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SPECIALIST ORTHOPAEDIC SURGEON

PATIENT INFORMATION — IN DEPTH

# Musculoskeletal Syndrome of Menopause

Falling oestrogen through menopause drives a recognised cluster of joint, tendon and bone problems.

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## What you're feeling

You are likely experiencing joint pain, or arthralgia, more than half of women do around the time of menopause. This pain often increases as your body transitions through menopause. The drop in oestrogen levels is a key driver of this change. You may notice that your muscles feel stiffer. Estrogen and muscle stiffness are negatively related, meaning less estrogen often means tighter, less flexible muscles.

The pain can show up in many places. Low back pain is more common in postmenopausal women than in men of the same age. This is linked to physiological changes from lower sex hormone levels. You might also feel pain in your shoulders. The risk of silent rotator cuff tears increases during this period. These are tears you may not feel until they affect your movement.

Daily tasks can become difficult. Reaching behind your back or over your head may feel awkward and stiff. You may find it harder to lift objects or sleep on your side. Your doctor knows that these changes are real and common. They are not just in your head.

Your symptoms may flare at night or after activity. Waking up with stiffness is also common. While hormone levels fluctuate during your cycle, these changes do not significantly affect knee looseness. However, they can influence how stiff your muscles and tendons feel. This variability can make some days better than others.

It is important to know that you are not alone in this. The prevalence of muscle loss, or sarcopenia, is 31% in postmenopausal women. This loss contributes to the pain and stiffness you feel. While some treatments show promise, the evidence for hormone therapy protecting against joint replacement is limited. Your doctor will focus on managing your specific symptoms and improving your quality of life.

**CQ HAND + UPPER LIMB**
Dr Kieran Hirpara — Specialist Orthopaedic Surgeon
Suite 2, Level 1, Mater Private Hospital Rockhampton, 31 Ward Street, The Range, QLD 4700
Phone 07 4863 6556 · office@cqupperlimb.com.au · cqupperlimb.com.au

## What's actually happening

During the perimenopause, your body undergoes significant hormonal shifts that make you particularly prone to musculoskeletal pain. More than half of women experience joint pain, or arthralgia, around this time. This discomfort often increases as you move through the menopausal transition. The primary driver is a reduction in oestrogen levels. This drop affects not just your joints, but also your muscles and bones.

Your joints rely on smooth cartilage to act as a shock absorber between bone ends. Lower oestrogen levels are linked to the development of knee osteoarthritis, which is wear-and-tear arthritis. In some cases, this combines with osteoporotic osteoarthritis, where the bone beneath the cartilage becomes less dense due to high remodeling. This structural change can lead to stiffness and pain. Low back pain is also more common in postmenopausal women than in men of the same age, driven by these same physiological changes in sex hormones.

Your muscles and tendons are also affected. Muscle stiffness has a negative relationship with estrogen, meaning lower levels lead to tighter, less flexible tissue. In fact, estrogen and muscle properties are closely correlated. This hormonal shift can contribute to sarcopenia, a loss of muscle mass. In one study of postmenopausal women, the prevalence of sarcopenia was 31%. Additionally, the tendons in your shoulder, such as the supraspinatus, contain receptors for estrogen and progesterone. When these hormone levels drop, the tendon fibres may weaken. This increases the risk of asymptomatic full-thickness rotator cuff tears, where the tendon fibres separate completely. These tears are more prevalent in the postmenopausal period and are associated with metabolic disorders.

Your shoulder joint is surrounded by a capsule, which is a sleeve of tissue that holds the joint together. Lower estrogen levels may also increase your odds of developing adhesive capsulitis, or frozen shoulder, where this capsule becomes thick and tight. While hormone replacement therapy may offer some protection against these conditions, the evidence varies. Ultimately, the pain and stiffness you feel are direct results of your body's changing hormonal landscape affecting the tissues that support your movement.

## What we can do about it

Musculoskeletal pain is particularly prevalent during the perimenopausal transition. Arthralgia affects more than half of women around the time of menopause. This rise is linked to reduced estrogen levels. You can start with self-care and physical therapy. Gentle exercise helps maintain mobility and strength. For thumb joint pain, combining proprioceptive neuromuscular facilitation exercises with strength training works better than strength training alone. This approach reduces disability and improves movement. If you have greater trochanteric pain syndrome and a body mass index under 25, combining hormone therapy with any exercise and education is superior to placebo. Give these conservative measures time to work. Consistency is key to seeing results in your daily comfort.

Medical options include pain medication and anti-inflammatories. Hormone therapy is another path, but it has trade-offs. Estrogen and selective estrogen receptor modulators may help postmenopausal patients with early-stage wear-and-tear arthritis or osteoporotic arthritis. However, long-lasting use of systemic menopausal

hormone therapy, particularly estrogen-only therapy, is associated with a greater risk of chronic low back pain. Unopposed estrogen shows a protective trend for incident radiological wear-and-tear arthritis of the knee. Yet, there is limited evidence for a protective effect of unopposed estrogen use on the incidence of hip or joint replacement. Estradiol supplementation may lead to better outcomes after rotator cuff repair, though differences in pain and function scores did not meet the minimal clinically important threshold. Discuss these benefits and risks with your doctor to find the right balance for you.

If symptoms remain severe despite these steps, seek specialist input. Your doctor may refer you for a deeper assessment. Imaging can check for issues like asymptomatic full-thickness rotator cuff tears, which are more common in the postmenopausal period. For some specific conditions, a procedure may occasionally be considered if conservative care fails. This decision depends on your individual health profile and the severity of your pain. Your doctor will guide you through the next steps based on what works best for your body and your lifestyle.

## What to expect

Musculoskeletal pain is common during the perimenopausal transition. More than half of women experience joint pain (arthralgia) around the time of menopause. This number increases as your estrogen levels drop. You may notice that low back pain is more prevalent in postmenopausal women than in men of the same age. This is linked to lower sex hormone levels after menopause.

Your outlook depends on how you manage these changes. For some conditions, such as early-stage wear-and-tear arthritis, estrogen or specific estrogen-modulating medications may be favourable. These treatments can help if you have reduced bone density. However, long-lasting use of systemic hormone therapy, particularly estrogen-only therapy, is associated with a greater risk of chronic low back pain. There is limited evidence that unopposed estrogen prevents the need for hip or joint replacement. It does show a protective trend for knee arthritis on X-rays.

If you have rotator cuff tears, they are more common in the postmenopausal period and linked to metabolic disorders. Estradiol supplementation may lead to better outcomes after rotator cuff repair, though the difference in pain scores might not feel significant to you. For thumb arthritis, combining specific stretching exercises with strength training is more effective than strength training alone. It reduces disability and improves mobility.

If you have greater trochanteric pain syndrome and a healthy weight (BMI <25), combining hormone therapy with exercise and education works better than placebo. Without treatment, symptoms like sarcopenia (muscle loss) affect 31% of community-dwelling postmenopausal women. This can impact your physical activity.

Overall, your doctor will tailor a plan to your specific symptoms. Some pain may settle with targeted therapy. Other symptoms, like chronic low back pain from long-term hormone use, may persist. Be honest about your pain levels. Realistic expectations help you manage the transition. Your body is changing, but you have options to stay active and comfortable.

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## When to see someone

The perimenopause is a state during which women are particularly predisposed to develop musculoskeletal pain. Arthralgia is experienced by more than half of women around the time of menopause. The prevalence of arthralgia appears to increase during the menopausal transition. Low back pain is more prevalent in postmenopausal women than age-matched men. See your GP if you have persistent pain not improving with rest, weakness or instability, locking or giving way, symptoms interfering with sleep or work, or sudden worsening. Long-lasting use of systemic menopausal hormone therapy is associated with a greater risk of chronic low back pain. Estradiol supplementation was associated with better outcomes in postmenopausal women undergoing rotator cuff repair.

## In more depth

This section goes further than you need for your own treatment decisions. It is written for the reader who wants the evidence itself, and it is worth the extra effort here because the obvious inference — that if falling oestrogen damages musculoskeletal tissue, then replacing the oestrogen should repair it — is not what the literature shows.

The cluster of symptoms now has a name. In 2024 it was formally described as the musculoskeletal syndrome of menopause, grouping arthralgia, loss of muscle mass, loss of bone density and progression of osteoarthritis under a single cause: the precipitous fall in oestrogen across the transition [1]. Around **47 million** women enter that transition each year. More than **70%** will develop musculoskeletal symptoms and **25%** will be disabled by them [1].

Two numbers from that paper deserve emphasis. The overall prevalence of musculoskeletal pain in perimenopausal women is approximately **71%** — and **40%** of women with these symptoms have **no structural findings** on imaging [1]. A normal scan does not mean nothing is wrong; it is the expected result in a large minority.

The authors group the syndrome into five processes, which is a more useful frame than a list of complaints: inflammation (arthralgia, joint pain, frozen shoulder), sarcopenia (poor balance, falls, loss of stamina), reduced satellite-cell proliferation (muscle that will not rebuild), osteoporosis (height loss, stooped posture, low-impact fracture) and arthritis (joint pain and stiffness) [1]. Naming it matters because these have historically been assessed one joint at a time, by different clinicians, none of whom saw the pattern.

### WHAT OESTROGEN IS ACTUALLY DOING IN THE TISSUE

Oestrogen receptors are present in tendon, muscle, cartilage and synovium, so the withdrawal of oestrogen is not a bone-only event. But the effect on tendon is more specific than a general decline.

When ageing and oestrogen deficiency were compared directly in rotator cuff tendon, ageing reduced expression of **both** major collagens and of elastin, while oestrogen deficiency selectively reduced **elastin alone**, leaving the collagen genes unaffected [2]. Collagen is the rope; elastin is the recoil. That fits what people describe — not a tendon that has become weak so much as one that has become stiff and less forgiving.

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Three further mechanisms are consistently proposed for the joint pain itself: oestrogen normally suppresses inflammatory cytokine production, so its withdrawal permits a low-grade inflammatory state; oestrogen has a direct antinociceptive effect, so losing it lowers the pain threshold independently of any tissue damage; and accelerated bone turnover contributes its own discomfort [3].

## THE CLEAREST NATURAL EXPERIMENT

The strongest causal evidence does not come from menopause research at all. Aromatase inhibitors, used in breast cancer, produce profound and deliberate oestrogen deprivation, and the musculoskeletal consequences have been measured carefully.

Across 21 studies and **13,177** women, the pooled prevalence of aromatase inhibitor-induced arthralgia was **45.9%** [4]. Around **9.3%** of women stop otherwise effective cancer treatment because of it [4]. Importantly, the arthralgia is predominantly **non-inflammatory** — the joints hurt without the markers of an inflammatory arthritis [3].

This is as close to an experiment as this field gets. Remove oestrogen abruptly and roughly half of women develop joint pain; the pain is real, common, and not explained by inflammatory disease. The parallel is drawn explicitly in the defining paper, which notes that increased arthralgia is reported both after aromatase inhibitors and after **sudden withdrawal of menopausal hormone therapy** [1] — two different routes to the same abrupt loss, producing the same complaint. It is the best reason to take the menopausal version seriously rather than treating it as coincidental ageing.

## THE CONDITIONS THAT CLUSTER AROUND IT

Shoulder pain is measurably more common after menopause. In a cross-sectional study of **21,095** people, shoulder pain was more prevalent in menopausal than non-menopausal women, with sleep duration and inflammatory markers among the associated factors [5].

Frozen shoulder is placed squarely inside the syndrome by the defining paper, which classifies it under the inflammatory arm alongside arthralgia and joint pain, and advises that a midlife woman presenting with adhesive capsulitis should prompt consideration of the syndrome as a whole rather than treatment of the shoulder in isolation [1].

It is worth being straight about the strength of that link. The independently replicated risk factors for adhesive capsulitis are diabetes, thyroid disease and hyperlipidaemia, and a genome-wide association study found a genetic contribution comparable in magnitude to the diabetes and hypothyroidism associations, implicating the Wnt pathway [6]. Menopause is not among those well-replicated factors. So the mechanism proposed — oestrogen withdrawal permitting an inflammatory state — is plausible and coherent with the arthralgia data, but the frozen-shoulder link rests at present on that reasoning rather than on epidemiology of the same weight as the diabetes association.

There is also an overlap with widespread pain that is worth naming, because it cuts both ways. Fibromyalgia symptoms often begin around menopause, tend to be more severe afterwards, and can worsen following hysterectomy with or without removal of the ovaries [17]. The symptom lists of the two conditions overlap heavily. But fibromyalgia also occurs in women who are not menopausal and in men, so sex hormones cannot be

the whole mechanism, and the review's conclusion is that the two should be recognised and treated as separate problems rather than collapsed into one [17]. Practically, that means widespread pain in a midlife woman should not be attributed to menopause by default — see our fibromyalgia page.

## DOES REPLACING THE OESTROGEN HELP?

This is where intuition fails hardest, and the answer differs depending on what you measure.

For tendon, the signal points the wrong way. In a propensity score-matched national cohort of **127,672** women aged 45 and over, systemic hormone replacement therapy was associated with an **increased** rate of tendon injury and a greater likelihood of proceeding to surgical repair [7]. In a separate prospective cohort of **6,007** women, long-lasting systemic therapy — oestrogen-only formulations in particular — carried a greater risk of chronic low back pain [8].

Given directly around surgery, the benefit is real but small. Among **184** postmenopausal women having rotator cuff repair, oestradiol supplementation produced better pain and shoulder-value scores, but the differences **did not reach the threshold at which a patient would notice them** [9]. Work on delivering oestradiol locally to the repair site rather than systemically has so far reached proof-of-concept in animals [10].

These are observational studies, and women who take hormone therapy differ systematically from those who do not. **None of this is a reason to stop hormone therapy**, which is prescribed for compelling reasons unrelated to tendons. What it does mean is that hormone therapy should not be expected to protect your tendons, and that new shoulder or elbow pain after starting it deserves assessment rather than the assumption that it must be unrelated.

## AROUND SURGERY THE PICTURE SPLITS

The perioperative literature is genuinely contradictory, and the contradiction is informative: medical complications and implant complications move in opposite directions.

On the medical side, oestrogen replacement looks protective. In a cohort of **2,554,672** patients undergoing total joint arthroplasty, oestrogen replacement therapy was associated with **decreased** venous thromboembolism and fewer major medical complications [11]. In **21,220** women having total hip arthroplasty, hormone therapy before surgery was associated with fewer periprosthetic fractures at ten years and no increase in thromboembolism [12].

On the implant side the direction reverses. In a propensity-matched series of **3,558** patients undergoing total shoulder arthroplasty — the upper-limb operation most relevant here — oestrogen replacement was associated with a long-term **increase** in revision procedures and mechanical complications [13]. Another series found higher rates of revision and periprosthetic joint infection in patients on perioperative oestrogen replacement [14].

The practical conclusion drawn by a systematic review is narrower than either extreme: non-oestrogen-containing transdermal preparations should **not** be withheld before elective joint arthroplasty, while the case for withholding oestrogen-containing forms remains unproven either way [15]. If you are on hormone therapy and surgery is planned, this is a decision to make jointly with your surgeon and your prescriber, not unilaterally.

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## WHAT IS ACTUALLY KNOWN TO HELP

The most useful evidence is the least glamorous. In a randomised trial of postmenopausal women with thumb base arthritis, adding proprioceptive neuromuscular facilitation — guided movement patterns that train coordination rather than raw strength — to a strength programme beat strength training alone across pain, disability, mobility and grip [16].

That is the shape of the evidence overall. Targeted, supervised exercise earns its place. Hormonal explanations are better at accounting for why the tissue changed than at telling us what to do once it has.

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