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Symptoms, Signs and Quality of Life (QoL) in Osteoarthritis (OA)

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1. Introduction

Osteoarthritis is a syndrome with heterogeneous clinical presentations. Joint pain is the cardinal symptom accompanied by varying degrees of functional alterations like joint stiffness and instability. Clinical presentations are diversified, depending on which joint is affected, how severely it is affected, and the number of joints involved. The disease onset is usually subtle and unrecognized, but at the later stages, symptoms can be overt and debilitating. In between the onset and the late stages, the symptoms progress at variable rate and the patterns can be stepwise, continual, or static. The implications of osteoarthritis towards individual's quality of life (QoL) are different among different individuals and may not be directly proportional to the severity of structural abnormalities of the joints. Biopsychosocial factors come into play, and associated co-morbidities may further complicate the situation.

2. Symptoms in osteoarthritis

2.1 Joint pain

It is usually the chief complaint of symptomatic osteoarthritis which leads patients to seek medical attention. There are 2 types of pain in joint osteoarthritis, the mechanical and inflammatory pain. Typical mechanical OA pain is often described as deep and dull ache, localized to one or a few joints. The pain is aggravated by prolonged use or after extreme-ranged movements of the involved joint(s), by the end of the day, or after an increased mechanical load (O'Reilly & Doherty, 1998). Usually, the mechanical pain is relieved by rest or by gentle massage. Early in the disease, the pain is only episodic; its precipitants are usually known and predictable and the pain episodes are self-limiting. With the progression of the disease, the pain may become constant; occur at rest or even at night. At late stages, this mechanical joint pain may turn into unanticipated episodes of sharp pain superimposed on the pain at baseline (Hawker et al., 2008). This sharp pain is stabbing in character, more severe and stressful, occurring more frequently during movements after a period of resting (Chan, K.K.W. & Chan, L.W.Y., 2011). In contrast, the onset and frequency of inflammatory pain was less predictable. It could be triggered by weather changes, prolonged walking, a minor sprain, or from misplacement of the feet during walking. Sometimes, inflammatory pain occurred as flares in the form of exaggerated pain on the background of mechanical pain. According to a qualitative study on knee OA, most patients (80%) could distinguish

between mechanical and inflammatory pain, describing the character of each very differently. Inflammatory pain was described as a burning pain that could persist for days without treatment. Patients found resting and ice packing helpful, but most help came from taking analgesics, especially the non-steroidal anti-inflammatory drugs (NSAIDs). The frequency of inflammatory pain was highly unpredictable, varying from once every few weeks to once every few months. Sometimes the inflammatory pain might have a relapsing pattern, with the pain regressing gradually and relapsing again a few days later. This pattern could persist for 3 to 4 months of a year. Irrespective of whether pain was mechanical or inflammatory in nature, patients would avoid events that would trigger or aggravate the pain or take analgesics before the event as a preventive measure (Chan, K.K.W. & Chan, L.W.Y., 2011).

Although joint pain from osteoarthritis is typically local, in some patients the pain may be referred. For example, pain from OA hip may refer to the knee; and pain from OA cervical facet joints may refer to shoulder, arm, forearm and hand. In case of OA involving cervical or lumber facet joints, the pain may involve radicular components with features of sharp shooting pain different from the typical OA pain which are dull and achy. Most patients with symptomatic OA had symptoms in more than one joint. In a study of 500 patients with limb joints OA, only 6% had symptoms confined to a single joint (Cushnaghan & Dieppe, 1991). The most frequently affected joints were knees (41%), hands (30%) and the hips (19%).

The cause of joint pain in osteoarthritis is not well understood. It has been suggested that different mechanisms may produce pain characteristics by different joint structures (Kellgren, 1983):

- As cartilage is aneural, joint pain should arise from adjacent structures.
- Subchondral bone microfracture and osteophytes which stretch nerve endings in the periosteum may cause consistent joint pain on use.
- Bone angina caused by distortion of medullary blood flow and by thickened subchondral trabeculae, leading to intraosseous hypertension and intraosseous stasis. This may contribute to nocturnal joint pain (Arnoldi et. al., 1972).
- Joint inflammation involving the enthesis, joint capsules and synovium may cause pain at rest.
- Structural alteration, muscle weakness and altered usage of the joint with osteoarthritis would lead to stretching of the joint capsule, muscle spasm, enthesopathy and bursitis which in turn lead to the typical mechanical or activity-induced joint pain.

The degree of joint pain does not always correlate with the degree of structural changes of osteoarthritis which is usually defined by abnormal change in appearance of the joints on radiographs (Hannan et al., 2000), e.g. pain can be absent in spite of severe joint damage in some cases. Nevertheless, those with significant radiographic changes are more likely to have joint pain than those with mild changes (Duncan et al., 2007), and the concordance between symptoms and radiographic osteoarthritis are greater with more advanced structural damage (Peat et al., 2006).

2.2 Joint stiffness

Joint osteoarthritis may present with joint stiffness especially in the morning. It is a tight and "gelling" sensation of the joints, periarticular soft tissues and musculature, rendering the joint difficult and slow to move. Unlike diffuse stiffness in rheumatoid arthritis, stiffness of OA is confined to the region around the affected joint. Typical OA joint stiffness occurs after

prolonged period of immobilization, not restricted to the time in the morning, and usually lasts less than 30 minutes. As the disease progresses, prolonged stiffness would be evident. It is attributable to joint incongruity and capsular fibrosis as a result of the process of osteoarthritis.

2.3 Joint instability

Patients with osteoarthritis of joints in lower limbs frequently experience a sensation of instability or buckling, i.e. shifting without actually falling or giving way. It tends to be more common in patients who have OA in multiple joints of lower limbs. Such buckling can be the results of:

- Weakness of periarticular musculature from joint disuse
- Muscle fatigue because the peri-articular musculature have to work harder to move the joint as the coefficient of friction increases due to the cartilage surface fissures or loses integrity
- Laxity of ligaments as a result of narrowing of joint spaces from the loss of the supporting cartilage or from other joint deformities

3. Signs in osteoarthritis

3.1 Crepitus

It is an audible and palpable cracking or crunching over a joint during its active or passive movement. It is presumably caused by irregular articular surface attributable to the degenerative process rubbing against each other during motion. The degree of crepitus may be correlated with the degree of degenerative process (Ike & O'Rourke, 1995). However, many people have significant crepitus at their joints in the absence of any joint pain.

3.2 Restricted joint motion

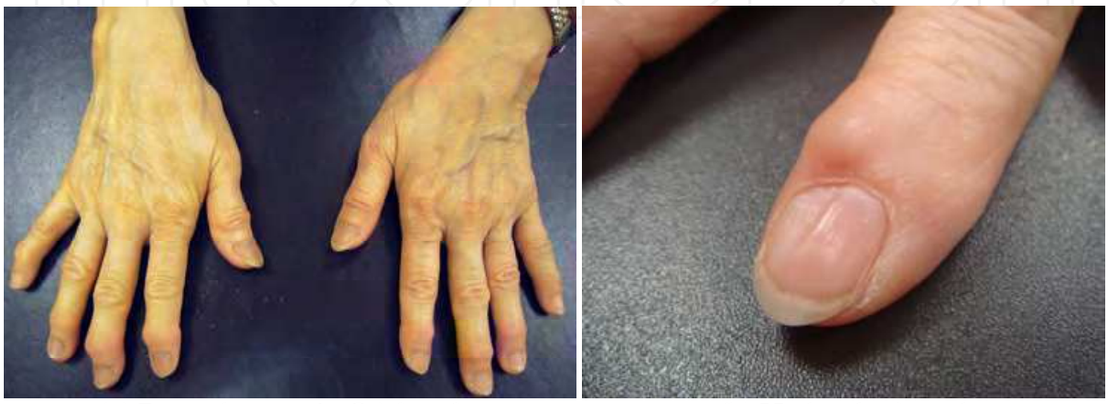
The restricted movement over the degenerated joint can be caused by pain, effusion, capsular contractures, muscle spasm or weakness, intra-articular loose bodies, mechanical constraints by loss of joint cartilage and joint misalignment. Such feature may or may not associate with stress pain at extreme range. In order to make a differentiation, both active and passive ranges of joint motion are tested. The active movements give a rough idea of range of motions available in the joint, the pain experienced by the patient and the power in the peri-articular muscle groups. Passive joint movement particularly gives information on pain, range and the end-feel, i.e. the specific sensation imparted from the joint onto the examiner's hands at the extreme of passive movement (Cyriax J & Cyriax P, 1993). The quality of the end-feel is dependent upon the nature of tissue that is compromising full motion of the joint. For example:

- Elastic end-feel may be attributable to joint effusion.
- A springy end-feel is appreciated when the joint is springing or bouncing back at the end range by an intra-articular loose body.
- An abnormal hard end-feel may be attributable to involuntary muscle spasm or capsular contracture
- A bony-hard end-feel could be due to bony restraints by loss of joint cartilage, osteophytes impingement and joint misalignment.

For better interpretation of abnormal end-feel of a joint, one can compare the sensation with the joint on the contralateral side.

3.3 Bony changes and joint deformity

Bony enlargement in OA is attributable to the formation of osteophytes and the remodeling process leading to peri-articular bony hypertrophy and subchondral cyst formation. Incongruent degeneration of the joints will also contribute to joint angulations and misalignment. The classical example is the deformities in the nodal OA of hand (Figure 1) which causes bony enlargement at the distal and proximal interphalangeal joints known respectively as *Heberden's nodes* and *Bouchard's nodes*; and the angular deformity of the carpometacarpal and metacarpophalangeal joint of the thumb giving rise to the *squared hand*.



(a) Bony changes in OA hand (b) Herberden's node

Fig. 1. Nodal OA of hand

3.4 Joint tenderness

Tenderness with pressure along the joint margin is typical for OA. However, peri-articular structures may also be tender contributing to the joint pain, e.g. myofascial trigger points, adjacent bursitis or tendonitis, and ligament enthesopathy. Point tenderness should be sought away from the joint line to find out concomitant painful structures to guide management.

3.5 Variable levels of inflammation

Variable degree of synovitis may be found in the joints with osteoarthritis, giving rise to local palpable warmth, effusion and synovial thickening (Figure 2). This could also be one of the sources of joint tenderness. These features are usually intermittent and appear with the flare-ups in the osteoarthritic joint.

3.6 Muscle atrophy

Peri-articular muscular wasting may be apparent as a result of OA of the corresponding joint due to disuse muscle atrophy. The associated muscle weakness would compromise joint stability and muscle tones around the joint which further jeopardize the integrity of the joint (Hurley, 1999). For assessment of muscle strength and size, examiner can perform resisted movement of the joint and by direct measurement of the diameter of the muscle bulk compared to that on contralateral limb.

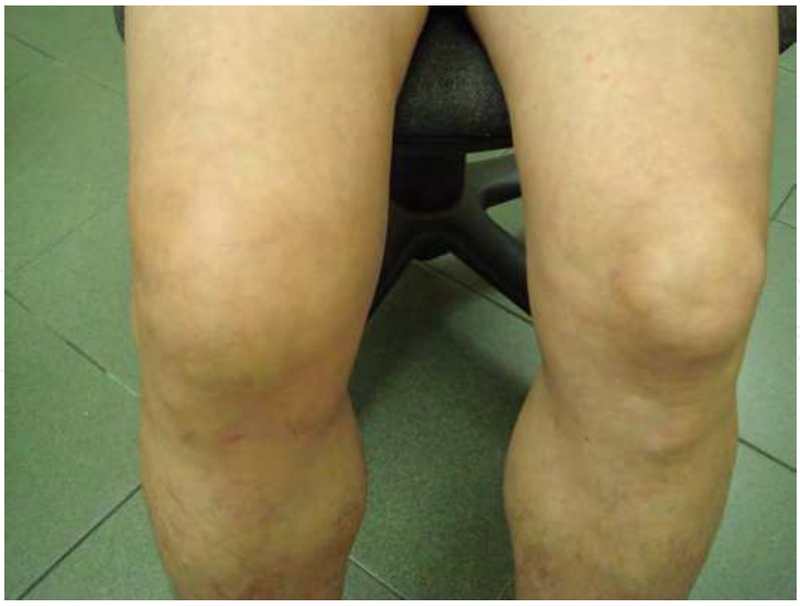


Fig. 2. Warm right knee effusion caused by synovitis in knee OA.

3.7 Absence of systemic manifestation

Symptoms of osteoarthritis are usually localized to the affected joint and systemic manifestations like fever, weight loss, anemia, fatigue and malaise are not features of primary OA. The presence of such features should alert the physician to consider other differential diagnoses like rheumatoid arthritis, although some subsets of OA occasionally give rise to systemic manifestation such as those crystal associated OA.

4. Clinical patterns and subsets

Osteoarthritis affects many joints, with heterogeneous clinical patterns, and may be triggered by diverse constitutional and environmental factors. The causes and clinical presentations among individuals with osteoarthritis could be quite different from each other. The trend in recent years is to separate osteoarthritis into more homogeneous grouping as subsets in order to better define respective etiological factors and to determine corresponding natural history and prognosis. The grouping of subsets was made according to the following clinical characteristics (Altman, 1991):

- Identifiable causes of osteoarthritis
- Joint sites involved
- The number of joints involved
- The pattern of joints involvement
- The presence of associated crystal deposition
- The presence of marked inflammation
- The radiographic bone response

It is important to note that the subset grouping of osteoarthritis is arbitrary and sharp distinction between subsets does not exist. It is possible that different subsets appear in one individual and evolution from one subset to another could occur with time and at different sites.

4.1 Nodal generalized OA (NGOA) or primary generalized OA (PGOA)

It is the best recognized OA subset. It has a female preponderance, marked familial predisposition, peaked onset in the middle age. Joints involved are symmetrically affected. Characteristically, it affects the distal interphalangeal joints (DIPJ) of the fingers with gelatinous cyst and bony outgrowth at the dorsal surface of the involved joints known as Heberden's nodes (Figure 1). In addition to DIPJ of the fingers, similar lesions may affect the proximal interphalangeal joints (Bouchard's nodes) of the fingers. Other frequently involved joints are carpometacarpal, metacarpophalangeal and interphalangeal joints of the thumbs, the acromioclavicular joint, the spinal facet joints, the hips, the knees and the first metatarsophalangeal joint. The disease typically goes through an episodic symptomatic phase (over one to three years) with considerable inflammation. In most cases, symptoms then subside resulting in a good deal of deformity but seldom give rise to serious disability (Pattrick et al., 1989).

4.2 Erosive OA (Punzi, 2004)

This is a rare condition and is considered as a more aggressive form of PGOA primarily affecting small joints of the hands. It has been documented to actually be a manifestation of calcium pyrophosphate deposition disease (Rothschild, 2006; Rothschild and Bruno, 2011; Rothschild and Yakubov, 1997; Rothschild et al., 1992). Women between 45 and 55 years are most typically affected and it has a strong familial predisposition. Joints are symmetrically affected and both the distal and proximal interphalangeal joints are equally affected with the typical Heberden's nodes and Bouchard nodes. The inflammatory features of the disease are florid with overt pain, synovial swelling and erythema. The hallmarks of the condition are the presence of destructive crumbling erosion demonstrated radiographically, with occasional joint instability at the interphalangeal joints. As a result, the functional outcome of the hand is much less favorable.

4.3 Local large joint OA

4.3.1 Hip

The classical symptom of hip OA is groin pain on weight bearing. The patients typically have difficulty in flexing and internally rotating their hips, so they may feel pain when getting in or out of the car, or when bowing forward to reach the ground or their feet. In rare cases, the patients may only present with referred knee pain from the hip. The condition is more common among white Caucasians, but is significantly less common among Chinese (Nevitt et al., 2002) and black African populations. There are two major subgroups of hip OAs defined by radiological patterns: the superior pole OA and the medial pole OA.

4.3.1.1 Superior pole OA

It is the common form of hip OA where degenerative process affects the weight bearing superior surface of the femoral head and the adjacent acetabulum. It has a male preponderance and associated with obesity or local structural abnormality. The disease is usually unilateral at presentation but likely to progress and may involve another hip as the disease evolves. The hallmark is superolateral femoral head migration with osteophytes at lateral acetabulum and medial femoral margins combined with typical buttressing of medial femoral neck cortex on hip radiographs (Figure 3).

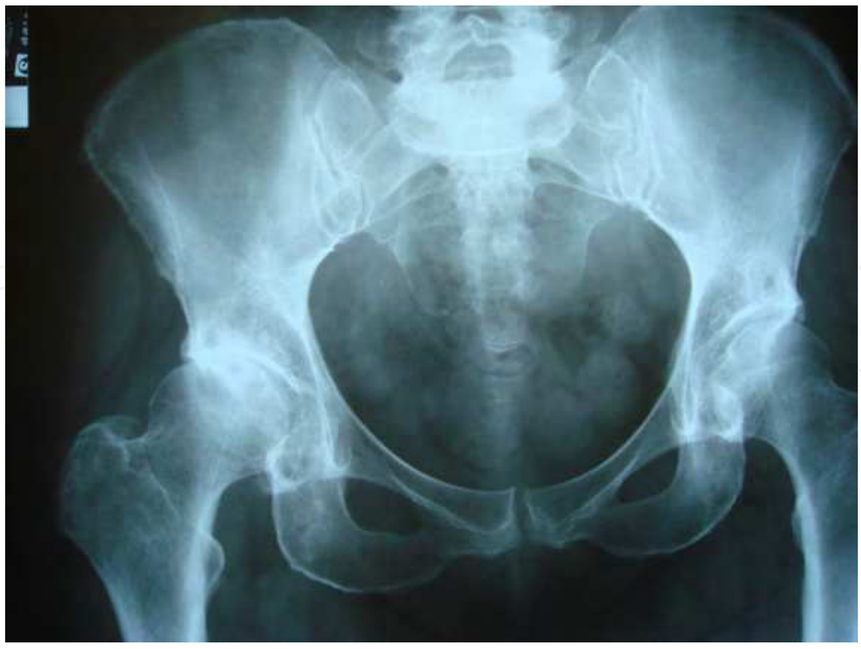


Fig. 3. Radiograph of Superior pole OA hip.

4.3.1.2 Medial pole OA

It is less commonly seen where more widespread, central cartilage loss are present at the hip. It has a female preponderance and usually associated with hand OA as part of the PGOA syndrome. The disease is more likely bilateral at presentation but less likely to progress.
Signs involve reproduction of the groin pain, typically on internal rotation and flexion of the hip. Trendelenburg sign may be present. When the patient stands on the unaffected side, the pelvis as viewed from the back remains level; while the patient stands on the painful side as a result of hip OA, the unsupported side of the pelvis will drop as a result of weak gluteus medius muscle on the side of painful hip. In moderate to severe diseases, hip flexion contracture may be demonstrated.

4.3.2 Knee

The knee is the commonly involved joint of osteoarthritis. The condition is primarily affecting elderly people with female preponderance and associated with obesity. There are three compartments of the knee that can be affected by OA: the medial tibiofemoral compartment is more commonly affected than the lateral tibiofemoral compartments; but there is a lack of data addressing prevalence of patellofemoral OA (chondromalacia patellae) and its correlation to tibiofemoral disease (McAlindon et al., 1992). The classical symptom of knee OA is knee pain on weight bearing. The pain is particularly aggravated when walking downstairs and raising up from chair after prolonged sitting if patellofemoral disease (chondromalacia patellae) is involved. Stiffness and gelling of the joint are frequent complaints of knee OA. Signs involve bony enlargement, joint tenderness, crepitus on movement and occasional joint effusion. Popliteal (Baker's) cysts are the bursae that communicate with the knee joint space which may become quite large and tense leading to posterior knee pain (Figure 4). Sometimes, patients with knee OA may have their Baker's cysts ruptured and present to the clinician with signs and symptoms mimic that of deep

vein thrombosis. In moderate to severe knee OA, there may be joint deformity (varus for medial compartment disease and valgus for lateral compartment disease).

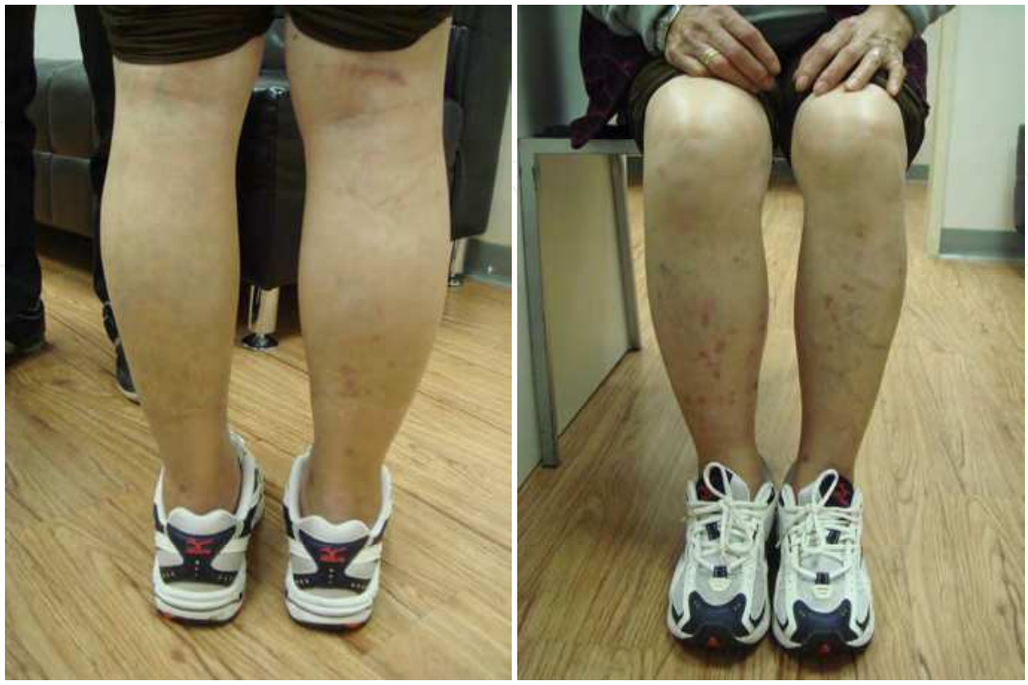


Fig. 4. Ruptured Baker's cyst of right knee with OA

	PGOA	Erosive OA	Superior pole hip OA	Medial pole hip OA	Knee OA
Preponderance	Female	Female	No	Female	Female
Familial predisposition	Marked	Marked	Sporadic	Marked	Mostly sporadic
Age	Middle age	45-55	↑ with age	Middle age	↑ with age
Symmetry	Yes	Yes	No	Yes	No
Joints involved	Multiple, DIPJ, PIPJ commonly involved	Mainly PIPJ & DIPJ	Superior pole of hip	Medial pole of hip	Medial > Lateral knee compartments
Inflammation*	Episodic	Florid	Episodic	Episodic	Episodic
Systemic manifestation	No	Yes	No	No	No
Progression	Slow	Aggressive	Variable	Less likely	Variable
Outcome disability	Low	High	Variable	Low	Variable
Hallmark	Herberden's/ Bouchard's nodes	Subchondral erosion on radiographs	Superolateral femoral head migration + osteophytes at lateral joint margin	Radiograhs: widespread central joint space narrowing	Bowed leg, Baker's cyst

* Any inflammation can be caused by the co-existing crystal arthritis

Table 1. Comparison between different OA clinical subsets

5. Natural history of osteoarthritis

Few studies assessed the natural history of knee OA (Dieppe et al., 1997; Ledingham et al., 1995; Massardo et al., 1989; Schouten et al., 1992); and the conclusions are that the natural history of osteoarthritis is highly variable (Hochberg, 1996). In a large scale prospective observational study of 188 participants with OA knees follow-up over 1-5 years, approximately 50% of the patients described worsening of their symptoms with time. However, a significant portion reported improvement (Schouten et al., 1992). In another smaller scale retrospective study on 72 patients with symptomatic knee OA even found that more than 50% of clinical phenomena improved within 6 months (Berkhout et al., 1985). Factors that may associate with the progression of disease especially pain remain speculative. But it is observed that the disease progression is the result of a complex interplay between structural changes of the joint(s) and related psychosocial factors of the affected patients which highlights the importance of study of morbidity associated with OA.

6. Morbidity with different degrees of quality of life (QoL) impairments

In 1947, the World Health Organization (WHO) defined health not just by the absence of disease or infermity, but as a state of complete physical, mental and social well-being (World Health Organization, 1980). The departure from such a state is morbidity. The health related QoL is used to describe different domains within such a broader term of health or as a measure of morbidity associated with any health conditions. The specific dimensions found in most health related QoL definitions include: degrees of physical symptoms, functional limitations, emotional well-being, social functioning, role activities, life satisfaction and health perception (Fioravanti et al., 2005; Rejeski & Shumaker, 1994). Osteoarthritis, being a highly diversified clinical condition, would lead to morbidity with different degrees of QoL impairments among affected individuals (Woo et al., 2004).

6.1 Physical symptoms: Pain

Pain is usually the predominant symptom in patients with symptomatic OA. Pain in OA affects different domains of one's QoL: sleep interruption (Leigh et al., 1998; Wilcox et al., 2000), psychological stress (Downe-Wamboldt, 1991), reduced independence (Gignac et al., 2000), poorer perceived health (Loborde & Powers, 1985) and increased healthcare utilization (Badley & Wang, 1996). The likelihood of mobility problems increases as pain increases (Wilkie et al., 2007).

Yet, some patients experience significant pain and with subsequent QoL compromise even before OA has progressed enough to produce radiographic abnormalities. The reverse is also common; some patients feel little or no discomfort with low morbidity even though their radiographs show advanced OA. Why is there such a great discrepancy? Several observations suggest that pain in OA is not simply attributable to the structural changes in the affected joint, but the result of interplay between structural change, peripheral and central pain processing mechanism (Creamer & Hochberg, 1997) which can be explained by multi-dimensional concept of pain via Loeser's onion ring pain model (Figure 5): although pain is a nociceptive event (cognition of pain sensation from nociceptors), whether pain may lead to suffering (negative affection) and subsequent pain behaviors e.g. absence from work or healthcare utilization is mainly shaped by external psychosocial and other environmental factors (Loeser & Cousins, 1990).

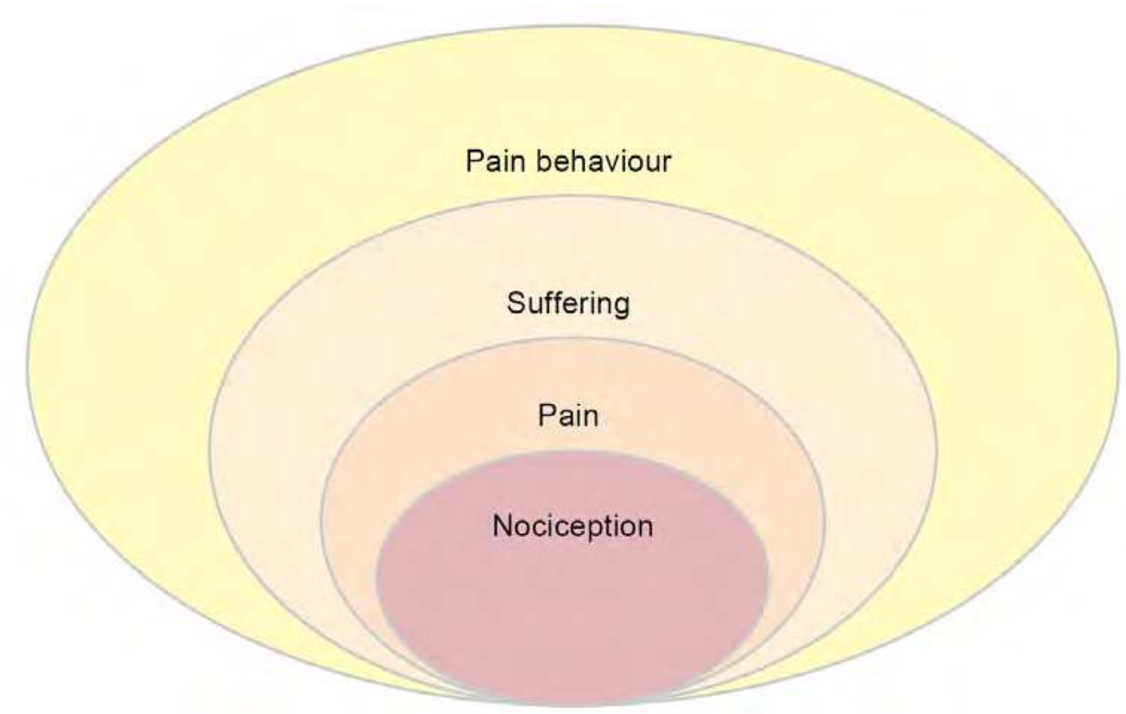


Fig. 5. Loeser's pain model

6.2 Biopsychosocial impairments

Similar concept which explains the impact of diseases upon individuals is the biopsychosocial (BPS) model (Engel, 1977). In such a model, OA is a *disease* which involves objective disorder at molecular, cellular, physiological, mechanical and structural levels confined to the affected individual. The affected individual will have a psychological awareness and perception of dysfunction at the personal level; such subjective state is known as the *illness*. The physical and/or psychological dysfunction as a result of the disease or illness at the personal level e.g. difficulty in climbing stairs is the *disability*. The compromised social role assumed by the affected individual as a result of the disease, associated illness and disability is termed as *handicap*. All the biological, psychological and social parameters are important determinants in the final outcomes, impairment of which would greatly compromise individuals' overall QoL in the context of the disease.

In the context of osteoarthritis, studies showed that pessimism was associated with poor physical outcomes (Brenes et al., 2002; Ferreira & Sherman, 2007). Observational studies found negative mood was correlated with more joints involvement and disability by OA and vice versa (Van Baar et al., 1998). Qualitative studies noticed patients with OA expressed declined life satisfaction and that depression and anxiety were their major mood problems (Tak & Laffrey et al., 2003). Patients felt distressed with not being able to participate in activities that they used to be able to do. The most frequently quoted activities are leisure activities such as travel, social activities, close relationships, community mobility, employment and heavy housework (Gignac et al., 2006). Some patients with advanced OA even perceived the disease threatened their self identities and felt lack of power to change their situation. However, studies also showed that a main bulk of OA patients would ignore their disease and tried to carry on their normal life regardless of symptoms exacerbation (Cook et al., 2007). From these studies, we can have a glimpse of the highly diversified OA morbidity as a result of inter-relationship between biopsychosocial factors among different OA patients.

6.3 Co-morbidities

Co-morbidity is defined as the co-existence of two or more health problems in a person. As OA is an age-related condition, patients with OA are also likely to suffer from a number of other disabling and chronic health conditions (Kadam et al., 2004). In a cross-sectional study of 455 patients suffering from knee OA, 78% of patients had at least one musculoskeletal co-morbidity and 82% had at least one non-musculoskeletal co-morbidity, on average they had 3.2 co-morbidities (Chan et al., 2009). The presence of co-morbidities would further complicate the clinical outcomes and impair patients' QoL in the following ways:

- Co-morbidities can interact with each other to produce high levels of disability. For example, there are increased pain and decreased mobility in patients with musculoskeletal co-morbidities (Croft et al., 2005); decreased ambulation and general health in patients with concomitant angina, chronic obstructive pulmonary disease, previous stroke or obesity.
- Polypharmacy issues may intervene. For examples, drug safety issues of COX-2 inhibitors among patients with cardiovascular co-morbidity; risk of gastro-intestinal upset or bleeding from the use of non-steroidal anti-inflammatory drugs (NSAIDs) among patients with gastro-intestinal co-morbidity.
- Depression can accompany any chronic condition like OA and lead to significant morbidity according to biopsychosocial model of disease. Treatment of the depressed individual with OA with antidepressants can improve pain, function, and quality of life scores (Lin et al., 2003).
- The presence of severe co-morbid conditions may influence the choice of treatment for OA, for example exercise, and joint replacement surgery.

7. Conclusions (Figure 6)

Osteoarthritis (OA) is not just a degenerative disease, but a clinical syndrome of joint pain with highly diversified clinical presentations. It is accompanied by varying degrees of functional limitation and reduction in quality of life. Discordance between osteoarthritis pathology, symptoms and disability is frequently encountered; hence structural pathology is not the absolute determinant to the clinical outcome. Psychosocial factors and co-morbidities are crucial issues which amalgamate the final health status of the affected individuals.

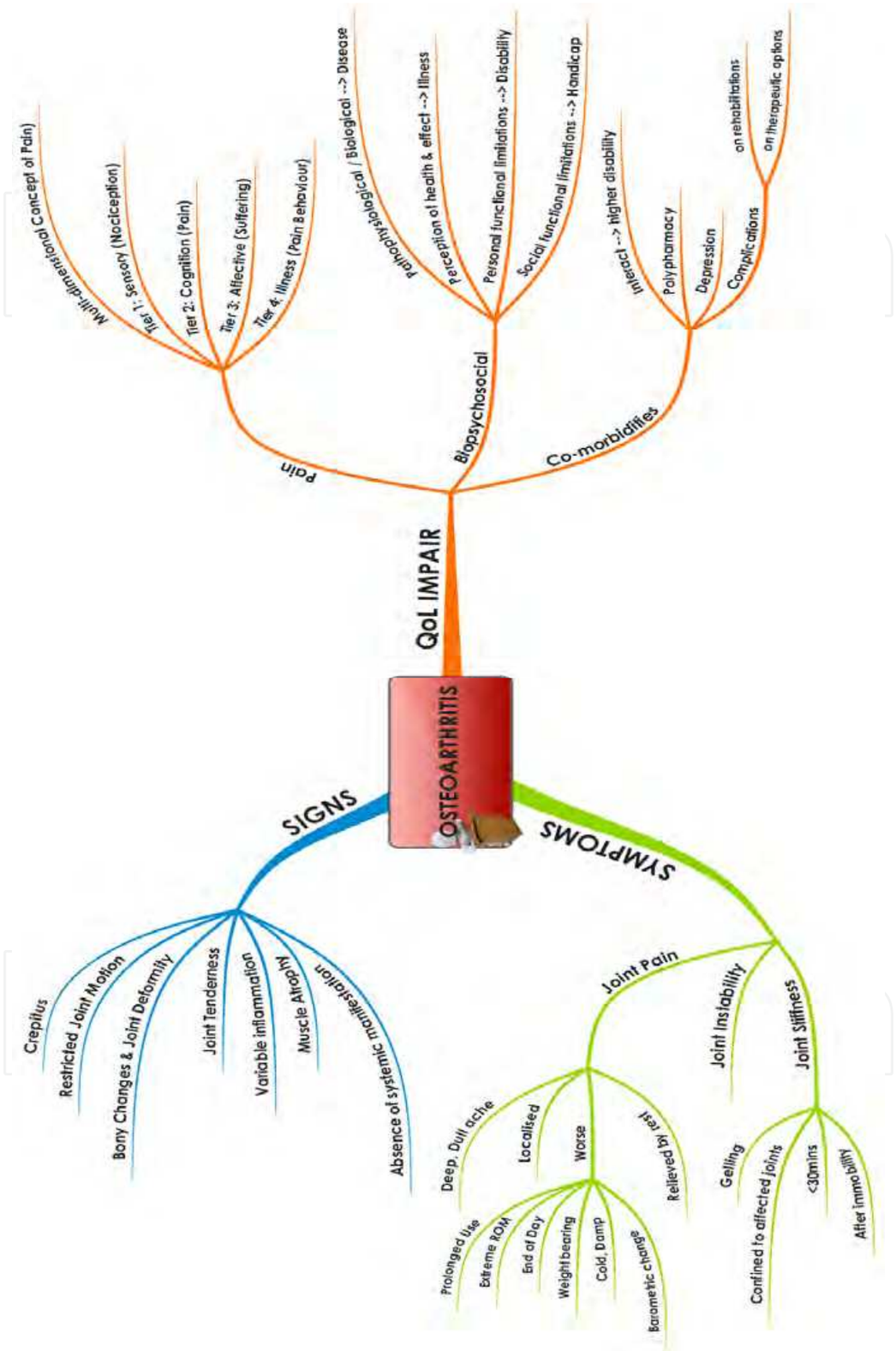


Fig. 6. Summary of symptoms, signs and QoL in OA.

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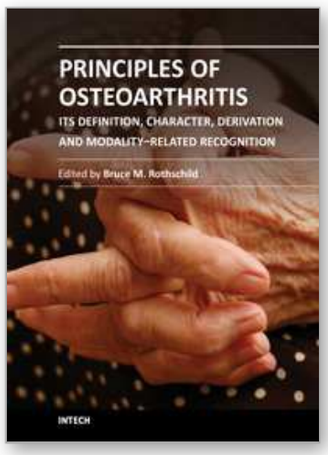

Principles of Osteoarthritis- Its Definition, Character, Derivation and Modality-Related Recognition

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This volume addresses the nature of the most common form of arthritis in humans. If osteoarthritis is inevitable (only premature death prevents all of us from being afflicted), it seems essential to facilitate its recognition, prevention, options, and indications for treatment. Progress in understanding this disease has occurred with recognition that it is not simply a degenerative joint disease. Causative factors, such as joint malalignment, ligamentous abnormalities, overuse, and biomechanical and metabolic factors have been recognized as amenable to intervention; genetic factors, less so; with metabolic diseases, intermediate. Its diagnosis is based on recognition of overgrowth of bone at joint margins. This contrasts with overgrowth of bone at vertebral margins, which is not a symptomatic phenomenon and has been renamed spondylosis deformans. Osteoarthritis describes an abnormality of joints, but the severity does not necessarily produce pain. The patient and his/her symptoms need to be treated, not the x-ray.

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