination his body mass index (BMI) is 18.4 kg/m^2. There are no signs of hypogonadism, Cushingoid features or other endocrinopathies, inflammatory arthritis, malignancy or chronic liver disease. The plaster is intact and his left hand has good capillary refill, movement and sensation.

Question 2

How likely is it that Colin has underlying osteoporosis?

Question 3

What further investigations should be considered for Colin? Why?

Further information

Colin's dual energy X-ray absorptiometry (DXA) result shows a femoral neck T-score of –1.9. His blood tests are normal, apart from a vitamin D level of 18 nmol/L (target >50 nmol/L).

Question 4

What non-drug options are available for Colin to prevent a second fracture from occurring?

Question 5

What drug options are available? What factors would guide the choice of drug for Colin?

Further information

Colin agrees to increase his regular physical activity and spend more time in the sun. He is not ready to stop smoking but will commence vitamin D and calcium supplements. He is given a prescription for an oral bisphosphonate with instructions to take it on an empty stomach and remain in an upright position for 30 minutes after taking the drug.

Question 6

When should Colin be reviewed? What would be indications for further referral?

CASE 3 Answers

Answer 1

As Colin's general practitioner (GP), you should assess whether he has developed any symptoms suggesting neurovascular compromise post-fracture, including paraesthesia, weakness or change in skin colour. You should also check that he has adequate analgesia and how he is coping with work and activities such as personal care and driving. It is important to ensure that Colin is aware of proper plaster care and encouraged to move his fingers.

Although Colin presents for a plaster check, you should additionally consider:

- other associated injuries sustained during this fall
- risk factors for further falls
- risk factors he has for sustaining a minimal trauma fracture (Table 1).

Physical examination should include review of the plaster and exclude underlying risk factors for falls and minimal trauma fracture.

Answer 2

Osteoporosis is a common condition. Fragility fractures affect one in three women and one in five men over the age of 50 years.[1–3] Osteoporosis in men is often under-diagnosed; however, the risk of having an osteoporotic fracture after the age of 50 years is higher than the risk of having prostate cancer.[3,4]

Colin has sustained a fracture from minimal trauma (caused by a fall from a standing height or less) and, therefore, may be at risk of underlying osteoporosis and future fractures. A prior fracture is associated with an 86% increased risk of any fracture.[5]

His other risk factors from your assessment so far include his history of minimal trauma fracture, family history of hip fracture, low BMI, low intake of dairy products, with possible lactose intolerance.

Given his presentation with minimal trauma fracture, he should undergo further investigations. It is estimated that 80% of individuals who have had a minimal trauma fracture

Table 1 – Risk factors for osteoporosis and minimal trauma fractures[49]

Personal risk factors	Medical conditions	Behavioural	Medications
Female sex Low body weight Older age Caucasian or Asian ethnicity Previous minimal trauma fractures Family history of osteoporosis or fractures History of falls Propensity to fall	Premature menopause Metabolic disorders (eg primary and secondary hypogonadism in men) Rheumatoid arthritis Malabsorption disorders (eg coeliac disease) Physical disabilities that restrict weight-bearing exercise	Smoking Excessive alcohol consumption Physical inactivity Poor calcium intake Lack of sunlight exposure	Long-term corticosteroid use Androgen deprivation agents Aromatase inhibitors Proton pump inhibitors Selective serotonin reuptake inhibitors

and are at increased risk of another fracture are neither identified nor treated.[6]

Answer 3

Because Colin has sustained a minimal trauma fracture, he qualifies for DXA funded through the Medicare Benefits Schedule (MBS). DXA is the preferred method of measuring bone mineral density (BMD; Box 1). The information obtained can then be used in an absolute fracture risk algorithm (eg the Garvan Fracture Risk Calculator available at www.garvan.org.au/bone-fracture-risk or the Fracture Risk Assessment Tool available at www.shef.ac.uk/FRAX) to more accurately determine Colin's individual fracture risk and assist him in deciding on treatment.[7]

DXA results are reported as T-score (number of standard deviations from bone density in healthy young adults) and Z-score (number of standard deviations from an age-matched and sex-matched control group). The World Health Organization (WHO) definitions of osteoporosis and osteopaenia are:[7]

- Normal: T-score of –1 or greater
- Osteopaenia/low bone mass: T-score between –1 and –2.5
- Osteoporosis: T-score of –2.5 or less

Box 1. Medicare rebates for DXA[50]

Item 12306: one service only in 24 months
- One or more minimal trauma fractures
- Monitoring of known low bone density

Item 12312: one service only in 12 months
- Prolonged glucocorticoid therapy
- Conditions associated with glucocorticoid secretion
- Male hypogonadism
- Female hypogonadism lasting six months before the age of 45 years

Item 12315: one service only in 24 months
- Primary hyperparathyroidism
- Chronic liver/renal disease
- Proven malabsorption disorders
- Rheumatoid arthritis
- Conditions associated with thyroxine excess

Item 12320: one service only in five years
- 70 years and over AND either:
  - not previously had a BMD OR
  - T-score is –1.5 or greater

Item 12321: one service only in 12 months
- BMD measurement at least 12 months after a significant change in therapy

Item 12322: one service only in 2 years
- 70 years and over AND T-score is less than –1.5 but more than –2.5

BMD, bone mineral density; DXA, dual energy X-ray absorptiometry

Fragility fractures often occur in individuals with osteopenia or osteoporosis; however, underlying conditions that may contribute to low BMD and fragility fracture[8] should be excluded. These include:

- endocrine causes (eg hypogonadism, Cushing's syndrome, hyperparathyroidism, hyperthyroidism, diabetes)
- other systemic illness associated with:
  - inflammation (eg connective tissue disease)
  - malabsorption (eg coeliac disease)
  - chronic organ failure (eg liver, kidney failure)
  - malignancy (eg myeloma)
- medications:
  - glucocorticoids used for more than three months
  - anticonvulsants
  - pioglitazone, rosiglitazone
  - anti-androgen, anti-oestrogen agents
  - chemotherapy.

There is no international consensus guideline regarding appropriate investigations for secondary causes of osteoporosis in a patient with minimal risk factors. However, proposed guidelines[9] and local HealthPathways[10] suggest the following baseline investigations:

- full blood count
- electrolytes, urea, creatinine
- liver function tests, albumin, globulin, alkaline phosphatase
- erythrocyte sedimentation rate
- calcium, phosphate
- 25-hydroxyvitamin D
- thyroid function tests.

Further tests should be done if there are abnormalities in the baseline tests or if they are indicated in the history/clinical examination.

Bone turnover markers are rarely indicated in the first instance; their role in monitoring osteoporosis treatment is not well established.[7]

Answer 4

Colin may be contacted by the fracture liaison coordinator at the hospital where he was treated for his fracture. Effective models have been developed in multiple countries, including Australia, to deliver secondary preventive care in the form of osteoporosis re-fracture prevention (ORP) services for patients presenting with minimal trauma fractures.[11] The coordinated approach provided by ORP includes:[12]

1. identification of patients with osteoporosis
2. investigation and determination of individual fracture risk (including falls risk)
3. initiation of interventions known to reduce fracture risk.

Rates of post-fracture assessment and treatment for secondary fracture prevention have been as low as 20%. Where an ORP is not available, the same approach can be undertaken in primary care, as should the continuation of interventions commenced by the ORP.

Lifestyle modifications should be considered in all patients with osteoporosis and include strategies to increase bone strength and reduce the risk of falls. International guidelines recommend healthy lifestyle choices to reduce risks associated with osteoporosis.[13,14] For Colin there are four key non-drug options to consider in his management plan, as outlined in the osteoporosis treatment algorithm in Figure 1.[7]

Colin should be encouraged and supported to undertake a weight-bearing exercise and strength training program,[15] to have a healthy diet including adequate intake of calcium (1000 mg/daily from dietary sources and supplements)[16] and protein, maintain a healthy weight and BMI, and have safe levels of sun exposure. Colin may require additional calcium supplementation if he cannot meet the minimum daily requirement through his diet. Colin's low BMI (18.4 kg/m^2) doubles his risk of hip fracture independently of BMD, and therefore referral to a dietitian and exploration of his low BMD should be considered. Colin should also maintain low or no alcohol consumption and cease smoking, as this is associated with increased rates of bone loss and fracture.[17,18]

Providing Colin with education, self-management resources and access to falls prevention programs is important. He may be able to access these through an ORP or through a combination of local service providers and peak national groups (eg Osteoporosis Australia).

Answer 5

Osteoporosis-specific pharmacotherapy approximately halves subsequent fracture risk and potentially reduces the risk of premature mortality. Much of this high-quality data is based on studies involving postmenopausal women; however, a similar direction of improvement in BMD and anti-fracture efficacy has underpinned the clinical utility in men.

Pharmacotherapy options include anti-resorptive (bisphosphonates and denosumab) and anabolic (teriparatide) agents. Testosterone replacement potentially improves bone density in men with hypogonadism but lacks good quality anti-fracture efficacy data.

Anti-resorptive agents (target osteoclast-mediated bone resorption)

Bisphosphonates

Alendronate, risedronate and zoledronic acid have been shown to reduce vertebral fractures by 40–70%, hip fractures by 50% and non-vertebral fractures by 25–40%.[19–28] Zoledronic acid has also been shown to reduce mortality.[29,30]

Bisphosphonates are relatively well tolerated, although myalgia (usually short-lived and managed with paracetamol) and hypocalcaemia can occur. Patients must be vitamin D replete prior to therapy, especially when using zoledronic acid. Oral options can potentially cause upper gastrointestinal disturbance in some patients.

Two rare side effects of bisphosphonates, osteonecrosis of the jaw (ONJ) and atypical femoral shaft fractures, have attracted considerable interest in recent years. Practical recommendations include having patients attend to dental work prior to starting therapy, reassuring patients and dentists alike about the low rate of ONJ (1 in 10,000 to 1 in 100,000 patient years), and being vigilant to the rare risk of atypical fractures (3.2–50 cases per 100,000 patient years). In

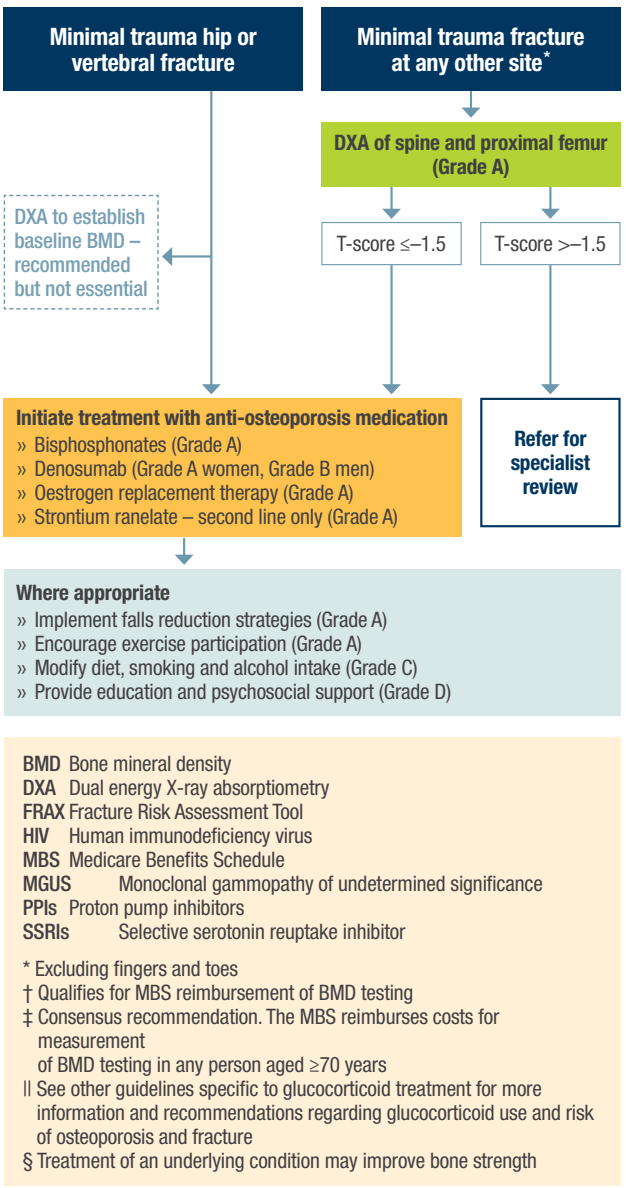


Figure 1. Osteoporosis risk assessment, diagnosis and management flowchart

Adapted with permission from The Royal Australian College of General Practitioners from Osteoporosis prevention, diagnosis and management in postmenopausal women and men over 50 years of age. 2nd edn. Melbourne: RACGP, 2017.

most cases, the risk of osteoporotic fracture or re-fracture far outweighs the extremely low risk of ONJ and atypical fractures.[31–34] As a result, the concept of a drug holiday/cessation is probably overemphasised and should only be carefully offered to patients where the risk of osteoporotic fracture is deemed to have reduced considerably or where the risks of therapy are thought to outweigh the benefits.

Denosumab

Denosumab has been shown to reduce vertebral fractures by 68%, nonvertebral fractures by 20% and hip fractures by 40%, with similar BMD improvements seen in men and women.[35–37] Denosumab is administered by six-monthly subcutaneous injections, which are well tolerated and have comparable efficacy to bisphosphonates. It can also be used in patients with stage IV kidney disease and above, although calcium levels need to be closely monitored in this group (ensure vitamin D is replete prior to therapy).

Compliance with denosumab is crucial because bone turnover increases rapidly if a dose is missed, potentially resulting in an increase in vertebral fractures.[38] This issue is particularly important in younger patients, where the choice of denosumab may commit patients to indefinite therapy, which in turn increases the likelihood of sub-optimal adherence. Risks of ONJ and atypical fractures, while extremely rare, are still evident with denosumab. Nonetheless, the anti-fracture efficacy of denosumab is comparable to bisphosphonates, and it is a first-line option in the management of osteoporosis.

Anabolic agents (stimulate bone formation by osteoblasts)

Teriparatide is a recombinant analogue of human parathyroid hormone (PTH) that, via daily subcutaneous injection, provides intermittent PTH, which enables anabolic effects on bone health. Teriparatide has been shown to reduce the risk of vertebral fractures by 65% and non-vertebral fractures by 35%, although no difference was seen with hip fractures.[39]

Colin is not currently eligible for access to this therapy through the Pharmaceutical Benefits Scheme, as it is limited to patients with two or more fractures despite anti-resorptive therapy and a T-score of less than –3. It is only available once in a patient's lifetime and must be initiated by a specialist. After the 18-month treatment course, consolidation with an anti-resorptive agent is necessary to maintain the improvement in bone density.

An anti-resorptive agent would be the most suitable first-line choice for Colin. There is no direct evidence to recommend bisphosphonates ahead of denosumab, or vice versa. Consideration of comorbidities, preferences and adherence may help to inform a final decision.

Answer 6

Follow-up for patients is important to monitor for side effects and to check adherence to pharmacological and non-pharmacological management. An estimated 50% of Spanish primary care patients were found to be still taking their osteoporosis medication at 12 months, although adherence to parenteral agents was higher.[40] Adherence rates for oral anti-resorptive agents was similar when followed up by an ORP, compared with follow-up by a primary care practitioner.[41] The Royal Australian College of General Practitioners (RACGP) osteoporosis guidelines[7] recommend follow-up at 3–6 months, then annually.

Serial DXA scans are not more predictive of subsequent fracture than the baseline measurement.[42] A repeat DXA scan may only be needed to check efficacy of treatment (eg if the patient re-fractures), or if changes or cessation of therapy are being considered. Although improvements in BMD may be expected with treatment, these effects may be modest.[43–45] Increased BMD can account for less than 25% of the overall reduction in fracture risk,[43] and in some studies, despite a reduction in BMD, fracture risk reduction was seen.[46] If a repeat DXA scan is indicated, many guidelines recommend a minimum of two years before repeating DXA because of limitations in the precision of testing.[7,47] Cessation of bisphosphonate therapy could be considered after 5–10 years of treatment if BMD T-scores are greater than –2.5 and there have been no further fractures or evidence of bone loss while on therapy.[7]

Referral for exercise therapy, dietetic advice, physiotherapy and falls prevention services should be considered for all patients with fragility fractures. There is little research published around specialist referral pathways for osteoporosis. Guidelines for GPs regarding referral for men with osteoporosis include those aged <50 years with severe osteoporosis, multiple or recurrent fractures, inadequate response to therapy, complications of treatment[48] or lack of access to a bone densitometry service.[7] In Australia, endocrinologists and rheumatologists have special interests in managing osteoporosis. Gynaecologists, orthopaedic surgeons and geriatricians also commonly manage patients with osteoporosis. Lack of clarity as to who should assume clinical responsibility for caring for patients with fragility fractures and osteoporosis may be one of the barriers to implementing secondary prevention of these fractures.[12]

Resources for doctors

- RACGP and Osteoporosis Australia. Osteoporosis prevention, diagnosis and management in postmenopausal women and men over 50 years of age. 2nd edn; www.osteoporosis.org.au/sites/default/files/files/20439%20Osteoporosis%20guidelines.pdf
- Osteoporosis Australia fact sheets

References

1. Melton LJ, 3rd, Atkinson EJ, O'Connor MK, O'Fallon WM, Riggs BL. Bone density and fracture risk in men. J Bone Miner Res 1998;13(12):1915–23.
2. Melton LJ, 3rd, Chrischilles EA, Cooper C, Lane AW, Riggs BL. Perspective. How many women have osteoporosis? J Bone Miner Res 1992;7(9):1005–10.
3. Kanis JA, Johnell O, Oden A, et al. Long-term risk of osteoporotic fracture in Malmo. Osteoporos Int 2000;11(8):669–74.
4. Merrill RM, Weed DL, Feuer EJ. The lifetime risk of developing prostate cancer in white and black men. Cancer Epidemiol Biomarkers Prev 1997;6(10):763–68.

5. Kanis JA, Johnell O, De Laet C, et al. A meta-analysis of previous fracture and subsequent fracture risk. Bone 2004;35(2):375–82.
6. Nguyen TV, Center JR, Eisman JA. Osteoporosis: underrated, underdiagnosed and undertreated. Med J Aust 2004;180(5 Suppl):S18–22.
7. The Royal Australian College of General Practitioners and Osteoporosis Australia. Osteoporosis prevention, diagnosis and management in postmenopausal women and men over 50 years of age. 2nd edn. Melbourne: RACGP, 2017.
8. Premaor MO, Compston JE. Testing for secondary causes of osteoporosis. BMJ 2010;341:c6959. doi: 10.1136/bmj.c6959.
9. Compston J, Cooper A, Cooper C, et al. UK clinical guideline for the prevention and treatment of osteoporosis. Arch Osteoporos 2017;12(1):43. doi: 10.1007/s11657-017-0324-5.
10. HealthPathways Sydney. Osteoporosis. Available at sydney.healthpathways.org.au/index.htm [Accessed 25 July 2018].
11. Mitchell PJ. Best practices in secondary fracture prevention: fracture liaison services. Curr Osteoporos Rep 2013;11(1):52–60. doi: 10.1007/s11914-012-0130-3.
12. Elliot-Gibson V, Bogoch ER, Jamal SA, Beaton DE. Practice patterns in the diagnosis and treatment of osteoporosis after a fragility fracture: a systematic review. Osteoporos Int 2004;15(10):767–78.
13. Cosman F, Beur S, LeBoff M, et al. Clinician's Guide to Prevention and Treatment of Osteoporosis. Osteoporos Int 2014;25(10):2359–81. doi: 10.1007/s00198-014-2794-2.
14. Scottish Intercollegiate Guidelines Network. Management of Osteoporosis and the prevention of fragility fractures. Edinburgh: SIGN, 2015.
15. Michaelsson K, Olofsson H, Jensevik K, et al. Leisure physical activity and the risk of fracture in men. PLoS Med 2007;4(6):e199. doi: 10.1371/journal.pmed.0040199.
16. Department of Health and Ageing and National Health and Research Council. Nutrient reference values for Australia and New Zealand. Canberra. Canberra: DoHA and NHMRC, 2006.
17. Drake MT, Murad MH, Mauck KF, et al. Clinical review. Risk factors for low bone mass-related fractures in men: a systematic review and meta-analysis. J Clin Endocrinol Metab 2012;97(6):1861–70. doi: 10.1210/jc.2011-3058.
18. Jutberger H, Lorentzon M, Barrett-Connor E, et al. Smoking predicts incident fractures in elderly men: Mr OS Sweden. J Bone Miner Res 2010;25(5):1010–16. doi: 10.1359/jbmr.091112.
19. Black DM, Cummings SR, Karpf DB, et al. Randomised trial of effect of alendronate on risk of fracture in women with existing vertebral fractures. Fracture Intervention Trial Research Group. Lancet 1996;348(9041):1535–41.
20. Cummings SR, Black DM, Thompson DE, et al. Effect of alendronate on risk of fracture in women with low bone density but without vertebral fractures: results from the Fracture Intervention Trial. JAMA 1998;280(24):2077–82.
21. Black DM, Thompson DE, Bauer DC, et al. Fracture risk reduction with alendronate in women with osteoporosis: the Fracture Intervention Trial. FIT Research Group. J Clin Endocrinol Metab 2000;85(11):4118–24.
22. Harris ST, Watts NB, Genant HK, et al. Effects of risedronate treatment on vertebral and nonvertebral fractures in women with postmenopausal osteoporosis: a randomized controlled trial. Vertebral Efficacy With Risedronate Therapy (VERT) Study Group. JAMA 1999;282(14):1344–52.
23. Reginster J, Minne HW, Sorensen OH, et al. Randomized trial of the effects of risedronate on vertebral fractures in women with established postmenopausal osteoporosis. Vertebral Efficacy with Risedronate Therapy (VERT) Study Group. Osteoporos Int 2000;11(1):83–91.
24. McClung MR, Geusens P, Miller PD, et al. Effect of risedronate on the risk of hip fracture in elderly women. Hip Intervention Program Study Group. N Engl J Med 2001;344(5):333–40.
25. Watts NB, Josse RG, Hamdy RC, et al. Risedronate prevents new vertebral fractures in postmenopausal women at high risk. J Clin Endocrinol Metab 2003;88(2):542–49.
26. Black DM, Delmas PD, Eastell R, et al. Once-yearly zoledronic acid for treatment of postmenopausal osteoporosis. N Engl J Med 2007;356(18):1809–22.
27. Zhou J, Wang T, Zhao X, Miller DR, Zhai S. Comparative efficacy of bisphosphonates to prevent fracture in men with osteoporosis: A systematic review with network meta-analyses. Rheumatology and therapy 2016;3(1):117–28. doi: 10.1007/s40744-016-0030-6.
28. Xu Z. Alendronate for the treatment of osteoporosis in men: A meta-analysis of randomized controlled trials. Am J Ther 2017;24(2):e130–e138. doi: 10.1097/MJT.0000000000000446.
29. Lyles KW, Colon-Emeric CS, Magaziner JS, et al. Zoledronic acid and clinical fractures and mortality after hip fracture. N Engl J Med 2007;357(18):1799–09. doi: 10.1056/NEJMoa074941.
30. Boonen S, Reginster JY, Kaufman JM, et al. Fracture risk and zoledronic acid therapy in men with osteoporosis. N Engl J Med 2012;367(18):1714–23. doi: 10.1056/NEJMoa1204061.
31. Gedmintas L, Solomon DH, Kim SC. Bisphosphonates and risk of subtrochanteric, femoral shaft, and atypical femur fracture: a systematic review and meta-analysis. J Bone Miner Res 2013;28(8):1729–37. doi: 10.1002/jbmr.1893.
32. Jamal SA, Dion N, Ste-Marie LG. Atypical femoral fractures and bone turnover. N Engl J Med 2011;365(13):1261–62. doi: 10.1177/2040622315584114.
33. Khan AA, Morrison A, Hanley DA, et al. Diagnosis and management of osteonecrosis of the jaw: a systematic review and international consensus. J Bone Miner Res 2015;30(1):3–23. doi: 10.1002/jbmr.2405.
34. Black DM, Schwartz AV, Ensrud KE, et al. Effects of continuing or stopping alendronate after 5 years of treatment: The Fracture Intervention Trial Long-term Extension (FLEX): A randomized trial. JAMA 2006;296(24):2927–38. doi: 10.1001/jama.296.24.2927.
35. Cummings SR, San Martin J, McClung MR, et al. Denosumab for prevention of fractures in postmenopausal women with osteoporosis. N Engl J Med 2009;361(8):756–65. doi: 10.1056/NEJMoa0809493.
36. Papapoulos S, Lippuner K, Roux C, et al. The effect of 8 or 5 years of denosumab treatment in postmenopausal women with osteoporosis: results from the FREEDOM Extension study. Osteoporos Int 2015;26(12):2773–83. doi: 10.1007/s00198-015-3234-7.
37. Orwoll E, Teglbjaerg CS, Langdahl BL, et al. A randomized, placebo-controlled study of the effects of denosumab for the treatment of men with low bone mineral density. J Clin Endocrinol Metab 2012;97(9):3161–69. doi: 10.1210/jc.2012-1569.
38. Lamy O, Gonzalez-Rodriguez E, Stoll D, Hans D, Aubry-Rozier B. Severe Rebound-Associated Vertebral Fractures After Denosumab Discontinuation: 9 Clinical Cases Report. J Clin Endocrinol Metab 2017;102(2):354–58. doi: 10.1210/jc.2016-3170.
39. Neer RM, Arnaud CD, Zanchetta JR, et al. Effect of parathyroid hormone (1-34) on fractures and bone mineral density in postmenopausal women with osteoporosis. N Engl J Med 2001;344(19):1434–41.
40. Martin-Merino E, Huerta-Alvarez C, Prieto-Alhambra D, Montero-Corominas D. Cessation rate of anti-osteoporosis treatments and risk factors in Spanish primary care settings: a population-based cohort analysis. Archives of osteoporosis. 2017;12(1):39. doi: 10.1007/s11657-017-0331-6.
41. Ganda K, Schaffer A, Pearson S, Seibel MJ. Compliance and persistence to oral bisphosphonate therapy following initiation within a secondary fracture prevention program: a randomised controlled trial of specialist vs. non-specialist management. Osteoporos Int 2014;25(4):1345–55. doi: 10.1007/s00198-013-2610-4.

42. Hillier TA, Stone KL, Bauer DC, et al. Evaluating the value of repeat bone mineral density measurement and prediction of fractures in older women: the study of osteoporotic fractures. Arch Intern Med 2007;167(2):155–60.
43. Small RE. Uses and limitations of bone mineral density measurements in the management of osteoporosis. MedGenMed 2005;7(2):3.
44. Gallagher JC, Rosen CJ, Chen P, Misurski DA, Marcus R. Response rate of bone mineral density to teriparatide in postmenopausal women with osteoporosis. Bone 2006;39(6):1268–75.
45. Bone HG, Bolognese MA, Yuen CK, et al. Effects of denosumab on bone mineral density and bone turnover in postmenopausal women. J Clin Endocrinol Metab 2008;93(6):2149–57. doi: 10.1210/jc.2007-2814.
46. Sebba AI. Significance of a decline in bone mineral density while receiving oral bisphosphonate treatment. Clinical Ther 2008;30(3):443–52. doi: 10.1016/j.clinthera.2008.03.008.
47. Nordin C. Screening for osteoporosis: U.S. Preventive Services Task Force recommendation statement. Ann Intern Med 2011;155(4):276; author reply 276–77. doi: 10.7326/0003-4819-154-5-201103010-00307.
48. Diamond T, Sambrook P, Williamson M, et al. Guidelines for treatment of osteoporosis in men. Aust Fam Physician 2001;30(8):787–91.
49. Kanis JA, Borgstrom F, De Laet C, et al. Assessment of fracture risk. Osteoporos Int 2005;16(6):581–89.
50. Australian Government Department of Health. Medicare Benefits Schedule. Book Category 2. Diagnostic procedures and investigations. Canberra: Commonwealth of Australia; 2018.

CASE

4 Ben has chronic flank pain

Your senior partner has retired, leaving you his patient Ben, an ex-security officer aged 52 years, whom you had previously seen occasionally. Ben has chronic flank pain of renal and/or lumbar origin. He has a referral to a urologist for assessment. His past history includes morbid obesity, refractory hypertension despite treatment with four antihypertensives, gout, sleep apnoea, foot surgery and aspiration of a renal cyst. He receives a script for tramadol slow release 100 mg and/or paracetamol/codeine 500/30 mg every couple of months. Ben's pain score is 5/10 on a visual analogue scale. You give him repeat scripts for his analgesic medications while he awaits his urology appointment.

Question 1

What do guidelines based on the World Health Organization (WHO) analgesic ladder advise about appropriate pain management?

Question 2

Why should we do pain scores? Are any suitable for the time-poor general practitioner (GP)?

Further information

Ben's blood pressure and medication regimen remain problematic despite numerous fax exchanges with the pharmacist. You have added pregabalin for neuropathic pain and await the urology outpatient clinic's scan. Today he twisted his ankle and he sees you for repeat scripts for both analgesics, which you provide.

Question 3

Who initiates long-term opioid therapy (LtOT)?

Further information

Over the next few months, you and your GP colleagues continue to grapple with Ben's many problems. Following review of Ben's scans, the urologists conclude that his flank pain is not urological and they recommend future follow-up by the neurosurgery clinic. Ben returns to see you and reports that he has been bed-bound for four days because of the pain. He says he feels 'empty' and unmotivated and so has stopped attending the gym. You are concerned about Ben's mental health and suggest a referral to a psychologist, but he declines as he finds talking about his pain a waste of time. You then commence duloxetine, although Ben protests, 'my pain is real and not just in my mind'.

Question 4

Why are psychological aspects of pain important? How can consideration of these aspects guide patient care?

Further information

Over the next six months, Ben has several hospital admissions for neck spasms and chest pain requiring oxycodone immediate-release and diazepam. At one point a neighbour called an ambulance after overhearing odd noises and being unable to wake Ben. You continue to attend to all of Ben's multimorbidities and repeat scripts.

Question 5

What is the evidence about the effectiveness and safety of opioid analgesia in chronic non-cancer pain?

Further information

In the past month, Ben has been employed as a traffic controller. He comes to see you and is limping. He tells you that he is now doing long shifts at work and this has aggravated his old back and foot pains. He adds, 'I've had to use more of the pain medication to be able to get to work each day, and now I have none left.' You give him repeat scripts for his usual and hospital-initiated medications, with a warning that they can be addictive. Ben laughs and says, 'Oh, don't worry, there's no fear of that for me – I've never been drunk, never used drugs and never even smoked'.

Question 6

Is Ben an addict? If so, does that make you a mispresciber of opioids?

Further information

Ben sees you again and requests a medical certificate as his back pain is preventing him from working or sleeping. 'Do you think you could prescribe something to help me sleep?' he asks. You agree to give him the week off with a script for temazepam.

Question 7

What are the specific risks of prescribing benzodiazepines for Ben? What alternatives would you consider?

Further information

Ben is soon totally broke and looking for work again, having been forced to quit his job because of the pain. Over the next six months, while he is waiting for an epidural, the neurosurgeons have initiated oxycodone 40 mg/naloxone (sustained release).

Today he presents and is feeling great and finally 'pain-free'. He admits he has been getting muddled up about whether he has taken his medications or not and must have doubled them all up. He begins sweating, then vomits and collapses on your floor. His pupils are pin-point and not responding to light. You give him naloxone and call the ambulance. He is awake by the time they take him to the hospital.

Question 8

How common are overdoses in patients on LtOT? Who is at risk?

Further information

You are concerned that this is not Ben's first overdose. You wonder whether the emergency detected by his neighbour was a serotonin syndrome or an overdose. You are aware that

take-home or bystander naloxone is now on the Pharmaceutical Benefits Schedule and consider whether this would be appropriate for Ben.

Question 9

What are the indications for take-home naloxone? Is it appropriate to prescribe it for Ben and his neighbour?

Further information

Post-overdose, Ben is discharged with a simplified analgesia regimen of buprenorphine 20 µg/hour patches and oxycodone. His patches keep coming off because of sweating and the neurosurgeons switch the patches to tapentadol while he is waiting for treatment with epidural cortisone. The first epidural is temporarily successful, but the second one completely fails.

Today he presents, walking with a stick. The neurosurgeons have concluded that his pain is not due to a back problem and have discharged him from their clinic back to urology. Ben is not keen about this, as the previous renal cyst aspiration was both painful and unsuccessful.

He now is on tapentadol (sustained release) 100 mg twice daily, oxycodone 5 mg (immediate release) three times daily when required, diazepam 5 mg at night, pregabalin 300 mg twice daily, duloxetine 120 mg, four antihypertensives and allopurinol. He reports some urinary incontinence and forgetfulness. His pain score is now 10/10.

Question 10

How would you manage Ben from now?

CASE 4 Answers

Answer 1

Fifty years ago, the hospice movement advocated for liberal use of opioids in end-of-life care. To operationalise this, the WHO in 1986 developed a stepwise model. Mild pain (1–4/10) required non-opioid analgesics. As pain increased to moderate levels (5–8/10), analgesia progressed to 'weak' opioids (codeine or tramadol) or to 'strong' opioids for severe pain (8–10/10).[1] The palliative care experts also recommended a set of strategies, known as universal precautions,[2] to reassure authorities or clinicians concerned about turning patients with pain into addicts.

Answer 2

Pain scores allow clinicians to measure progress over time or across services. A 0–10 scale may be sufficient in terminal care or post-operatively but is simplistic for chronic pain care. This is because chronic pain, by definition, extends past the period of tissue healing and so may last for decades. Using such a scale predisposes to the provision of opioids,[1] which may account for their industrial support. The PEG (pain, enjoyment, general activity) outcome measure covers pain intensity, function and quality of life. It can be completed in less than a minute and is feasible for use in the general practice setting.[3]

Answer 3

It is never easy to identify who initiates LtOT. Patients with chronic pain are commonly already on opioids when they present to a new medical practitioner. Acute therapy provided intermittently and repeatedly quickly transitions to LtOT. Provision of more than a week's initial supply doubles the risk of continued use at one year (6–13%) and providing one month's supply increases it further (29.9%).[4] This case is an example of care focused on addictive pharmacotherapies rather than holistic care. A home medication review may help redirect polypharmacy in patients with multimorbidity.

Answer 4

It is important to explain to Ben that the brain and the mind are one and the same and are intertwined with all bodily functions. Managing both cognitive and affective issues helps address what may appear to be solely a physical problem to patients and biomedically focused clinicians.

Chronic pain care will be derailed by unidentified depression or anxiety. Treatments may include the scheduling of pleasurable activities, relaxation and exercise.[5] Calming the nervous system and mind can assist in relieving pain as well as treating insomnia and anxiety, if these are present. Teaching awareness to remove judgementalism, self-regulate distress and remain calm in the presence of pain is described as 'mindfulness'.[6] 'Self-compassion' describes a gentle acceptance of one's own limitations and is part of reducing a habitual stress response. Certain antidepressants, such as serotonin–noradrenaline reuptake inhibitors (SNRIs), low-dose

tricyclics and the melatonin agonist agomelatine, may have an impact on pain.[7] The numbers needed to treat to have a 50% reduction in pain with SNRIs such as duloxetine is 6.4.[8]

Recent studies of the endogenous opioid system reveal it has powerful roles apart from pain modulation, such as directing behaviours towards family and community.[9] Thus, social connectedness such as family and work relationships are legitimate analgesia focuses.

Opioid dose and duration has been associated with rates of new-onset treatment-resistant depression.[10]

Answer 5

The Centre for Disease Control is the peak public health body for the US. Its 2016 guidelines[11] advise that opioids should be avoided for chronic pain outside active cancer treatment, palliative care and end-of-life care. In other cases, they should be provided only for a few days.

Despite the prevalence of long-term opioid provision, this year, 2018, saw the first randomised controlled trial (RCT) of their long-term efficacy. It included 240 patients with musculoskeletal pain commencing comprehensive pain care. After three, six, nine and 12 months, those provided with opioids had no better pain intensity or function and had more adverse reactions to medications, compared with controls.[12]

Harms of LtOT include worsening pain, depression, sleep interference and poorer functional outcomes.[10,13]

Answer 6

The most significant barrier to a clinician providing opioid analgesics is fear about risking addiction.[14] The recommended 'universal precautions' reflect eminence-based medicine, not evidence-based medicine, and have not been shown to reliably predict or identify abusers or mitigate risks.[10,15] In a recent systematic review and data synthesis, the prevalence rates of misuse or addiction were estimated at about one in four and one in 10 respectively.[16] At a population level, safer prescribing restrictions do give some benefit, a reduction in deaths from overdose.[17]

While weaning or maintaining opioids, it is important to comply with state or territory regulations.[18] In every Australian jurisdiction, it is unacceptable to provide any opioids to a patient who is opioid-dependent, although each jurisdiction relies on a different definition of dependence. Misprescribing is the second most common reason, after sexual misbehaviour, that Australian doctors are sanctioned.[19]

Answer 7

The majority of GPs manage a presentation with insomnia by providing a benzodiazepine-related drug, such as temazepam.[20] Co-consumption of benzodiazepine-related drugs with opioids quadruples overdose rates.[21] Tolerance develops quickly, and over one year, people with sleeping difficulties who are on benzodiazepines deteriorate more than those not taking them.[22] Given that insomnia is common, it is important that GPs feel confident offering non-pharmacological options, such as cognitive behavioural therapy for insomnia (CBT-i). CBT-i includes four elements: relaxation therapy, psychoeducation/sleep hygiene, stimulus control and sleep (or bedtime) restriction strategies. CBT-i is said to produce reliable, durable benefits in 70–80% of patients.[23]

Answer 8

Accidental drug-related deaths have exceeded deaths from motor vehicle accidents since 2011.[24] While non-fatal overdoses exceed fatal ones by six to one, the risk increases with dose. Those on 100 mg morphine equivalents per day or more have about a ninefold increase in overdoses at a rate of 1.8% per annum.[25] Even after non-fatal overdoses, LtOT is rarely ceased, with a second overdose occurring in 7% of patients over the next two years.[26]

Answer 9

Take-home naloxone should be provided for those at higher risk of overdose, such as those who:

- have had a previously confirmed or suspected overdose
- are opioid-dependent or are on higher doses or undertaking opioid rotation
- have been recently released from prison
- have current or past alcohol or illicit drug use disorders
- are on, or recently been on, opioid substitution therapy
- are co-consuming other sedating drugs (eg benzodiazepines, Z-drugs or antipsychotics)
- have significant medical conditions, including sleep apnoea, liver disease or depression.

Answer 10

Ben's case suggests iatrogenic harm and the direction of his management needs to change. His incontinence may be from serotonin syndrome from his polypharmacy or a direct adverse effect from the duloxetine or oxycodone. His pain management has been very focused on a curative model of diagnosis and treatment aiming to reduce the sensory experience, but this has failed. Care needs to, first, shift to multimodal holistic pain care while, second, reducing and eventually ceasing the opioids. How to do this in Australian general practice has been recently reviewed by a multidisciplinary team.[27]

Holistic pain care will require specifically allocated appointments or referral to allied health practitioners or a multidisciplinary pain clinic. Care will address his diet and obesity, recognising the importance of gut–brain signalling, as well as insomnia and social isolation.[28,29] Ben will need to be assisted to increase movement and fitness, as well as address his anxiety, depression and therapeutic passivity.

These things can and should be done concurrently. A 22-week RCT of 35 outpatients with chronic pain on LtOT compared treatment as usual with administration of CBT-based pain self-management training by a physician assistant (not a

specialist psychologist). The opioid doses of the taper-support group were relatively decreased (though not statistically significantly), with no worsening of pain. Supported tapering did give significantly reduced pain interference, pain self-efficacy and perceived opioid problems.[30] Even in patients with substance use disorders and without specialist pain care, stopping opioids may improve pain outcomes. A Danish study of 551 patients showed that pain intensity remained similar or was slightly reduced one year after cessation of long-term opioids.[31]

References

1. Sullivan MD, Ballantyne J. Must we reduce pain intensity to treat chronic pain? Pain 2016;157(1):65–69. doi: 10.1097/j.pain.0000000000000336.
2. Gourlay DL, Heit HA, Almahrezi A. Universal precautions in pain medicine: A rational approach to the treatment of chronic pain. Pain Med 2005;6(2):107–12.
3. Krebs EE, Lorenz KA, Bair MJ, et al. Development and initial validation of the PEG, a three-item scale assessing pain intensity and interference. J Gen Intern Med 2009;24(6):733–38. doi: 10.1007/s11606-009-0981-1.
4. Shah A, Hayes C, Martin B. Characteristics of initial prescription episodes and likelihood of long-term opioid use — United States, 2006–2015. Morbidity and Mortality Weekly Report (MMWR) 2017;(66):265–69. doi: 10.15585/mmwr.mm6610a1.
5. Nicholas MK, Blyth FM. Are self-management strategies effective in chronic pain treatment? Pain manag 2016;6(1):75–88. doi: 10.2217/pmt.15.57.
6. Zgierska AE, Burzinski CA, Cox J, et al. Mindfulness meditation-based intervention is feasible, acceptable, and safe for chronic low back pain requiring long-term daily opioid therapy. J Altern Complement Med 2016;22(8):610–20. doi: 10.1089/acm.2015.0314.
7. Danilov A, Kurganova J. Melatonin in Chronic Pain Syndromes. Pain Ther 2016;5(1):1–17. doi: 10.1007/s40122-016-0049-y.
8. Finnerup NB, Attal N, Haroutounian S, et al. Pharmacotherapy for neuropathic pain in adults: a systematic review and meta-analysis. Lancet Neurol 2015;14(2):162–73. doi: 10.1016/S1474-4422(14)70251-0.
9. Carr DB. Endogenous opioids' primary role: Harmonizing individual, kin/cohort, and societal behaviors. Pain Med 2017;18(2):201–03. doi: 10.1093/pm/pnw362.
10. Scherrer JF, Salas J, Copeland LA, et al. Prescription opioid duration, dose, and increased risk of depression in 3 large patient populations. Ann Fam Med 2016;14(1):54–62. doi: 10.1370/afm.1885.
11. Dowell D, Haegerich T, Chou R. CDC guideline for prescribing opioids for chronic pain — United States, 2016. Morbidity and Mortality Weekly Report (MMWR) Recommendations and Reports [Internet] 2016;65(1):1–49. Available at www.cdc.gov/mmwr/volumes/65/rr/rr6501e1.htm [Accessed 25 July 2018].
12. Krebs EE, Gravely A, Nugent S, et al. Effect of opioid vs nonopioid medications on pain-related function in patients with chronic back pain or hip or knee osteoarthritis pain: The SPACE randomized clinical trial. JAMA 2018;319(9):872–82. doi: 10.1001/jama.2018.0899.
13. Angarita GA, Emadi N, Hodges S, Morgan PT. Sleep abnormalities associated with alcohol, cannabis, cocaine, and opiate use: a comprehensive review. Addic Sci Clin Pract 2016;11(1):1–17. doi: 10.1186/s13722-016-0056-7.
14. Potter M, Schafer S, Gonzalez-Mendez E, et al. Opioids for chronic nonmalignant pain. Attitudes and practices of primary care physicians in the UCSF/Stanford Collaborative Research Network. University of California, San Francisco. J Fam Pract. 2001;50(2):145–51.
15. Busse JW, Craigie S, Juurlink DN, et al. Guideline for opioid therapy and chronic noncancer pain. CMAJ 2017;189(18):E659–E66. doi: 10.1503/cmaj.170363.
16. Vowles KE, McEntee ML, Julnes PS, Frohe T, Ney JP, van der Goes DN. Rates of opioid misuse, abuse, and addiction in chronic pain: a systematic review and data synthesis. Pain 2015;156(4):569–76. doi: 10.1097/01.j.pain.0000460357.01998.f1.
17. Franklin GM, Mai J, Turner J, Sullivan M, Wickizer T, Fulton-Kehoe D. Bending the prescription opioid dosing and mortality curves: Impact of the Washington State opioid dosing guideline. Am J Ind Med 2012;55(4):325–31. doi: 10.1002/ajim.21998
18. Jammal W, Gown G. The pitfalls of opioid prescribing – What prescribers need to know. Aust Prescr 2015;38(6):198–203. doi: 10.18773/austprescr.2015.069.
19. Elkin K, Spittal M, Elkin D, Studdert D. Doctors disciplined for professional misconduct in Australia and New Zealand, 2000–2009. Med J Aust 2011;194(9):452–56.
20. Charles J, Harrison C, Britt H. Insomnia. Aust Fam Physician 2009;38(5):283.
21. Park TW, Saitz R, Ganoczy D, Ilgen MA, Bohnert ASB. Benzodiazepine prescribing patterns and deaths from drug overdose among US veterans receiving opioid analgesics: case-cohort study. BMJ 2015;350:h2698. doi: 10.1136/bmj.h2698.
22. Bourgeois J, Elseviers M, Van Bortel L, Petrovic M, Vander Stichele R. One-year evolution of sleep quality in older users of benzodiazepines: A longitudinal cohort study in belgian nursing home residents. Drugs Aging 2014;31(9):677–82. doi: 10.1007/s40266-014-0203-3.
23. Buysse DJ, Rush AJ, Reynolds III CF. Clinical management of insomnia disorder. JAMA 2017;318(20):1973–74. doi: 10.1001/jama.2017.15683.
24. Pentington Institute. Australia's Annual Overdose Report 2017. Carlton: Penington Institute, 2017. Available at www.penington.org.au/australias-annual-overdose-report-2017/ [Accessed 13 August 2018].
25. Dunn KM, Saunders KW, Rutter CM, Banta-Green CJ, Merrill JO, Sullivan MD, et al. Opioid prescriptions for chronic pain and overdose: a cohort study. Ann Intern Med 2010;152(2):85–92. doi: 10.7326/0003-4819-152-2-201001190-00006.
26. Larochelle MR, Liebschutz JM, Zhang F, Ross-Degnan D, Wharam JF. Opioid prescribing after nonfatal overdose and association with repeated overdose: A cohort study. Ann Intern Med 2016;164(1):1–9. doi: 10.7326/M15-0038.
27. Holliday S, Hayes C, Jones L, Gordon J, Harris N, Nicholas M. Prescribing wellness: Comprehensive pain management outside specialist services. Aust Prescr 2018;41(3):86–91. doi: 10.18773/austprescr.2018.023.
28. Brain K, Burrows T, Rollo ME, Hayes C, Hodson FJ, Collins CE. Population characteristics in a tertiary pain service cohort experiencing chronic non-cancer pain: weight status, comorbidities, and patient goals. Healthcare 2017;5(2):28. doi: 10.3390/healthcare5020028.
29. Fleck AK, Schuppan D, Wiendl H, Klotz L. Gut-CNS-axis as possibility to modulate inflammatory disease activity-implications for multiple sclerosis. International journal of molecular sciences. Int J Mol Sci 2017;18(7):E1526. doi: 10.3390/ijms18071526.
30. Sullivan MD, Turner JA, DiLodovico C, D'Appollonio A, Stephens K, Chan YF. Prescription opioid taper support for outpatients with chronic pain: A randomized controlled trial. J Pain 2017;18(3):308–18. doi: 10.1016/j.jpain.2016.11.003.
31. McPherson S, Smith CL, Dobscha SK, Morasco BJ, Demidenko MI, Meath TH, Lovejoy TI. Changes in pain intensity following discontinuation of long-term opioid therapy for chronic non-cancer pain. Pain 2018. doi: 10.1097/j.pain.0000000000001315. [Epub ahead of print].

ACTIVITY ID **139636**

Musculoskeletal problems

This unit of *check* is approved for six Category 2 points in the RACGP QI&CPD program. The expected time to complete this activity is three hours and consists of:

- reading and completing the questions for each case study
  - you can do this on hard copy or by logging on to the *gplearning* website, http://gplearning.racgp.org.au
- answering the following multiple choice questions (MCQs) by logging on to the *gplearning* website, http://gplearning.racgp.org.au
  - you must score ≥80% before you can mark the activity as 'Complete'
- completing the online evaluation form.

You can only qualify for QI&CPD points by completing the MCQs online; we cannot process hard copy answers.

If you have any technical issues accessing this activity online, please contact the *gplearning* helpdesk on 1800 284 789.

If you are not an RACGP member and would like to access the check program, please contact the *gplearning* helpdesk on 1800 284 789 to purchase access to the program.

Case 1 – Lina

Lina, 41 years of age, presents with pain in her right heel. She says the pain started after a high-impact aerobics session at the gym. Lina says she has been trying to lose weight, so she joined the local gym – her current weight is 75 kg and height is 166 cm. The pain is localised to the heel.

Question 1

Which of the following is the most likely diagnosis?

A. Inflammatory arthropathy

B. Sacroiliac nerve entrapment

C. Rheumatoid arthritis

D. Plantar fasciitis

Question 2

In plantar fasciitis, heel pain typically:

A. worsens with movement

B. subsides after rest

C. occurs with dorsiflexion of the first toe

D. is all of the above.

Further information

Lina's history and examination findings confirm a diagnosis of plantar fasciitis.

Question 3

Conservative treatment of plantar fasciitis includes:

A. exercises to stretch the fascia

B. use of night splints

C. low-dye taping

D. use of orthoses.

Case 2 – Leon

Leon is 70 years of age and is one of your regular patients. He has been seeing you recently for ongoing pain in his knees, which you suspect is due to osteoarthritis.

Question 4

Which of the following physical examination findings is not characteristic of osteoarthritis?

A. Redness, warmth and swelling

B. Crepitus

C. Reduced range of motion

D. Wasting of muscles around the joint

Question 5

Which of the following would you consider as a first-line investigation for osteoarthritis?

A. Ultrasonography

B. Plain anteroposterior X-ray

C. Computed tomography

D. Magnetic resonance imaging

Question 6

Which of the following would you advise as first-line management of Leon's osteoarthritis?

A. Non-steroidal anti-inflammatory drugs

B. Intra-articular corticosteroids injections

C. Acupuncture

D. Advice about exercise and weight loss

Case 3 – Belinda

Belinda, 65 years of age, presents after falling earlier in the day and injuring her left wrist. She had been having brunch with some friends at a local café, but when leaving she had

fallen, not noticing a low step. Plain X-rays show a fracture in the wrist. Belinda has seen you previously for low back and hip pain. She does not have any other medical conditions. She has a body mass index (BMI) of 26 kg/m^2, walks for at least 30 minutes a day for exercise, does not smoke and is a vegan. You organise for Belinda's wrist to be plastered and for a follow-up appointment to check the plaster. Given Belinda's fall and her history, you tell Belinda that after she recovers, you would like to do some additional investigations to check her bone mineral density.

Question 7

Belinda would qualify for dual energy X-ray absorptiometry (DXA) funded through the Medicare Benefits Schedule (MBS) because:

A. she has a BMI in the overweight range

B. she is a vegan

C. she has had a minimal trauma fracture

D. she is 65 years of age and has never had her bone mineral density assessed.

Further information

After Belinda recovers and the plaster is removed, you refer her for a DXA scan and baseline blood tests. The DXA scan shows a T-score of –2.0.

Question 8

Belinda's result indicates that she has:

A. a normal bone mineral density

B. osteopaenia

C. mild osteoporosis

D. severe osteoporosis.

Question 9

What lifestyle modifications would you advise to reduce Belinda's fracture risk?

A. Add strength training exercises

B. Consider calcium supplementation if her current intake is <1000 mg per day

C. Maintain a healthy weight

D. All of the above

Case 4 – Byron

Byron, 42 years of age, has had chronic back pain for the past five years for which he has been seeing you. You organised investigations and referrals to specialists, but to date, no specific cause has been identified. Initially, you prescribed paracetamol plus codeine for the pain, but this did not provide adequate pain management, so you started a trial of tramadol. In spite of your intention for this to be a short-term measure, and advice to Byron that ongoing opioid therapy is not recommended, he has frequently requested repeat scripts. He says that the pain interferes with his work and sleep, which in turn is causing him to be depressed.

Question 10

What can you do to better manage Byron's chronic pain?

A. Switch to a low-dose fentanyl patch

B. Prescribe a benzodiazepine to help with his sleep difficulties

C. Advise holistic care by referring him to a multidisciplinary pain clinic or facilitating active self-management

D. All of the above

See you on the Gold Coast

GP18 General practice: The centre of health in Australia

GOLD COAST 11–13 OCTOBER 2018

GP18 – Your platform to engage

GP18 is your place to discover, engage and learn. There are more interactive hands-on skill development sessions than ever before in one place over four days.

With 14 ALMs, 14 clinical sessions and over 13 hours of research-specific content, this conference has something for everyone, for active and passive learners alike.

Are you a working parent? Why not take advantage of the on-site crèche facilities.

Show your support for World Obesity Day on Thursday 11 October 2018 and join your fellow conference peers on a 'walk against obesity'.

To register and view the program, visit **gp18.com.au**

THE GOLD COAST CONVENTION AND EXHIBITION CENTRE

Join the **#GP18gc** conversation

RACGP
Royal Australian College *of* General Practitioners

5890