Osteoarthritis Flares

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**SYNOPSIS**

The phenomenon of flares is a common feature in the daily life of people with osteoarthritis (OA). Characterized by episodes of sudden-onset increases in signs and symptoms, their impact can often be distressing and disabling. Despite their potential to have both short and long-term consequences for patients across the whole course of the condition their occurrence and optimal management is not fully understood. This article provides a contemporary perspective on defining OA flares and their potential triggers, and offers suggestions for how health professionals might explore flare patterns with patients in clinical practice and frame timely best-practice treatment approaches.

**KEYWORDS**

Osteoarthritis; Flare; Pain; Acute; Exacerbation; Symptom variability; Management

**KEY POINTS**

* Osteoarthritis (OA) flares are episodic sudden-onset increases in signs and symptoms that can be distressing and disabling.
* The triggers and impact of OA flares vary between-people and within-people across the disease course.
* All healthcare professionals can help patients recognize and understand their own flare behaviours.
* Viewing and managing OA as an ‘acute-on-chronic’ condition, with a practical distinction between short-term ’fast’ flare management strategies and long-term ’slower’ management strategies may be beneficial.

**INTRODUCTION**

Osteoarthritis (OA) is a leading cause of long-term disability worldwide1 and places a significant burden on individuals, healthcare providers and the wider economy.2 Multifactorial in onset3 and heterogeneous in course e.g.,4 the OA disease process can affect the whole joint complex.5 Clinical interventions for OA prioritize personalized symptom management, with a range of strategies designed to minimize distress and disability.6

OA-related pain is best understood through a biopsychosocial perspective7 and patients’ active engagement with available treatments and interventions is central to their efficacy. For example, the known benefits of exercise and weight control for prevention and management are largely achieved and maintained by motivated lifestyle adherence, with the support of healthcare professionals.

The inherent variability in OA prognosis is mirrored in daily life, which is commonly experienced as periods of relative comfort punctuated by episodic flares (exacerbations) often of unpredictable frequency, intensity and duration. These can be physically and psychologically distressing and disrupt self-management routines.8 In the community, between 23-32% of people with knee OA experience significant pain variability,9 and average flare episode frequency of 2.4 per year among people with hip, knee or hand OA,10 illustrating the scale of the problem, which can also impact work productivity.11

Historically, physical, behavioral and mind-body flare management has only received sporadic research attention compared to pharmacological management. Previously there has been a longstanding focus on flare-design trials, whereby flare episodes are induced by suspending current treatment before testing new pharmacological treatment effectiveness (e.g., non-steroidal anti-inflammatory drugs [NSAIDs]). These studies appear to produce comparable findings to non-flare trial designs12, but cannot be presumed to characterize ‘naturally occurring’ flares.13 More recently empirical studies are emerging internationally, as are coordinated efforts to address key issues in the area of ‘naturally occurring’ flares with an emphasis on improving patient care.

In this article, we undertake a critical narrative synthesis of evidence on recognizing and defining OA flares, present conceptual ideas for exploring their behavior in clinical practice, provide an overview of best-practice treatment approaches and offer suggestions on the timing of self-management strategies.

**RECOGNIZING AND DEFINING OA FLARES**

A challenge for both patient education and the research endeavor is recognizing and defining what is meant by a flare. From the patient perspective, flares are recognized as discrete episodes of increased pain, often with rapid onset, that can impact daily activities.14 Despite researcher efforts to develop diagnostic criteria15 and formal flare assessment,16 a wide variety of flare definitions exist within the literature.13,17 Spontaneous or ‘naturally occurring’ flares should be considered distinct from induced flares in drug withdrawal trials,13 and the cardinal defining feature of flares has been pain.17

Recent observational studies have predominantly defined flares as a 0-10 numerical rating scale change score of ≥2 from baseline ‘usual’ pain18-21 versus participant self-report.22 Flares have been perceived as transient events that can occur multiple times a day23 or viewed as distinct episodes, or changes in state, that must endure for pre-specified periods of time beyond usual within-day/between-day pain fluctuations (e.g., ≥8 hours,18,20 ≥24 hours22, 48 hours19). Distinction between a flare versus worsening of symptoms into a persistent, progressive state can be unclear. Clinically, flares are discrete sudden-onset episodes that resolve. Worsening that persists (i.e., deteriorating state as opposed to a short term fluctuation in disease activity) would therefore not be considered a flare, but this may be difficult to distinguish in the early stages.

A growing consensus that flares are more than just pain is reflected in a position statement reached by an international community of clinicians and patients recently proposing and further endorsing five key domains, that represent worsening from peoples ‘usual’ state, and endure for a few days24,25:

* Pain
* Swelling
* Stiffness
* Psychological aspects (e.g., low mood, irritation)
* Impact of symptoms (e.g., sleep/activity changes, function)

Flare experiences themselves are heterogeneous and likely to exist on a continuum. For example, there may be circumstances where people experiencing flares have no swelling15 or no perceived distress. Despite this, there is some evidence that patient and physician flare recognition correlates well15 and imposing a dichotomized definition of flare informed by available evidence follows the convention across many conditions. Clinical definitions (diagnoses) are most helpful if they have prognostic significance or inform treatment selection.26

Capturing flares as they occur often presents a significant challenge and a valid patient reported outcome measure has recently been developed.27 The FLARE-OA questionnaire will offer the ability to adopt a more standardized approach for identifying flare occurrence and reoccurrence to better inform planned interventions.

Furthermore, people often consult health services with flares not just because they are disruptive but because of concerns about what they mean and whether they might signal something serious in underlying pathological change. These may or may not relate to OA. A challenge when recognizing and defining flares is judging and excluding rare but serious underlying causes28 (e.g., insufficiency fracture, malignancy or infection).

**TRIGGERS FOR OA FLARES**

The underlying susceptibility to flares is likely to include distal causes (e.g., possibly obesity) and proximal triggers (e.g., unaccustomed activity). Bracken’s ‘Cone of causation’ simultaneously acknowledges causal contributors, induction periods between risk factors in a causal chain, and time.29 The cone shape reflects greater number of earlier distal factors than proximal factors in causation.29 Although little is known about distal causes that increase susceptibility to flares, they might reasonably look similar to those associated with OA incidence and progression. Figure 1 outlines a conceptual model of causality recognizing the potential role of both distal and proximal causes of OA flares. Although the relative contribution, number and timing of distal and proximal causes is unknown, this may represent a useful framework to conceptualize OA flares. The remainder of this section focuses on synthesizing evidence from studies on proximal triggers.

**[Figure 1]**

The episodic nature of flares means that exposures (triggers) are commonly time-varying events with short induction times. Investigating flare triggers in daily life is challenging. Self-controlled case-crossover study methodology30 combined with mechanisms for ‘real-time’ data capture, such as web-based platforms, have facilitated empirical observation of flare triggers in community settings. This design enables study participants to provide data at the time of a flare and the days leading up to it, and compares these observations with equivalent data collected during non-flare periods. Questions in this context are not concerned with why people experience flares (“Why me?”), but aim to examine what might trigger flares (“Why now?”).31

Table 1 summarises potential trigger exposures examined for hip/knee OA32, knee OA19,21,22,33-35 and hip OA.20,36-39 Broadly, positive proximate trigger exposures for flare onset fall into categories of activity-related, psychosocial and environmental factors. However, the exact mechanisms by which exposures precipitate flares and their relationship to each other remains unclear. Collectively across hip and knee studies, triggers with supportive evidence from one or more other study are activity-related factors including moderate physical activity,21,22 squatting or kneeling, lifting or moving heavy objects, climbing ladders,19,22 joint buckling/giving way;20,22,34 psychosocial factors including worse mental health,32 higher negative affect and passive coping strategies,33 low mood/depression, feeling angry, irritable or hostile22 and poor sleep22,36; and environmental factors including weather temperature changes.22,39 Contrasting observations across studies in terms of direction and magnitude of associations might be explained by differences in OA and flare definition, sample size, exposure measurement and observed induction times.

**Table 1.** Potential trigger exposures and association with osteoarthritis flares.

|  |  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- | --- |
| **Study** | **Design** | **Mode of data collection** | **OA definition** | **Flare definition** | **Sample size** | **Exposure-flare onset induction time** | **Potential trigger exposure and association with flare onset** |
| **Hip/knee** | | | | | | | |
| Wise et al. 32  United States | 12 week telephone-based case-crossover study | Retrospective,  self-reported | Physician clinically diagnosed hip or knee OA | WOMAC score in highest 30% of all WOMAC scores (yes/no) | 91 | 1 week prior | *Positive risk factor*  Worse mental health |
| **Knee** | | | | | | | |
| Efrani et al.33  Australia | 90 day Web-based case-crossover study | Retrospective,  self-reported | Meet ≥1 ACR criteria for knee OA40 and ≥2 KL41 tibiofemoral OA or patellofemoral OA42 on plain radiography | NRS (0-10) change score of ≥2 from baseline mildest knee pain, lasting >8 hours | 149 & 54 | Affect measures 10 days prior; pain coping/perceived stress 30 days prior | *Positive risk factors*  Higher negative affect; passive coping strategies.  *Protective factor*  Higher active coping strategies  *None*  Positive affect; all coping strategies; problem-focused coping; emotion-focused coping; pain catastrophizing; control over pain; ability to decrease pain; overall coping strategies effectiveness, perceived stress |
| Zobel et al.34  Australia | 90 day Web-based case-crossover study | Retrospective,  self-reported | Meet ≥1 ACR criteria for knee OA40 and ≥2 KL41 tibiofemoral OA or patellofemoral OA42 on plain radiography | NRS (0-10) change score of ≥2 from baseline mildest knee pain, lasting >8 hours | 157 | Knee injury 7 days prior; knee buckling 2 days prior | *Positive risk factors*  Knee injury; knee buckling |
| Ferriera et al.35  Australia | 90 day Web-based case-crossover study | Prospective,  objective | Meet ≥1 ACR criteria for knee OA40 and ≥2 KL41 tibiofemoral OA or patellofemoral OA42 on plain radiography | NRS (0-10) change score of ≥2 from baseline mildest knee pain, lasting >8 hours | 171  (404 hazards, 1021 control) | 72 hours prior | *None*  Temperature; relative humidity; barometric pressure; precipitation |
| Parry et al.19  England, UK | 3 month pen-paper daily diary study | Prospective,  self-reported | GP defined & Self-reported knee symptoms on ≥1 day in last 12 months | NRS (0-10) change score of ≥2 from baseline in last 24 hours, last ≥2 days | 67 | 48 hours prior | *Positive risk factors*  ≥1 selected physical activity (prolonged kneeling, lifting/moving heavy objects, climbing stairs, prolonged squatting, climbing ladders) |
| Atukorala et al.21  Sri Lanka | 3 month telephone-based case-crossover study | Retrospective,  self-reported | Meet ACR criteria for knee OA40 | NRS (0-10) change score of ≥2 from baseline usual knee pain, lasting ≥4 hours | 120 | Footwear use/being barefoot 2 days prior;  Physical activity 7 days prior | *Positive risk factors*  Moderate physical activity 1,2 and 3-7 days prior; using footwear 1 and 2 days before  *Protective factor*  Increased duration barefoot (>8 hours) 1 and 2 days prior |
| Thomas et al.22  England, UK | 13 week Web-based case-crossover study | Retrospective,  self-reported | GP defined/ self-reported | Self-reported sudden onset of worsening signs and symptoms sustained for ≥24 hours | 376  (568 hazards/ 867 controls) | 24 hours prior | *Positive risk factors*  Squatting or kneeling; standing for long periods without rest; lifting or moving heavy objects; going up and down ladders, moderate-to-vigorous physical activity; knee buckling; slip trip or fall; taking extra analgesia in anticipation of increased physical activity; poor night’s sleep; low mood/depression; feeling angry, irritable or hostile; cold/damp weather  *Protective factors*  Sitting for long periods without a break; missing or reducing planned medication; cough, cold or minor infection  *None*  Going up/down stairs; driving; stressful events at home, work and family-related stress |
| **Hip** | | | | | | | |
| Fu et al.20  Australia | 90 day Web-based case-crossover study | Retrospective,  self-reported | Meet ACR criteria for hip OA43 and ≥2 KL44 hip OA | NRS (0-10) change score of ≥2 from baseline mildest hip pain, lasting >8 hours | 133 | Hip injury 7 days prior; hip giving way 2 days prior | *Positive risk factors*  Hip injury; hip giving way |
| Fu et al.36  Australia | 90 day Web-based case-crossover study | Retrospective,  self-reported | Meet ACR criteria for hip OA43 and ≥2 KL44 hip OA | NRS (0-10) change score of ≥2 from baseline mildest hip pain, lasting >8 hours | 130 | 7 days prior | *Positive risk factors*  Poor sleep; increased fatigue |
| Fu et al.37  Australia | 90 day Web-based case-crossover study | Retrospective,  Self-reported | Meet ACR criteria for hip OA43 and ≥2 KL44 hip OA | NRS (0-10) change score of ≥2 from baseline mildest hip pain, lasting >8 hours | 137 | 24 hours prior | *Protective factors*  Shoes with heel height ≥2.5 cm; longer duration of heel height ≥2.5 cm |
| Fu et al.38  Australia | 90 day Web-based case-crossover study | Retrospective,  self-reported | Meet ACR criteria for hip OA43 and ≥2 KL44 hip OA | NRS (0-10) change score of ≥2 from baseline mildest hip pain, lasting >8 hours | 131 | 7 days prior | *Positive risk factors*  Pain catastrophizing; pain self-efficacy beliefs  *None*  Depression, anxiety and stress, positive and negative affect |
| Fu et al.39  Australia | 90 day Web-based case-crossover study | Prospective,  objective | Meet ACR criteria for hip OA43 and ≥2 KL44 hip OA | NRS (0-10) change score of ≥2 from baseline mildest hip pain, lasting >8 hours | 129 | 72 hours prior | *Positive risk factors*  Temperature variation  *None*  Maximum/minimum temperature; relative humidity; precipitation; barometric pressure |

OA, Osteoarthritis; WOMAC, Western Ontario and McMaster Universities Osteoarthritis Index; ACR, American College of Rheumatology; KL, Kellgren and Lawrence; NRS, Numerical Rating Scale.

A strength of within-person case-crossover methodology is that person-level and slow-varying potential confounders are controlled by design. A potential weakness of how most OA case-crossover studies have typically been designed is with retrospective exposure measurement. Consequently, there may be differential recall between hazard and control periods leading to some inherent bias. With the exception of objective weather analyses,35,39 only one previous case-crossover analysis employed prospective self-reported daily measurement19 and the advantages of intensive 3-month daily diary study over periodic measurement must be considered in terms of acceptable participant burden.

A common patient belief is that weather changes can trigger flares. A 15-month prospective study of 2658 UK-based participants with several long-term pain conditions observed that relative humidity, pressure and wind speed had a modest significant relationship with pain.45 Further, in a subgroup analysis limited to those with OA, OA pain remained positively associated with relative humidity. This observation contrasts three OA flare studies.22,35,39 However, these study samples were smaller and Thomas et al’s.22 retrospective exposure measurement were vulnerable to recall bias. Dixon et al. 45 also found a positive association between pain events and low mood, but not high physical activity. Contrasting physical activity associations with those in Table 1 may be explained by recall bias, smaller sample sizes and differences in exposure measurement.

Currently, the literature appears to indicate that flare triggering effects of weather are likely modest. In general, activity-related and psychosocial triggers have been observed more consistently and are likely to be more amenable to modification. Further investigation to understand flare triggers and their relationship with OA pathophysiology is warranted.

**OA FLARE PATTERNS AND SYMPTOM VARIABILITY**

Flares appear to occur from the earliest phases of OA46 but the predictability of episodic pain appears to diminish in later phases, when their impact can also become more distressing46 and unacceptable.47

In terms of predicting flares in people with knee OA, Atukorala et al.48 suggested that baseline risk factors including increasing age, years lived with OA, body mass index, background/worse levels of pain, knee injury, buckling, intermittent and constant pain score and footwear type/heel height predicted a pain flare within 30 days. In adults with, or at risk of, knee OA, Thomas et al.22 reported that flares appeared to be slightly more common among adults of working age, women and people with more frequent pain.

Preventing or reducing flares by understanding and anticipating daily life circumstances that might trigger onset, and modifying these if possible, provides an opportunity for people to develop personalized flare prevention strategies. Focussed consultation support to help patients understand their own flare patterns and recognize potential triggers would appear beneficial, particularly as a recent study found that in adults with, or at risk of, knee OA, 70% of reported flares were unexpected.22

Studies that capture flare episode duration are broadly comparable, with flares typically lasting around 3-8 days.19,22,49 For patients, flares of unfamiliar impact (e.g., resulting in time off work) or that last longer than usual should perhaps signal a need to seek additional healthcare. Potential relationships between different triggers and flare duration are unknown and likely to vary across the disease course.

In the short-term, flares can be distressing and disrupt daily life. Whether flares have implications for long-term prognosis remains unclear. It has previously been proposed that the theoretical natural history could chart a course where intermittent and benign flare episodes may cumulate over time and occur with increased frequency, duration and intensity, eventually leading to consequences that culminate in joint failure50 (Figure 2). Future empirical demonstration of the relationship between potential flare triggers, episode duration and flare frequency may help to establish the potential longer-term consequences of flares and identify people most at risk of poor outcomes earlier in the disease course. This could potentially help steer people onto more favourable trajectories.49 Flare patterns are likely to be heterogeneous, both between-people and within-people over time.

**[Figure 2]**

Symptom variability (within-day and between-day) may also be an important related yet distinct phenomenon. For example, in other conditions such as cardiovascular disease, blood pressure variability is a strong predictor of stroke.51 Although prospective studies examining short-term symptom variability exist within the OA literature e.g.,23,52-54 extending the application of intensive longitudinal designs55 to studies of OA symptom fluctuations may offer new perspectives on clinically relevant short-term changes and their impact on longer-term outcomes. Furthermore, attempting to reduce symptom variability itself may be a worthy treatment target56 that might also have utility as part of self-monitoring or behaviour change strategies in clinical settings.57

**ACUTE FLARE MANAGEMENT AND LONG-TERM OA MANAGEMENT**

***Access to care***

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OA is predominantly managed in primary care settings by general practitioners/physicians and more recently other health professionals, such as physiotherapists.58,59 Flare episodes and consultations do not always align well, but when they do, they represent opportunistic encounters to share key messages and advice60 at times when people may be most receptive to change behaviour. Whilst access to care varies61, adequate knowledge and understanding of OA as a condition, that gives rise to appropriate acute flare and long-term condition management, are skills that all first contact clinicians should equip themselves with to positively frame patient care and provide consistent messages. A recent qualitative literature synthesis promotes the notion of participatory discourse where positive communication focuses on what patients can, rather than cannot, do to facilitate physical activity engagement.62

Consequences of poor flare management, particularly early in the disease course, may be escalating treatment and unnecessary healthcare utilization. For example, the prescription of stronger opioid-based medication, Magnetic Resonance Imaging or referral for surgical opinion may occur earlier than desirable in response to poorly managed flares. This could be detrimental to patient self-management behaviours and self-efficacy, leading to poor outcomes. The potential role of pharmacists to support timely flare management advice and education in community settings could also be better utilized.

***Physical, behavioural and lifestyle interventions for acute OA flare management***

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In terms of evidence-based practice, a recent scoping review concluded that robust evidence for a range of OA flare management strategies (behavioural, lifestyle, and adjunctive treatments) is lacking.63 Although two studies examining retro-walking64 (walking backwards) and modified ‘rescue’ exercise65 during a flare of knee OA found tentative evidence of safety and pain improvement, the clinical meaningfulness of these physical activity/exercise outcomes was unclear.63 Furthermore, the predominant focus on knee pain means broader inclusion of other joint sites (e.g., hip or hand) and other flare symptoms (e.g., stiffness, swelling, or psychological distress) are lacking.63 Although flares often co-occur with increased or unaccustomed physical activity, and appear to relate to movement mechanics,66,67 exercise-induced flares are likely to decrease with graded exposure to exercise68 and patient-focused physical activity promotion. For example, a recent study concluded that knee pain flares were associated with recent knee injury, more severe radiographic OA and lower quadriceps strength.69 Factoring such presentations into treatment planning may provide more targeted care.69

***Pharmacological management of acute OA flares***

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Pharmacological management options for OA broadly involve topical NSAIDs applied directly to affected joints, oral medications (e.g., paracetamol, NSAIDs) and intra-articular steroid injections for moderate-to-severe pain e.g.,70-73. Pharmacological efficacy is often complicated by a lack of data relating to patient usage habits.70 Data observing oral NSAIDs use for acute knee pain flares identified favourable recovery trajectories within 3-5 days, with pain levels, activity interference, stiffness and swelling following a similar course.49 Recent meta-analyzed data from knee OA clinical trials concluded pain and function improvements from NSAIDs use peaking at 2 weeks.74 Due to heterogeneity of pharmacological efficacy and side effect profiles the lowest effective dose for the shortest possible duration is advocated70-73 and patients should be encouraged to regularly rationalize medication use in the context of comorbidities via shared decision-making with healthcare professionals.70-72

***Managing OA as an ‘acute-on-chronic’ condition***

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Lack of evidence to underpin flare management recommendations, alongside chronic disease management, in current international clinical guidelines highlights further research opportunities. Better understanding flare mechanisms,75 their potential role in OA etiopathogenesis, and their response to treatment could lead to new insights for clinical intervention. The notion that complementary chronic disease and acute pain care models could help OA management is not new.76 However, the increased emphasis of OA as an ’acute-on-chronic’77 condition could support strategies designed to manage, i) the underlying (chronic) disease course, ii) (acute) flare episodes and iii) behaviour modification to potentially avoid or minimize exposure to flare triggers.50 Patient management in the context of these three inter-related components could guide personalized care.

**Short-term and long-term OA management strategies**

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One proposed way to frame currently recommended self-management strategies is by recognizing two sets of actions: short-term ‘fast’ strategies and long-term ‘slower’ strategies. The goal of short-term management strategies is to act ‘fast’ to reduce acute flare episode duration, minimize their impact and empower patients to feel more in control of their symptoms, with a focus on short-term outcomes. Although empirical evidence underpinning short-term ‘fast’ management strategies would be needed, examples of fast-action response models for flare interventions include NSAIDs use for the shortest possible period76 and short-term decrease in usual activities/exercise.65

The goal of long-term ‘slower’ management strategies is to optimize chronic condition management and reduce acute flare episode frequency. These can include core best-practice elements; education, support with weight loss (as required) and physical activity/exercise participation.70-72 An overall goal is to support people to develop and maintain physically active lifestyles. Avoidance of flare-provoking triggers may be advisable in some contexts but for others may not always be practical, for example, if frequent kneeling is a feature of occupation. Periods of decreased activity or avoidance, if needed, should be followed by a return to usual activities as much as possible on flare resolution. This will prevent negative emotions due to increased sedentary time and inactivity78 and prevent deconditioning, which themselves may lead to more flare episodes. Patients should be educated to try to avoid catastrophizing activity-related pain increases79 and flares, and be supported to expect and manage their periodic occurrence. Social interactions80 and partner support81 can also positively affect the OA pain experience.

Adopting short-term ‘fast’ management strategies has implications for the speed of access to healthcare. As discussed above, patient flares and healthcare consultations rarely coincide and patients’ ability to establish fast-response strategies as required may need to be developed through self-management education (see Figure 3, for example). More responsive healthcare, for example, through digital platforms, may also facilitate quicker supportive action. Furthermore, integrated ‘fast’ and ‘slower’ management strategies could reduce the disruption to chronic condition management caused by flares.

**[Figure 3]**

There remain a number of research knowledge gaps around OA condition management and timing of treatment responses. Viewing and managing OA as an ‘acute-on-chronic’ condition with current core and adjunctive interventions contributing to short-term ‘fast’ management and long-term ‘slower’ management strategies could translate into improvements in patient care.

***OA flare management and joint deterioration***

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Currently, there is no evidence to suggest that flares signify deterioration of joint structures. However, the extent to which painful inflammatory flare responses in OA are necessary processes for the joint to restore homeostasis versus reactions within the joint that should be minimized to preserve joint health remains unclear.49 This potentially has implications for how NSAIDs are prescribed. For example, in the theoretical context of Figure 2, whether NSAIDs might be more or less effective early or late in the disease course is unknown. In consultation with patients, healthcare professionals should focus on managing the cumulative daily disability caused by flares, rather than structural joint changes.

**SUMMARY**

OA is a chronic long-term condition, and yet the lived experience is often familiar day-to-day pain punctuated by distinct episodic flares that likely vary in predictability, frequency, duration and intensity across the disease course. By emphasizing OA as an ‘acute-on-chronic’ condition, patient education and self-management advice can proactively support patients to formulate bespoke sets of strategies for acute flare episode management and chronic condition management to facilitate the primary goal of maintaining an active lifestyle.

**CLINICS CARE POINTS**

* Recognizing, defining and exploring OA flares in clinical practice should include consideration of joint pain, swelling and stiffness, psychological distress and the impact of symptoms on functioning and activities, whilst excluding serious underlying causes.
* A broad range of activity-related, psychosocial and environmental factors can trigger OA flares, many of which are potentially modifiable if patients are supported to understand their symptoms and gain more control over them.
* Flare patterns are often unique to each individual and likely to change over time, therefore flare management strategies may need to evolve within patients over the course of the condition and continual monitoring may be beneficial.
* In addition to acute flare episode prevention and management, aiming to reduce overall symptom variability may also be beneficial for optimizing self-care and prognosis.
* All healthcare professionals involved in the provision of patient care should provide consistent messages about OA, flares and management options, which may reduce unnecessary healthcare utilization and treatment escalation.
* Framing current OA best-practice guidance as short-term ‘fast’ management and long-term ‘slower’ management strategy interventions may help to optimize patient self-management and better conceptualize OA as an ‘acute-on-chronic’ condition.
* Helping patients better explore and understand their own flare behaviours and triggers should be discussed in the context of maintaining an active lifestyle and not catastrophizing their occurrence.

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**FIGURE LEGENDS**

**Fig 1.** Conceptual model of causality for OA flares acknowledging the contribution of potential distal and proximal causes. Illustration based on the ‘Cone of causality’.(*Adapted from* Bracken MB. Risk, chance and causation: Investigating the origins and treatment of disease. London: Yale University press; 2013; with permission.)

**Fig 2.** Theoretical natural history of symptomatic OA progression. The impact of intermittent discrete (potentially benign) flare-up episodes progress in frequency, intensity and duration, with reducing periods of remission and capacity for complete symptom resolution. These acute symptom events drive the underlying disease process, eventually resulting in constant pain, complete loss of organ reserve (capacity to restore homeostasis) and synovial joint failure. Each red bar representing a flare-up is preceded by potential exposure flare-up triggers. (*From* Thomas MJ, Neogi T. Flare-ups of osteoarthritis: what do they mean in the short-term and the long-term? Osteoarthritis Cartilage 2020;28(7):870-3); with permission.)

**Fig 3.** Examples of currently recommended self-management strategies and advice for OA70,72 that may serve well as short-term ‘fast’ flare management strategies. Patients could be taught how to implement these alongside long-term ‘slower’ management strategies such as condition education, weight loss (as required) and physical activity/exercise participation.70-72 (Figure courtesy of J Bowden (PhD) and J Eyles (PhD), Institute of Bone and Joint Research, Sydney, NSW, Australia.)