REVIEW ARTICLE

The Role of Structural Deterioration and Biomechanical Changes of the Necrotic Lesion in Collapse Mechanism of Osteonecrosis of the Femoral Head

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Osteonecrosis of the femoral head (ONFH) is a crippling disease which is due to a lack of effective therapeutic measures. Its natural progression is rapid, the internal bone structure of the femoral head changes dramatically, and the subsequent fractures and collapse cause severe hip pain and loss of hip function. Femoral head collapse is a critical turning point in the development of ONFH and is related to the prognosis of patients. Early prevention and intervention help to preserve the hip joint and delay femoral head collapse. However, the mechanism of collapse still needs to be further studied because it is affected by different complex factors. This review discusses the underlying causes of femoral head collapse from two aspects: structural degradation and regional changes of biomechanical properties in the necrotic femoral head.

Key words: biomechanical changes; collapse mechanism; femoral head; structural deterioration; osteonecrosis

Background

Osteonecrosis of the femoral head (ONFH) is a common crippling disease, which leads patients to great suffering[1]. Two major categories of ONFH are traumatic and non-traumatic osteonecrosis. The former is mainly caused by femoral head neck fracture, the latter is mainly for use of corticosteroids and chronic alcohol overconsumption[2]. The main pathological features of ONFH are osteocyte death and bone marrow composition changes due to the damage or interruption of arterial blood supply and venous blood stasis. The repair process starts immediately to cure the necrotic lesion but without success. The result is that bone structural changes, fractures, and collapse cause severe hip pain and joint dysfunction, which seriously affect the patient's life quality[3,4].

ONFH usually progresses very quickly, and most affected femoral head will collapse within a few years if left untreated[5]. The occurrence of collapse not only brings severe hip pain but also greatly affects the prognosis of patients[6,7]. Femoral head collapse signifies a failure in biomechanical properties of subchondral bone and eventually leads to the dysfunction of the affected hip joint. That is when total hip

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arthroplasty (THA) is a reliable and last option to retrieve the function of hip joint[8–10]. THA can achieve a preferable outcome to relieve hip pain and maintain articular function, but it is not recommended for middle-aged and young patients owing to the limited operational life span of artificial joints and postoperative revision[11].

The surgical treatment of ONFH aims to retain the biological hip joint as much as possible[12]. It is now widely accepted that surgical operation performed at an early stage of ONFH can greatly improve the success rate of biological joint preservation[13–15]. It had been recently proposed that the peri-collapse stage, that was a continuous period in the development of ONFH from the appearance of subchondral fracture to the early collapse (<2 mm), was a key turning point for successful hip preservation treatment[16]. The prognosis of patients is determined by early detection and timely treatment to a great extent before femoral head collapse, so the collapse prediction and mechanism analysis have been the major issues considered by domestic and international scholars[17].

Femoral head collapse means the disease develops to a severe status, which determines the prognosis of patients[18]. This review discusses the mechanism of femoral head collapse and elaborates its underlying causes from two aspects: structural degradation and biomechanical changes of the necrotic femoral head.

Structural Deterioration in Necrotic Femoral Head

Structural Change in Necrotic Lesion

As shown in the Figure 1, in our previous study we found ONFH is an ischemic disease with a yellow wedge-shaped necrotic lesion of the femoral head. Necrotic lesion is often surrounded by an irregular repair reaction zone composed of granulation, fibrous tissue, and sclerotic cancellous bone. The bone trabeculae localized in the necrotic lesion were thinner, discrete, and disrupted[19]. Due to local blood supply blocking and incomplete repair process, the micro-architecture of the necrotic area changes dramatically during the progression of ONFH[20,21].

Disorganized bone tissue appeared in the necrotic area, which was intertwined or replaced by fibrous tissue[22]. It showed that osteocytes disappeared, calcified marrow appeared, carbonate-to-phosphate ratio increased, and phosphate-to-amide ratio decreased by using biophysical and ultra-structural analysis technology, which signified increased remodeling and reduced bone mineral density[23]. Structural degradation of cancellous bone was thought to be the initial factor of femoral head collapse[24]. Kawano et al. found that in the collapsed area, bone mineral density, trabecular thickness, and bone volume fraction were all significantly reduced compared to that in the nearby non-collapsed area[25].
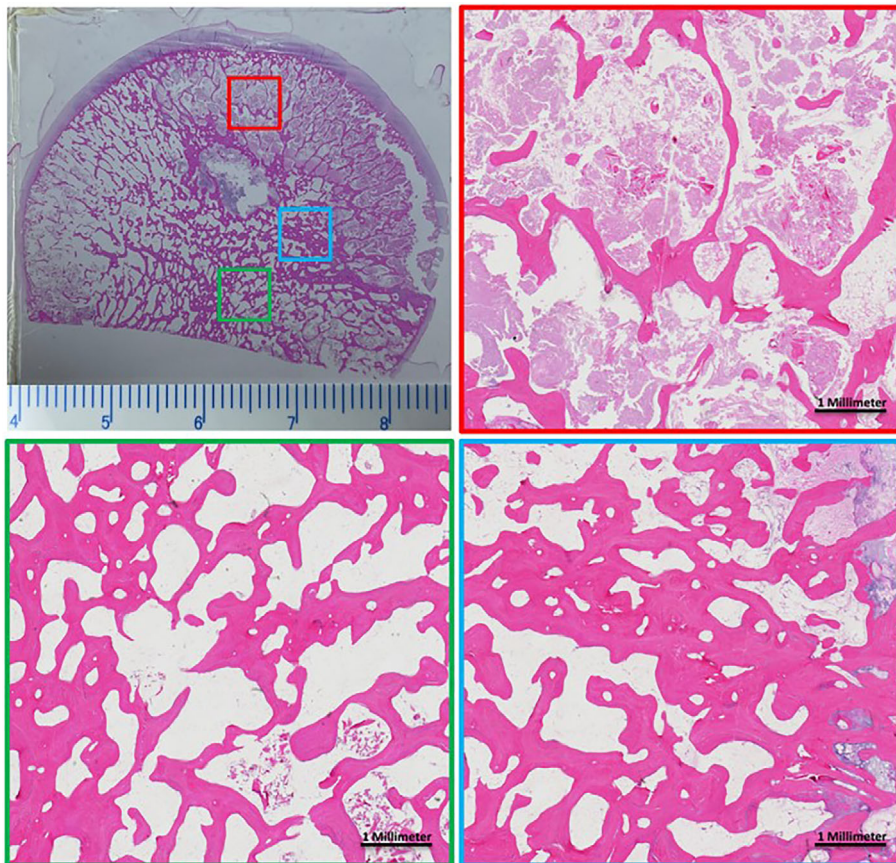

Fig. 1 Necrotic lesion is surrounded by an irregular repair area. The bone trabeculae localized in necrotic lesion are thinner, discrete, and disrupted. Red square: the necrotic area; Blue square: the sclerotic area; Green square: the normal area

In the early radiological stage, patchy osteoporosis, sclerosis, cysts, and crescent sign were observed in the necrotic area[26]. Crescent sign was considered to be the consequence of subchondral bone fracture and the beginning of femoral head collapse[3,27]. Fracture lines occur mostly in the subchondral area or in the deep necrotic portion adjacent to the necrotic-viable interface. In advanced collapse stages of ONFH, subchondral delamination, subchondral resorption, and chondral discontinuity were observed frequently[16]. With the time, the articular cartilage began to degenerate, causing irreversible hip damage[28].

Necrotic Lesion Size

Many studies have indicated that the collapse risk was affected by the size of necrotic lesion in ONFH[29,30]. A larger lesion indicates that the bone structure and biomechanical property are seriously damaged, which influences the disease's progression. Therefore, quantitative evaluation of the necrotic lesion is common in clinical practice. It is an important portion for collapse prediction and outcome[31,32].

At present, the commonly used clinical methods of measuring the size of necrosis include simple visual estimate and angular measurement. Visual estimate is subjective, and it is a rough estimated percentage of involved area compared with the area of entire femoral head by human vision[33,34]. Angular measurement has been considered more accurate than visual estimate, which mainly contains the combined necrotic angle, the index of necrosis, and weight-bearing value[35]. In previous studies, those measuring methods demonstrated that lesion size had a rough correlation with femoral head collapse rate, which is of clinical value. However, those measurements in two-dimensions cannot give the correct result of the real size of the irregular three-dimensional lesion. There is considerable variability between measuring methods in some cases, and it does not apply to scientific research.

Three-dimensional volumetric measurement (necrotic volume measurement) has previously been first described in the 1980s, and volumetric measurement using magnetic resonance image (MRI) provides a more precise assessment for the lesion size in ONFH[36–39]. MRI can sensitively predict the progression of early-stage osteonecrosis and accurately determined the volume of osteonecrosis both before and after collapse[40,41].

Ansari et al.[42] drew the necrotic area in outline in coronal planes of MRI, calculated the necrotic lesion/the entire femoral head area ratio in each slice, and finally determined the sum of the area ratio multiplied by the slice thickness to obtain the volumetric measurement. Their results showed that 90.6% of hips collapsed within 1 year in lesion volume greater than 25% of femoral head, while 31.3% of hips collapsed in lesion volume less than 25%. It seemed that the necrotic volume greater than 25% was the critical value for predicting femoral head collapse (Table 1). Zhao et al.[43] reconstructed the three-dimensional MRI of the femoral head by finite element analysis (FEA). When the necrotic lesion volume was more than 30%, femoral head had a high propensity for

TABLE 1 Three-dimensional volumetric measurements					
Authors	Year	Classification	Mean follow-up	Number of hips	Methods	Relationship between volume and collapse
Ansari et al.[42]	2020	ARCO stage I or II	12m	48	Divide necrotic area by the area of entire femoral head in 3-mm multiple coronal MR images and use the sum of these measurements multiplied by the thickness of MRI slices.	31.3% (5/16) hips collapsed in lesion volume < 25% as compared to 90.6% (29/32) of hips collapsed in lesion volume > 25%.
Bassounas et al.[32]	2007	—	7.8m (postoperative)	87	Tracing of the lesion area was performed using a semi-automatic image processing technique.	The lesion size of 63 hips with good clinical and radiological results was 24% ± 12%; The lesion size of 24 hips with poor outcome was 37% ± 9%.
Zhao et al.[43]	2005	ARCO Stage III or IV	—	38	Use an image-analysis program to outline the lesion area on each coronal MR image, then multiply the necrosis area by the slice thickness.	The collapse rate was as high as 80% when the lesion volume > 30%. If the volume was less than 30%, while necrotic areas occupied the anterolateral part of the femoral head, collapse referred to happen.
Nishii et al.[44]	2002	ARCO stage I or II	30m	65	Estimate the entire femoral head and the necrosis areas to obtain three-dimensional necrotic morphology.	One (6%) of the 16 hips with volume < 15% collapsed, eight (42%) of the 19 hips with volume between 15% and 30% collapsed, and 24 (80%) of the 30 hips with volume > 30%. Collapsed
Mazières et al.[31]	1997	Ficat stage II	24m (postoperative)	20	Calculate the areas of necrosis in relation to that of the femoral head on each frontal MRI slices, the sum of the percentage areas was necrotic volume.	seven of eight whose necrotic volume was <23% did not deteriorate; nine of 12 whose volume was >23% deteriorated and required THA.

TABLE 2 Japanese Investigation Committee (JIC) classification	
Type A	Lesions occupy the medial one-third or less of the weight-bearing portion.
Type B	Lesions occupy the medial two-thirds or less of the weight-bearing portion.
Type C1	Lesions occupy more than the medial two-thirds of the weight-bearing portion but do not extend laterally to the acetabular edge.
Type C2	Lesions occupy more than the medial two-thirds of the weight-bearing portion and extend laterally to the acetabular edge.

Necrotic lesions are classified into four types, based on their location on T1-weighted images or X-ray images.

collapse; if the volume was less than 30%, the location of necrosis was the main reason that affects the collapse.

The whole process of volumetric measurement is rather complicated, but modern imaging technology and computer tools make it easier to use. It is important to use the most accurate measurement techniques to explore the role of necrosis size in the progress of ONFH[45].

Necrotic Lesion Location

Alongside lesion size, lesion location is also involved in the development of femoral head collapse[44,46]. The necrotic lesion mostly occurs at the superior, medial, and anterior areas of the ischemic femoral head[32]. According to the Japanese Investigation Committee (JIC) classification system (Table 2), several studies had indicated that the lesion exceeding the medial 2/3 of the weight-bearing portion had a higher collapse rate and a faster progression of stage, and femoral head collapse occurred more often when the lesion extended laterally to the acetabular edge[47–51](Table 3). One explanation was that the changes of bone structure in weight-bearing portion reduced the stress transfer efficiency of principal compressive trabeculae after osteonecrosis and caused severe damage to the load-bearing capacity. Interestingly, if the necrotic lesion occupied less than the medial 2/3 of the weight-bearing portion and collapse was less than 2 mm, the potential for collapse cessation was great[44].

In addition, Sun et al.[52] emphasized that the lateral pillar was core to maintain the sphere of the femoral head. The better the lateral pillar was preserved, the lower the probability of collapse progress was. So effective mechanical support on the lateral pillar might delay the progress of collapse[53].

Necrotic lesion involving the anterior boundary of the femoral head was another important factor for the collapse progress. Previous reports indicated that if the lesion was small, its position, especially those located at the anterosuperior portion, need to be considered in the collapse prediction[33,44,54–57]. Kubo et al.[58] conducted research to explore the relation between collapse risk and necrotic lesion at anterior boundary. They found that the anterior necrotic angle, which was between the midline of femoral neck shaft and the line passing from femoral head center to anterior boundary of the necrotic lesion, was an independent correlate of collapse risk. Only one femoral head collapsed in 27 cases with anterior necrotic angle less than 79°. However, all cases with anterior necrotic angle greater than or equal to 79° developed to collapse, even though the lesion located on the medial boundary was usually classified as low collapse risk.

Cystic Lesion

Cystic lesion is a common pathologic feature in osteonecrosis and is related to local bone resorption[59]. By analyzing the three-dimensional distribution of cystic lesion, Liu et al.[60] found that the predilection locations of cystic lesion were mainly at the junction of the necrotic and viable areas and in the anterolateral part of the femoral head. Gao et al.[61] also observed that cystic lesion was often close to sclerosis rim; and there was a high incidence of microfracture, collapse, and crescent sign in patients with cystic lesion compared to that without cystic lesion. The authors further suggested two possibilities which need to be studied further: (1) cystic lesion destroyed the stress transfer path of internal femoral head, reducing the stress transfer efficiency of bone trabeculae; (2) cystic lesion near sclerosis rim broke off the support of sclerotic rim on cortical shell.

The formation of cystic lesion aggravates bone structural instability and plays a role in the progression of femoral

TABLE 3 Relationship between location of necrotic focus and collapse according to JIC classification										
		Number of hips					Collapsed hips and collapse rates, (n, %)			
Authors	Year	Total	A	B	C1	C2	Mean follow-up, yrs	A	B	C1	C2
Xin et al.[49]	2020	115	13 (A/B)		40	62	Initial diagnosis	1 (7.7) (A/B)		23 (57.5)	51 (82.3)
Kuroda et al.[48]	2019	505	21	34	173	277	Initial diagnosis	0 (0)	4 (11.8)	54 (31.2)	180 (65.0)
		212*	21	30	119	97	5	(0)	(7.9)	(36.6)	(84.8)
Takashima et al.[50]	2018	86	15	16	28	27	9	0 (0)	1 (6)	17 (61)	20 (78)
Min et al.[51]	2008	81	3	35	15	28	4.1	0 (0)	0 (0)	2 (13)	24 (86)

* 212 of 505 hips did not collapse hips at the initial diagnosis.

head collapse. Hamada et al.[62] found that all initial fracture cracks ran between separated bone resorption areas at the anterosuperior part of the femoral head in the early collapse stage (less than 3 mm of collapse). Baba et al.[63] investigated the bone resorptive areas by high-resolution computed tomography scan (CT). Their results showed that ONFH stage was independently related to cystic resorptive volume ratio, and high bone-resorptive volume ratio in the anterior femoral head related to collapse risk. Shi et al.[64] suggested that when initial bone resorption located in the lateral and anterior medial regions and its maximum area in coronal position was larger than 49mm^2, the femoral head progressed to collapse rapidly. Therefore, the occurrence of cystic changes should be paid attention in patients with early non-collapse osteonecrosis.

Activity of Osteoblast and Osteoclast Changes

The cellular level changes in the necrotic area were considered to be involved in collapse progression[65]. Osteonecrosis was local death of osteocytes and the component of bone marrow that was associated with blood supply damage or interruption from various factors[66,67]. It had been demonstrated that there was dysfunction of endothelial progenitor cells, which participated in vasculogenesis[68]. The dying cells will release endogenous inflammatory factors, leading to increased osteoclast activity and further tissue damage. The etiologies of ONFH also cause the functional decline of bone marrow cells and osteoblasts[69]. Corticoid administration and alcohol consumption were currently the most common causes of nontraumatic ONFH[70]. Studies found that corticosteroids could induce a vasoconstriction and increase the procoagulant factor production[71]. Corticosteroid use also increased osteocyte apoptosis, prolonged the osteoclast lifespan, and promoted differentiation of pre-adipocytes and mesenchymal stem cells to mature adipocytes[72–74]. Alcohol consumption altered the mesenchymal differentiation and reduced the ability to differentiate toward an osteoblastic lineage[75].

The normal repair process usually includes two parts: neonatal blood vessels grow into the necrotic area to bring new cells (osteoblasts derived from bone marrow mesenchymal stem cells and osteoclasts from mononuclear cells), then sequestrum is absorbed along with the formation of new bone[65,76]. Although the repair reaction occurs immediately after osteonecrosis, the repair process is usually limited or ineffective in ischemic femoral head, because the reparative fibrovascular tissues are difficult to grow inside the dead marrow space of the sequestrum[65,77]. The bone cells involved in bone formation have a low proliferative capacity, leading to a limited bone reparatory process[78]. Increased osteoclast activity in the necrotic area causes bone loss and structural weakening of bone trabeculae. It is worth noting that osteogenic activity exceeds osteoclast activity in the necrotic-viable interface, resulting in thickening and hardening of the trabecular bone, namely a sclerosis rim[79]. However, the neonatal bone trabeculae have weak mechanical properties and are prone to fractures under stress. Besides, osteoclast also involved in the

formation of fracture line in the deep necrotic area near the viable-necrotic interface in the later stage of ONFH. Li et al.[80] found that tartrate resistant acid phosphatase positive cells (osteoclasts) increased through the reparative interface where necrotic trabecular bone had disappeared in the late radiologic stages. Therefore, promoting vascular repair and regulating the osteoblast and osteoclast activity in the necrotic area may be an efficient approach in delaying disease progression[81].

Biomechanical Changes in Necrotic Femoral Head

Microstructural Changes in Necrotic Lesion

Bone microarchitecture is the main factor affecting bone strength, and its integrity determines bone mechanical properties[82,83]. Bone reconstruction after osteonecrosis has an impact on the bone microstructures in the necrotic lesion, and alters the bone mechanical properties gradually[84]. Wang et al.[85] performed nanoindentation experiments on single bone trabeculae and found that the nanomechanical performance (elastic modulus and degree of hardness) of single trabecular bone in the necrotic region showed no difference with that in healthy bone region. The authors suggested that the direct result for the degradation of mechanical performance and subsequent femoral head collapse was the structure changes of bone trabeculae in macrostructure. By comparing the biomechanical properties of the necrotic and the fractured femoral head, Ma et al.[21] found that the elastic modulus, the yield strength, and the ultimate strength of the necrotic area in the necrotic femoral head were lower when compared with the corresponding zone in the fractured femoral head. Furthermore, Zhang et al.[86] analyzed stress changes in different classifications of ONFH based on the JIC classification system. They found that the stress transfer path of type A was similar to the healthy level, while that of types B and C were broken. The damage of principal stress of type B was approximately 25% of the healthy level, and the types C1 and C2 was more than 50%. This finding indicated that the femoral head lost its bearing capacity when the lesion was large and located laterally.

The stress in the necrotic area also changes with bone structure. Wen et al.[87] virtually established five osteonecrosis FE models based on China-Japan Friendship Hospital (CJFH) classification (Table 4). They found that the peak von Mises stress and stress index of the necrotic areas in type L2 and L3 ONFH models were significantly higher than the critical value, making collapse risk increase significantly. Li et al.[88] found that, based on the finite element simulation, the maximum von Mises stress in the collapse group statistically increased compared to that in the non-collapse group. Discordance of internal structure and stress leads fragile trabeculae to fracture, and improving local bone strength may be an effective method to delay femoral head collapse.

The introduction of FEA provides new technical innovation in stress analysis of the femoral head. It is a potentially powerful non-destructive technique in the prediction of mechanical behavior. By reconstructing the femoral head

TABLE 4 China-Japan Friendship Hospital (CJFH) classification	
Type M	Lesions involve the medial pillar
Type C	Lesions involve both medial and central pillars
Type L1	Lesions involve three pillars but the partial lateral pillar is preserved
Type L2	Lesions involve the whole lateral pillar and partial central pillar
Type L3	Lesions involve three pillars including the cortical bone and marrow

CJFH classification is to divide the femoral head into three columns according to the coronal median plane of MRI or CT, namely, the lateral column (30%), the middle column (40%) and the medial column (30%).

model from the imaging data and assigning specific mechanical parameters, the stress changes of the femoral head is simulated *in vitro* under various conditions. The application of FEA helps better explore the role of stress in femoral head collapse.

Fatigue Fractures

Typically, femoral head collapse is secondary to subchondral fractures in ONFH. The early theory of collapse mechanism was that subchondral fractures were related to fatigue fractures of bone trabeculae[89,90]. Brown et al.[91] found that the stress level was markedly lower than normal at most points within the necrotic area through three-dimensional FEA. Although the static yield strength of necrotic cancellous bone was also below normal, the stress level was still substantially below the gross yield strength of necrotic bone in the necrotic area, which indicated that precipitous macrofractures were unlikely. Therefore, the authors emphasized that fatigue events played a decisive role in the collapse process. Fatigue fractures occurred even when external stress was lower than the yield strength of the trabecular bone[92,93].

The same conclusion was also drawn by Yang et al.[94]. They found that the maximum stress index value (effective stress/yield strength) of the normal femoral head was 0.1. This index of the necrotic femoral head was greater than 0.1, but never more than 1.0, which meant that the external stress would never exceed the yield strength of the trabecular bone. They also found that the area of stress index greater than 0.1 coincided with the common fracture sites which were the subchondral area and the deep necrotic area near the underlying necrotic-viable junctional area. In other words, fractures occur when the stress index of necrotic bone tissue is greater than 0.1. In the development of ONFH, local high stress continued to act on the necrotic and reconstructed bone trabeculae, causing material fatigue and microfractures[95]. Those microdamages gradually accumulated, leading to visible subchondral fractures and secondary collapse.

Stress Concentration in Sclerotic Rim

Sclerotic rim commonly presents around the necrotic lesion in ONFH, but its formation mechanism and function remain unclear[96,97]. Certain scholars proposed that the formation of sclerotic rim was a common phenomenon in the repair process of bone ischemia, while others proposed that it formed to adapt to the redistributed high stress around the necrotic-viable interface according to Huiskes' bone remodeling theory[98]. Besides, there is still no consensus of the impact of sclerotic rim on the development of femoral head collapse.

It has been hypothesized that the concentrated stress along the sclerotic area was the starting point of femoral head collapse. Karasuyama et al.[99] and Utsunomiya et al.[100] performed FEA using CT data of ONFH patients and found that stress was equivalently equally distributed on the femoral head surface in necrotic femoral head without sclerotic changes and collapse. On the contrary, stress was concentrated along the thickened bone trabeculae at the sclerotic boundary when sclerotic changes formed. They also found that the fractured area corresponded to the sclerotic boundary (the stress concentration area). Similarly, Motomura et al.[89] histologically observed that the collapsed femoral head inevitably involved the fractures around the necrotic-viable interface at the lateral boundary of the femoral head. Thus, subchondral fractures were correlated with stress concentration at the lateral sclerotic boundary.

However, studies pointed out that sclerotic rim around the necrotic tissue had a protective effect on femoral head collapse. The biomechanical properties of sclerotic rim strengthen in femoral heads markedly. The sclerotic changes help to enhance anti-deformation ability of the femoral head. It was found that the incidence rate of collapse in patients without sclerotic changes was much higher than that with a continuous sclerotic rim formed beneath subchondral bone. Yu et al.[101] found that there was a negative correlation between the proportion of proximal sclerotic rim and collapse rate. When the sclerotic proportion was <30%, the collapse risk was high, and effective mechanical support was recommended for the femoral head. Furthermore, Yu et al.[102] found that, by using FEA, increase in proximal rim sclerosis decreased total femoral head deformation and maximum principal stress in compression, which was an important factor for prevention of collapse.

The different morphology of sclerotic rim also affected the collapse rate by changing the stability of mechanical conduction[103]. When the edge of sclerotic rim was connected to the subchondral bone plate, it provided additional support to the subchondral plate. Chen et al.[97] found that closed sclerosis rim effectively reduced the original high stress of the subchondral bone, resulting in delayed collapse, while the sclerotic rim connecting along the upper edge to the lower edge of the spherical necrotic area could not stop femoral head collapse. Wu et al.[104] found that collapse risk was significantly higher in type transverse interface. Conversely, type "V" and type zigzag interface had a relatively low collapse risk. The authors explained that stress was dispersed in type "V" and type zigzag interface, but stress in type transverse interface was in the same direction, and sclerotic rim

below the necrotic area created a stress shelter effect, leading to local stress concentration.

Stress Changes in Cortical Bone

FEA indicated that the cortical bone played a prominent part in the bearing capacity of normal femoral head[87]. The strength and elastic modulus of the cortical bone were greatly higher than that of the cancellous bone, so the cortical bone bore more load than the cancellous bone[105]. This difference in mechanical properties was important. Volokh et al.[106] proposed that the local buckling of the cortical shell seemed to be a driving force of the progressive fracturing of the femoral head, leading to its entire collapse. During the development of ONFH, the critical buckling pressure of the cortical shell will decrease if the Young modulus of the cancellous bone decreases[106]. The deterioration of the cortical shell leads to further reduction of the critical buckling load as well. The result is that the cortical shell will bend, deform, and further deteriorate under load, resulting in massive collapse. If the buckling of the cortical shell initiates collapse, reinforcing the cortical shell with certain support materials may prevent and delay femoral head collapse.

Brown et al.[24] emphasized, however, that cortical bone might not play a major role in femoral head collapse. They proposed that the occurrence of collapse might be more strongly affected by the degree of structural degradation of the cancellous bone in the necrotic area than by the degradation of the cortical shell (subchondral plate). Through the simulation of finite element technology, it was found that the difference of principal stress distribution between the normal femoral head and the necrotic femoral head with a weakening subchondral plate was not significant, but the tendency for local structural failure was substantially higher in the femoral head with a weakening subchondral plate. Moreover, the stress level of necrotic lesion in the femoral head with normal subchondral plate was still over 70%, as high as that with a weakening subchondral plate, which indicated that the subchondral plate had only a rather modest protection effect on the underlying necrotic lesion. Thus, more attention should be focused on assessing the structural integrity of the cancellous bone.

Contact Stress on Femoral Head Surface

The hip joint is the biggest load bearing joint in the human body[107]. Contact stress on the femoral head surface is evenly distributed in a healthy hip joint. Nevertheless, stress will redistribute with the progress of osteonecrosis. Daniel et al.[108] investigated contact stress distribution in the articular surface using mathematical models. They found that the peak contact stress increased significantly if the load-bearing capacity of the necrotic lesion was decreased, if the size of the necrotic lesion was increased, and if the necrotic lesion was located more laterally. This explained why lesions with large size and lateral location easily led to fractures or collapse. According to the results of Krebs et al.[12] and Yoshida et al.[109], the total load was lower while the peak load was higher in the anterior hemisphere which continually switched between being covered and uncovered by the acetabulum during daily life. The higher stress in the anterior and lateral hemisphere causes fractures and collapse to occur more often here.

Conclusion

Femoral head collapse means that the disease has progressed to an advanced stage, directly relating to the life quality and prognosis of the patients. Although the concrete mechanism of collapse remains unclear, understanding the influencing factors of collapse is conducive to collapse prediction and guiding treatment.

The collapse means severe deterioration of the bone structure in the necrotic lesion and is the result of repeated actions of bone reconstruction and mechanical factors. In the necrotic area, the activity of osteoclast is increased and the activity of osteoblast is decreased, which leads to structural weakness of the necrotic bone and the formation of cystic change. Correspondingly, the mechanical properties of the necrotic bone decrease and the bearing capacity reduces, accelerating the development of subchondral fractures and subsequent collapse. This process is faster and more obvious if the necrotic area is large or in the specific location of the femoral head. Certainly, the repair reaction area around the necrotic lesion is also involved in the collapse progress, which provides mechanical support but causes local stress concentration. In brief, femoral head collapse is the result of the combined effect of many factors.

Conflict of Interests

The authors declare that they have no competing interests.

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