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Current Concepts Review

# Deepening the subchondral insufficiency fracture and osteonecrosis of the knee dilemma: Time for a new classification: Current concepts

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ABSTRACT

Knee osteonecrosis (ON), often subclassified as spontaneous ON of the knee, secondary ON, and post-arthroscopic ON of the knee, is a common disorder often associated with suboptimal outcomes. Magnetic resonance imaging is the current gold standard for diagnosis, revealing bone marrow edema and subchondral fracture lines. Therapeutic methods range from conservative treatments, such as partial weight-bearing, pharmaceutical interventions, and physical therapy, to surgical procedures in cases of advanced joint collapse. Available evidence from histological studies consistently shows the absence of bone necrosis, highlighting microfractures and bone remodeling as central features of these lesions. Therefore, the appropriateness of this terminology has recently been questioned, with knee ON being more accurately reinterpreted as subchondral insufficiency fracture of the knee (SIFK). This clinical dilemma stems from longstanding misclassification that has led to diagnostic confusion and inconsistent treatment approaches. Despite this progress, several unresolved issues persist. The precise biomechanical and biological factors that initiate SIFK remain unclear, and the optimal timing for intervention is still debated. In addition, long-term outcomes of both nonoperative and operative treatments have yet to be definitively established. Addressing these gaps requires comprehensive clinical trials and advanced imaging studies that correlate histological findings with patient outcomes. This evolving understanding calls for a reclassification of knee ON lesions, aiming to enhance diagnostic accuracy and inform more effective, targeted treatment strategies.

**Current concepts:**

- Knee osteonecrosis (ON) is a common disorder characterized by severe pain, weight-bearing intolerance, and bone marrow lesions at magnetic resonance imaging.
- Traditional classifications include spontaneous ON of the knee, secondary ON, and post-arthroscopic ON the knee.
- Histological evidence shows the absence of true bone necrosis in many cases.

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**Future perspectives:**

- The term subchondral insufficiency fracture of the knee (SIFK) should be preferred over osteonecrosis.
- Refined diagnostic criteria based on advanced imaging and histological techniques are needed.
- Research on the biomechanical and biological mechanisms of knee lesions must be expanded.
- Treatment protocols should be developed based on updated definitions and improved understanding of these conditions.

## INTRODUCTION

Originally described in 1968 by Ahlbäck et al. [1], knee osteonecrosis (ON) was defined as "spontaneous", in the absence of an identifiable cause or histopathological evidence. In the same year, Ahlbäck also identified a second form of knee ON associated with the use of corticosteroids (CCSs). Later in 1991, Brahme et al. [2] described a third type, diagnosed after arthroscopic surgery and therefore defined as "post-arthroscopic". Even in this case, no obvious causes were recognized. The current classification of knee ON [3] (Fig. 1) has remained largely unchanged despite the emergence of new evidence into the field. Recently, the appropriateness of the term "ON" has been questioned, with some authors advocating for its replacement by "subchondral insufficiency fracture of the knee" (SIFK) [4–11]. Nevertheless, over 50 years after its initial description, these three forms—spontaneous ON of the knee (SONK), secondary knee ON, and post-arthroscopic ON of the knee (PONK)—remain broadly recognized.

### *Subchondral insufficiency fracture of the knee*

Ahlback et al. [1] first described SONK based on radiological findings and criteria derived from other joints and applied it to the knee. However, no histological evidence of necrosis was provided. In 2000, Yamamoto and Bullough [5] demonstrated the absence of necrosis in bone samples from patients diagnosed with SONK who had undergone surgery. Subsequent studies [9] confirmed this finding, identifying subchondral fractures as the primary pathology in such cases. At advanced stages, Koshino et al. [12] recognized small, localized areas of necrosis within the context of subchondral fractures not to be mistakenly classified as ON [10]. While small areas of necrosis may coexist with SIFK in advanced stages, how SIFK leads to cell necrosis remains unclear. It has been hypothesized that increased pressure and reduced blood supply might induce cell death and necrosis [13]. Lotke et al. [14] suggested that microfractures in the osteoporotic subchondral bone plate could allow fluid transfer from the cartilage to the bone marrow, raising local pressure within the latter and potentially leading to focal areas of necrosis. However, the most widely accepted theory, supported by histopathological evidence, posits that delayed or non-healing fractures may result in necrotic areas due to failed bone repair mechanisms [10]. The current nomenclature has already been questioned by previous authors who suggested replacing the term "ON" with "SIFK" [4–11]. The latter refers to microfractures of the subchondral layer caused by repetitive stress on the knee joint [15]. Several risk factors for SIFK have been reported, including meniscopathy [4], osteoarthritis [16], osteoporosis [17], obesity [18], age [1], and female sex [1,16]. It remains unclear whether SIFK leads to osteoarthritis or whether preexisting joint degeneration predisposes to the development of SIFK. It is likely that both mechanisms coexist. In advanced cases of SIFK, collapse of the articular surface and subsequent cartilage damage can trigger a pronounced inflammatory response, ultimately leading to osteoarthritis. Conversely, in an already degenerated knee, compromised bone quality and altered biomechanics may increase susceptibility to SIFK [19]. A recent review by Yokota et al. [17] highlighted the pivotal role of subchondral bone fragility in the development and progression of SIFK. The authors proposed that SIFK can be conceptualized as a form of "macrofracture" of the subchondral region surpassing osteoblasts' reparative capacity. Moreover, growing evidence indicates that pharmacological agents commonly used in the management of osteoporosis, such as bisphosphonates, may exert protective effects on subchondral bone integrity and contribute to cartilage preservation [20]. Obesity has also been identified as a potential risk factor for SIFK, likely due to increased peak joint pressures, particularly in the presence of meniscal pathology [21].

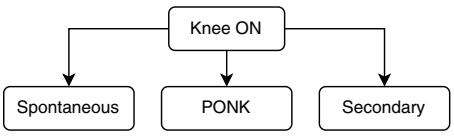


**Fig. 1.** Current classification of knee osteonecrosis (ON).

Meniscopathy represents the main risk factor for the development of SIFK since a meniscal lesion is described in more than 80% of SIFK cases [21], causing altered joint biomechanics, stability, and contact pressures between femur and tibia. A growing body of evidence highlights a strong association between SIFK and meniscal tears, especially medial meniscus posterior root tears (MMPRTs) [21–23], which compromise the meniscus's hoop stress function, leading to increased tibiofemoral contact pressures and subsequent SIFK [22]. A study by Clark et al. [21] reported a high incidence of MMPRTs and radial tears in patients with SIFK. Specifically, 78% of patients with SIFK had concomitant MMPRTs, indicating that such tears may play a key role in the pathogenesis of SIFK. Further supporting this association, Okazaki et al. [22] showed that the severity of SIFK correlated with MMPRTs. Longstanding MMPRTs were associated with more advanced SIFK, emphasizing the importance of early detection and management of meniscal root tears to prevent progression to SIFK. These authors also showed that pullout repair of MMPRT was effective in halting the progression of early-stage SIFK and led to improved clinical outcomes across all treated patients [22]. Pareek et al. [24] introduced the SIFK score, a predictive model for progression to arthroplasty following SIFK, which they later validated as having a high conversion rate [25]. Their findings suggest that addressing meniscal pathology, particularly through surgical repair of MMPRTs, may modify the disease trajectory and reduce the risk of progression to total knee arthroplasty. This effect is likely due to the restoration of native knee biomechanics. Supporting this, a recent in vitro biomechanical study by Boksh et al. [26] demonstrated that a combined technique using single root repair and centralization tunnel effectively reestablishes tibiofemoral contact mechanics and minimizes meniscal extrusion following MMPRT. In addition to timely meniscal repair, high tibial osteotomy (HTO) has emerged as an effective surgical option for patients with SIFK and concurrent varus malalignment [17,27]. This procedure redistributes mechanical loads across the knee joint, thereby reducing stress on the medial compartment. Evidence indicates that HTO can provide symptomatic relief and potentially halt lesion progression, particularly when performed in the early stages of SIFK [27]. A study by Mukai et al. [28], reported significant improvements in clinical outcomes, including pain reduction and enhanced joint function, in patients undergoing HTO for SIFK, especially when combined with mosaicplasty. Furthermore, the timing of intervention appears to be

critical: early HTO, performed before subchondral bone collapse, is associated with more favorable outcomes compared with delayed surgery [17].

Meniscectomy is also an important risk factor as it causes biomechanical changes on the articular surface [29], especially in cases of varus alignment following medial meniscectomy [30]. What has been historically referred to as PONK was, in the past, mistakenly attributed to the arthroscopic procedure itself, rather than to the biomechanical consequences of the meniscectomy often performed during such interventions. In reality, PONK has already been questioned by several authors [4,31], who have suggested that ON diagnosed after arthroscopy may actually represent a preexisting condition that was unrecognized prior to surgery. In fact, the only prospective study on PONK included only patients who had undergone meniscectomy [32], a well-established risk factor for the development of SIFK [4,33,34]. Other studies on PONK are primarily limited to case reports and small case series, offering a low level of evidence, as highlighted in a recent review [31]. Moreover, it cannot be ruled out that SIFK was already present before the arthroscopic procedure in many of these cases [4,16]. It has now been shown that many cases previously labeled as PONK are, in fact, more accurately characterized as SIFK, given the predominance of subchondral fractures over true ON in the imaging findings [30,35]. Several potential intraoperative factors have been proposed as contributors to SIFK in the setting of arthroscopy, including increased intra-articular pressure from irrigation fluids, the use of radiofrequency devices, and mechanical shavers, but there is currently no strong evidence that any of these factors directly cause SIFK [32]. Furthermore, the extremely low number of reported PONK cases relative to the vast number of arthroscopic procedures performed worldwide casts further doubt on arthroscopy as a significant causal factor [36]. A recent systematic review [31] identified only 55 cases of PONK in the literature, and of these, only 14 had undergone MRI outside the so-called “window period” defined by Nakamura et al. Furthermore, no differences in clinical presentation between SIFK and PONK have been identified, apart from a potential sex difference (Table 1) [3,31], which may simply reflect the small sample size. Given the absence of histopathological, clinical, or radiological distinctions between the two entities, it appears that SIFK and PONK may represent the same condition described under different names. There is currently no convincing evidence that arthroscopic procedures themselves induce ON. Rather, it is the underlying pathology, such as meniscal extrusion, radial tears, or flap tears, and subsequent surgical interventions, particularly extensive meniscectomies, that may predispose patients to the development of SIFK [34]. Based on the current body of evidence, there seems to be no justification for maintaining a distinction between PONK and SIFK.

Regarding clinical features, SIFK presents with an acute onset of pain, worsening with load and persisting at rest and at night. It typically affects women over 50 years of age and commonly involves the medial femoral condyle [11]. Ahlbäck et al. [1] described SONK in similar terms as the sudden onset of severe and persistent pain, particularly at night, without an apparent cause, localized at the medial femoral condyle within the weight-bearing zone. Therefore, from a clinical standpoint, SIFK and SONK are indistinguishable. MRI is the gold standard for diagnosing early-stage SIFK [11] and typically reveals a single, unicondylar bone marrow lesion (BML) in the epiphyseal area with bone marrow edema-like signal intensity, usually extending through two-thirds of the condyle and a subchondral hypointense line paralleling the articular surface [5]. This radiological description is superimposable in case of SONK and SIFK, as described by numerous authors [9,10]. In SIFK, the lesion can progress, leading to subchondral cracks, fluid infiltration under the subchondral plate, extended bone marrow edema-like signal intensity, cyst formation, and eventual collapse, resulting in an osteochondral defect [37]. These findings correspond to the final stage of SIFK [30]. At advanced stages, standard radiographs demonstrate gradual articular surface collapse [1]. Several radiologic classifications originally developed for SONK [38] are now applicable to SIFK. For example, Sayyid et al. [33] proposed an MRI classification describing the progression of SIFK. SIFK

**Table 1**
Characteristics of SONK and PONK compared.

| | SONK | PONK | Ref |
|---|---|---|---|
| Age (y) | >50 | >50 | [3,18] |
| Gender (M/F) | 1:3 | 1:1 | [3,16,31] |
| Pain | Sudden | Sudden | [3] |
| Laterality | Unilateral | Unilateral | [3,33] |
| No. of lesions | Single | Single | [3,17] |
| Condylar involvement | >MFC | >MFC | [3] |
| Other joint involvement | No | No | [3] |
| Associated diseases | No | No | [3] |
| Pathologic findings | Bone fibrosis, healing fracture | Bone fibrosis, healing fracture | [3] |

Abbreviations: MFC = medial femoral condyle; PONK = post-arthroscopic osteonecrosis of the knee; SONK = spontaneous osteonecrosis of the knee.

**Table 2**
Characteristics of SIFK and secondary ON compared.

| | SIFK | Secondary ON | Ref |
|---|---|---|---|
| Age (y) | >50 y | <45 y | [3] |
| Gender (M/F) | 1/3 | Underlying condition-dependent | [3] |
| Pain onset | Acute | Gradual | [3] |
| Laterality | Unilateral | Possibly bilateral | [3] |
| No. of lesions | Single | Multiple | [3] |
| Condylar involvement | >MFC | Multiple | [3,50] |
| Other joint involvement | No | Yes | [3,50] |
| Associated factors | Meniscectomy, meniscopathy | CCS use, smoking, alcohol, drugs, COVID-19, etc. | [3,45,46,50] |
| Pathologic findings | Trabecular fractures, fibrosis, callus | Bone infarction | [37] |
| MRI findings | Fractures visible as linear low-signal intensity on T1-weighted images in the subchondral layer and monocondylar bone marrow edema | Multicondylar geographic bone marrow changes, with a well-demarcated margin between necrotic and viable bone visible as “double-line sign” on T2-weighted images | [6,11,50] |

Abbreviations: CCS = corticosteroid; MFC = medial femoral condyle; MRI = magnetic resonance imaging; ON = osteonecrosis; PONK = post-arthroscopic osteonecrosis of the knee; SONK = spontaneous osteonecrosis of the knee.

usually responds to conservative treatment, but in later stages, surgery may be required to achieve satisfactory outcomes [29]. From a purely radiological standpoint, it is unclear when the injury becomes irreversible, as several risk factors influence prognosis. Among these, lesion size is considered the most important factor, though meniscal lesions, chondrosis, collapse, and bone edema progression should also be considered [33]. Given these considerations, what Ahlbäck described in 1968 was, in fact, SIFK misclassified as ON without any histological evidence. This systematic error was later confirmed histologically by several authors [9,30,39].

*Secondary knee ON*

Avascular ON of the knee is a distinct form of ON characterized by specific clinical and radiological features and it is invariably associated with various risk factors. It typically affects patients under the age of 45, with a gradual onset of pain. The condition is bilateral in over 80% of cases and often presents with multiple lesions, particularly in the femur. The affected areas commonly include the epiphysis, metaphysis, and sometimes the diaphysis, and it can involve multiple joints [6]. Histologically, the condition corresponds to tissue necrosis [3]. General risk factors include CCS use [40], alcohol consumption [41], smoking [42], chemotherapy, dialysis, organ transplantation, and a range of systemic diseases such as sickle cell anemia [43], Gaucher's disease, systemic lupus erythematosus [44], myeloproliferative disorders, dysphoric disorders [11], and COVID-19 [45,46]. CCS has been recognized as a major risk factor for knee ON. Evidence confirms a clear causal relationship between CCS exposure and the onset of bone necrosis. CCS-induced ON develops through multiple pathophysiological mechanisms, including osteoblast and osteocyte apoptosis, endothelial dysfunction, lipid metabolism disorders, and increased intraosseous pressure [47]. Importantly, high-dose and prolonged CCS therapy significantly increases the risk of ON [48]. However, cases have also been reported following short-term or relatively low-dose CCS administration, suggesting a relevant role of individual susceptibility. A dose-response relationship has been clearly demonstrated both in animal models and in clinical cohorts [49]. Histopathologically, osteonecrotic lesions are characterized by cellular death, bone marrow necrosis, bone matrix degradation, and inadequate reparative attempts, ultimately leading to subchondral collapse and joint failure.

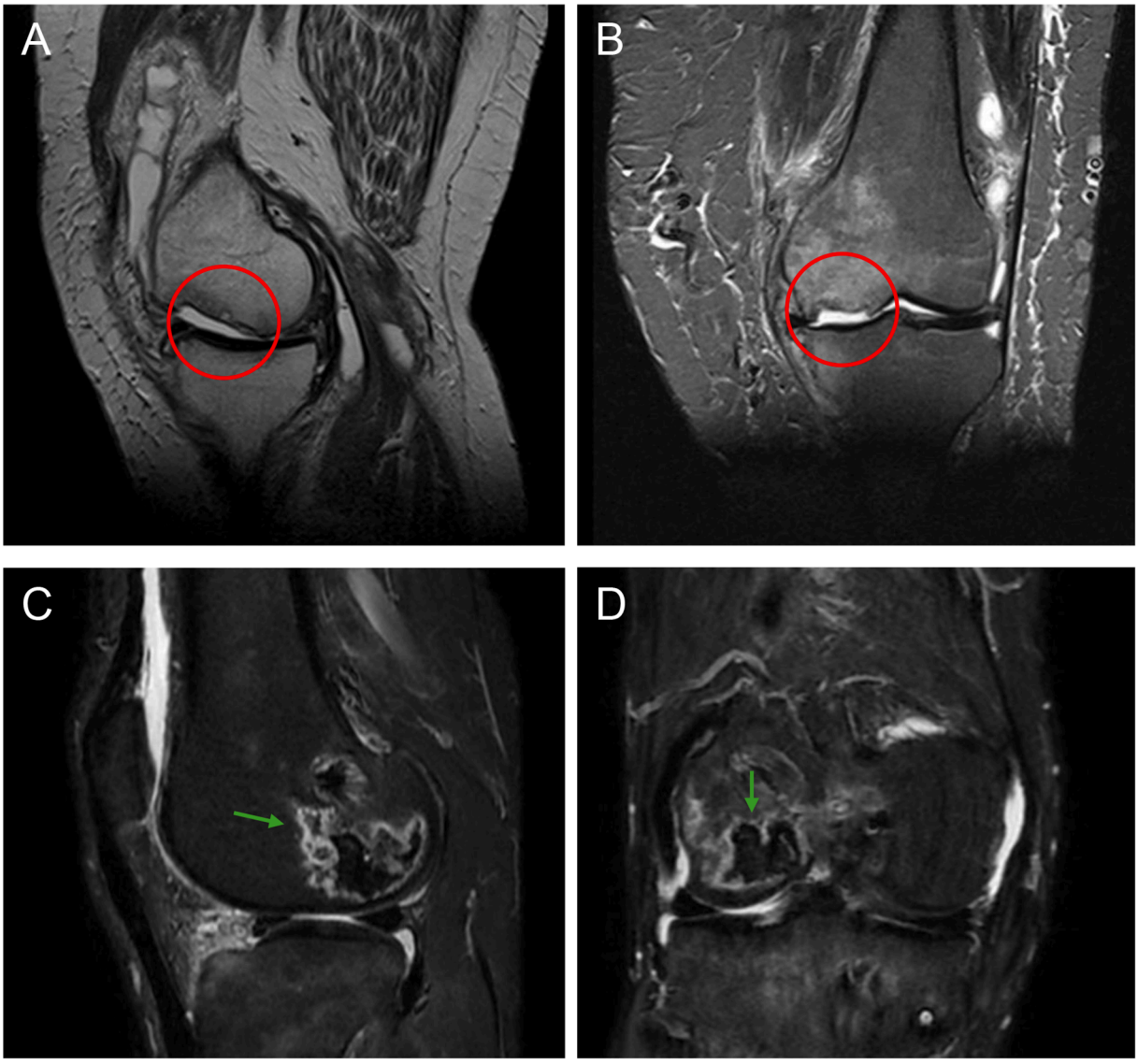


**Fig. 2.** Magnetic resonance imaging (MRI) characteristics of spontaneous osteonecrosis of the knee (SONK) and spontaneous insufficiency fractures of the knee (SIFK). Sagittal (A) and coronal (B) plane T2-wieighted MRI showing advanced SIFK of the medial femoral condyle (red circle). Sagittal (C) and (D) plane T2-wieighted MRI displaying the typical SONK's "double-line sign" (green arrow).

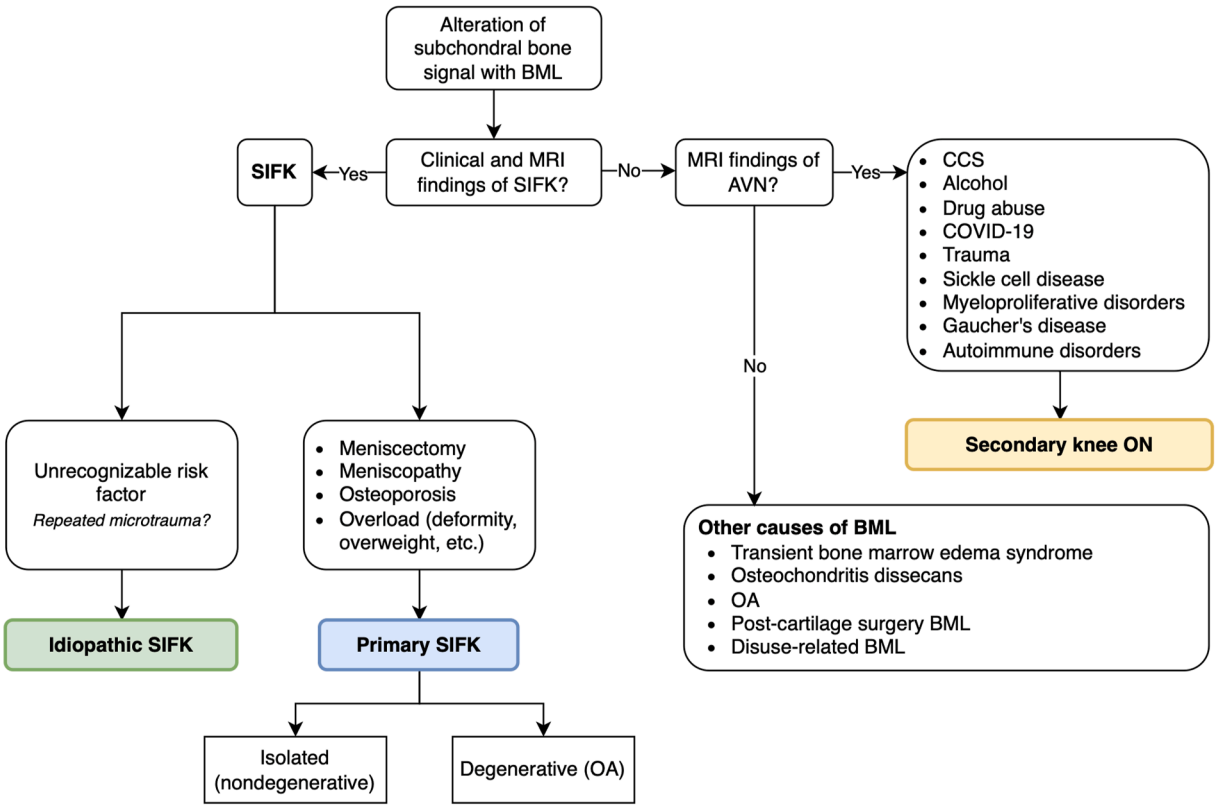


**Fig. 3.** New proposed classification of knee BMLs. Abbreviations: AVN = avascular necrosis; BMD = bone mineral density; BML = bone marrow lesions; CCS = corticosteroids; MRI = magnetic resonance imaging; OA = osteoarthritis; ON = osteonecrosis; SIFK = subchondral insufficiency fracture of the knee.

Imaging of avascular ON typically reveals necrotic lesions with a serpentine or "leopard-like" appearance. On T2-weighted MRI sequences, the necrotic lesion is surrounded by a distinctive "double-line sign," consisting of a clear rim with one low-signal line and one high-signal line [6]. The main characteristics of SIFK and secondary ON are shown in Table 2, showing substantial differences compared with SIFK from clinical, radiological, and histological standpoints (Fig. 2), further confirming the clear distinction between the two nosological entities and supporting the need for their differentiation and the use of distinct nomenclature.

Recently, with the onset of the COVID-19 pandemic, several cases of knee ON have been reported in patients with COVID-19 [45] with clinical and MRI features similar to those of a secondary necrosis rather than a SIFK. Notably, patients diagnosed with COVID-19 who received CCS therapy developed ON earlier and with lower doses of CCS than typically observed in patients without COVID-19 [46]. Additionally, some cases of ON occurred in COVID-19 patients who had not been prescribed CCS, suggesting a possible etiopathological role of the virus. Additionally, arthralgia and knee pain in COVID-19 patients often occur during the period commonly referred to as long COVID [46]. Given this potential relationship, COVID-19 should be considered a potential risk factor for avascular ON of the knee.

## DISCUSSION

When medical history, signs, and symptoms raise suspicion for SIFK or other types of BMLs, an MRI should be performed as soon as possible. MRI can identify the early stages of the disease and differentiate between SIFK and other BMLs [6,33,50]. If the MRI and medical history exclude SIFK and secondary ON, further investigation into other pathologies is necessary as part of the differential diagnosis. If MRI reveals signs of avascular necrosis, it is essential to identify and address any underlying risk factors to optimize treatment. Similarly, if SIFK is diagnosed, determining the associated risk factor is important. SIFK is classified as primary if at least one major risk factor is identified, or idiopathic if no trigger is recognized. SIFK can progress to advanced SIFK where the articular surface collapses, and subchondral fractures coexist. For both SIFK and secondary ON, initial treatment focuses on addressing modifiable risk factors and implementing appropriate therapy. In the early stages, conservative treatment—such as partial weight-bearing, bisphosphonates, pulsed electromagnetic field therapy, low-molecular-weight heparin, and supplementation with calcium, vitamin D, and vitamin C—has shown effectiveness [29,35]. In advanced stages, or if there is no clinical or radiographic improvement after three months of nonoperative management, surgical intervention is required [29,35]. This new approach is summarized in an algorithm proposing a new classification of BMLs (Fig. 3). According to this new framework, the term "SONK" has been removed due to the lack of histopathological evidence of necrosis. Similarly, "PONK" has been omitted as there are no differences between the previous descriptions of spontaneous and post-meniscectomy ON. Both conditions have been unified under the designation of SIFK. Secondary ON has been considered as a true avascular necrosis distinguished from SIFK; therefore, it was described separately.

## CONCLUSION

The traditional classification of knee ON, which identifies three distinct types, has led to confusion between SIFK and ON, resulting in inaccurate terminology. Based on current evidence, we have developed a new framework to clarify the distinctions between SIFK and ON. This renovated terminology could help better recognize the underlying pathophysiology and optimize treatment outcomes.

## Author contributions

PZ screened the literature and wrote the first draft of the manuscript. LA, SV, and AF contributed to the advanced draft of the study. SS, BZ, and RP revised the final draft of the manuscript and supervised the whole study. All authors read and approved the final manuscript.

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## Article summary

This review supports the notion that knee “osteonecrosis” would be better defined as subchondral insufficiency fractures (SIFK), calling for a reclassification of SONK & PONK for improved accuracy.

## Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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