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Risk Factors for Avascular Necrosis After Closed Reduction for Developmental Dysplasia of the Hip

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Background: Avascular necrosis (AVN) is a major complication after closed reduction for developmental dysplasia of the hip. The factors that predispose to AVN remain controversial. The purpose of this study was to analyze the risk factors, especially patient factors, such as age at reduction, grade of dislocation, and ossific nucleus development, related to AVN.

Materials and Methods: We retrospectively reviewed children with dysplasia of the hip treated by closed reduction between 1997 and 2006. AVN was evaluated using Salter criteria and Kalamchi and MacEwen classification. Related factors were analyzed.

Results: One hundred and eight children (140 hips) with an average age of 16.6 months at closed reduction (range: 6-24 mo) were included in the study. For an average duration of 10.1 years (range 7–16 y) of follow-up, 44 hips (31.4%) developed AVN. Grade II or higher AVN occurred in 14 hips (10%). The incidence of AVN increased with the grade of dislocation ($P=0.022$) and underdevelopment of the ossific nucleus ($P<0.001$). Underdevelopment of the ossific nucleus was also found to be positively correlated with the dislocation grade ($P=0.047$). The age at the time of reduction, sex, and side were not significant factors. Children who underwent secondary operation were all older than 1 year at reduction.

Conclusions: High-grade dislocation correlates with the underdevelopment of the ossific nucleus. Patients with these 2 characteristics are predisposed to AVN. As underdevelopment of the ossific nucleus occurred regardless of age, it is not advisable to delay reduction because it does not alter the AVN rate, and instead, it increases the secondary operation rate.

Level of Evidence: Level IV case series.

Key Words: developmental dysplasia of the hip, avascular necrosis, closed reduction, ossific nucleus

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All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

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Avascular necrosis (AVN) is thought to be an iatrogenic phenomenon following the treatment of developmental dysplasia of the hip (DDH). It may lead to the deformation of the femoral head, joint incongruence, acetabular dysplasia, and consequently, early osteoarthrosis. Various incidence rates have been reported in the literature, ranging from 6% to 60%.[1–4] External causative factors for AVN are well studied. Immobilization in forced abduction is considered the foremost causative factor for AVN.[1,2] Preventive methods, such as human position casting, preliminary traction, and adductor tenotomy have been applied to decrease pressure to the femoral epiphysis and vasculature and to decrease the AVN rate.

However, the association of objective patient-related factors with AVN remain debatable. Controversy exists about whether the presence of ossific nucleus at the time of reduction protects against the development of AVN. For younger children, their unossified epiphyses are fragile and may be susceptible to severe AVN.[5] Clark advises that treatment should be intentionally delayed until the appearance of the ossific nucleus or until 13 months of age.[6] However, some authors have argued that the ossific nucleus did not protect for AVN, and that AVN is more commonly found among older patients in some papers. Moreover, delayed reduction increases the rate of a secondary procedure.[7] And the grade of dislocation becomes higher over time, which is also a risk factor for AVN.[8,9] To clarify the role of these patient-related factors is important for the preoperative evaluation of patients.

The purpose of this study was to (1) evaluate the incidence and severity of AVN after closed reduction and spica cast application for children with DDH, and (2) identify the risk factors, especially patient-related factors, associated with AVN. As AVN is believed to be caused by an increased pressure on the femoral head and tension on the surrounding vessels after hip reduction, we hypothesize that high-grade dislocation and underdevelopment of femoral epiphyses are the factors that predispose to AVN.

MATERIALS AND METHODS

After obtaining our hospital's institutional review board approval, we retrospectively reviewed the medical records and radiographs of patients who were diagnosed with DDH from 1997 to 2006 and treated by closed reduction and spica cast application at our hospital. The patients were aged between 6 months and 24 months at the time of treatment. In total, 223 patients were treated. Exclusion criteria included a diagnosis of teratologic hip dislocation, history of failure of closed reduction, open reduction as initial management, history of re-dislocation during immobilization, and inadequate radiographs and clinical records. The minimum duration of follow-up was 7 years after reduction, as most of AVN can be further sub-graded radiographically when patients approach adolescence.

One hundred eight patients (100 girls and 8 boys) had 140 hip dislocations and met the criteria for inclusion in the study. The left hip was involved in 50 patients, the right in 26 patients, and both hips in 32 patients. Mean age at treatment was 16.6 ± 3.6 months (range: 6-24 mo). The average length of follow-up was 10.1 years (7-16 y). Secondary surgeries for residual hip dysplasia or subluxation were performed in 25 hips (Table 1).

Closed reduction was performed under general anesthesia in all cases. We did not apply any type of traction before closed reduction and spica casting. Adductor longus was released by open approach to widen the hip's well-tolerated zone. In case of high-grade dislocation, the iliopsoas tendon was also released at the lesser trochanter simultaneously to decrease the tension of reduced hip. After stable closed reduction with more than a 25 degree safe zone, a so-called human position spica cast was applied with the hip in 90 to 100 degree flexion and no more than 70 degree abduction. Reduction was confirmed with intraoperative fluoroscopy. The hip was maintained in the spica cast for 3 months to stabilize the hip joint, then it was changed to a second abduction cast under general anesthesia. The second cast was a long leg broomstick cast with 30 degree hip abduction, which allowed hip flexion movement and promoted acetabular remodeling, lasting for another 3 months. After cast removal, patients were placed into a night time abduction brace for an additional 3 months. Patients had a radiographic follow-up annually to identify the residual dysplasia and AVN.

International Hip Dysplasia Institute (IHDI) classification was used to evaluate the preoperative dislocation grade.[10] Postoperative radiographs were scrutinized for the signs of AVN as described by Salter et al.[11] The hips that had necrosis were further sub-graded according to the criteria of Kalamchi and MacEwen:[5] For Grade I, changes affecting the ossific nucleus, which was mottled or displayed fragmentation, and the ossific nucleus mostly retained its spherical shape with minimal residual height loss or coxa magna; for Grade II, lateral physeal damage with coxa valga development; for Grade III, central physeal damage with prominent coxa brevis and magna; and for Grade IV, total damage to the head and physis. The development of the epiphyseal ossific nucleus was evaluated by measuring the epiphyseal ossific nucleus diameter to the neck diameter ratio (ENR) (Fig. 1), and the patients were classified into 3 groups as follows: those with ENR > 50%, those with ENR < 50%, and those with delayed un-ossification (absent ossific nuclei in patients older than 1 y). The age at the time of reduction was divided into 3 groups: < 1, 1 to <1.5, and 1.5 to 2 years.

Statistical analysis was performed using SPSS version 21.0 (IBM, Armonk, New York, USA). Data were presented as the mean (range) for continuous variables and as frequencies for categorical variables. Logistic multivariate regression was used for risk factor analysis, including gender, side, adductor and iliopsoas tenotomy, age at the time of reduction, dislocation grade, and development of the epiphyseal ossific nucleus. A χ^2 test was performed to examine the relationship between the factors. All tests were 2-sided, and a *P*-value <0.05 was considered significant.

TABLE 1. Demographic of Patients and General Results

Sex (patients)	Female 100; male 8
Side (patients)	Left 50; right 26; bilateral 32
Age at reduction	16.6 ± 3.6 mo (6-24 mo)
Duration of follow-up	10.1 y (7–16 y)
Secondary procedure	25 hips: pelvic osteotomy (salter 14 hips, pemberton 5 hips, triple 2 hips, periacetabular 3 hips, and shelf 1 hip) ± femoral osteotomy
AVN rate and grade*	31.4% (44 hips)
Grade I:	31 hips;
Grade II:	10 hips
Grade III:	2 hips
Grade IV:	1 hip

*Classified by Kalamchi and MacEwen criterion.
AVN indicates avascular necrosis.

RESUILTS

Using the Salter criteria for AVN, 44 of 140 (31.4%) hips developed AVN. Upon further classification using the Kalamchi and MacEwen classification system, most of AVN were Grade I (31hips ,70%), whereas Grade II or higher AVN occurred in 14 hips (10%). Among them, 10 hips (23%) were Grade II with varying degree of coxa valga that developed depending on the extent of necrosis and fragmentation of the ossific nucleus. Some cases of Grade II AVN were detected when the patient approached adolescence (Figs. 2, 3). Two hips were Grade III with obvious coxa brevis and magna (Fig. 4). One hip was Grade IV, in which damage to the entire physis resulted in severe femoral head deformity. Shelf osteotomy was performed at nine years of age (Table 1).

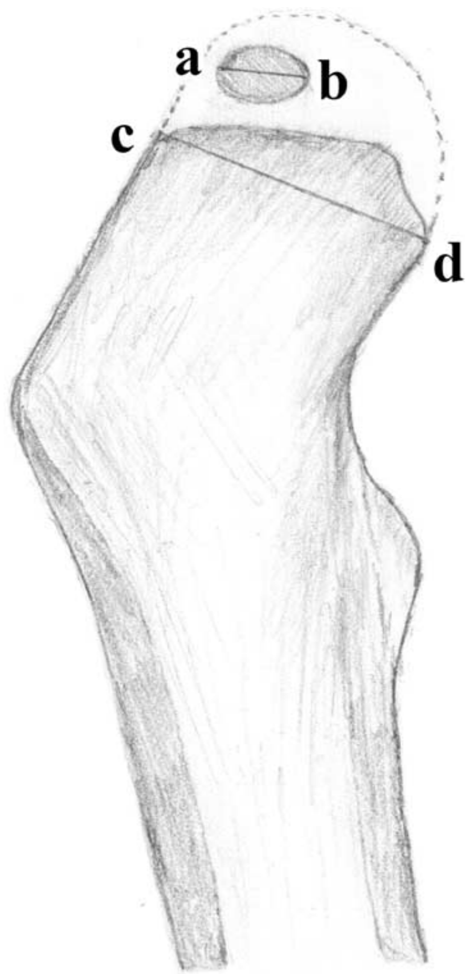

FIGURE 1. Ossific nucleus evaluation: the epiphyseal ossific nucleus diameter (A, B) to neck diameter (C, D) ratio.

Multivariate analyses showed that AVN was not related to sex ($P = 0.95$), side ($P = 0.42$), adductor and iliopsoas tenotomy ($P = 0.33$), and age at the time of reduction ($P > 0.99$). Whereas, the development of the epiphyseal ossific nucleus ($P < 0.001$) and dislocation grade ($P = 0.02$) were significantly correlated with AVN (Table 2).

AVN was found in 3.4% (2/58) of hips with an ENR > 50%, in 51% (35/68) of hips with an ENR <50% (including absent ossific nuclei in children younger than 1 year) and 50% (7/14) of hips with delayed un-ossification (in patients older than 1 y). Upon comparison of ossific nucleus development based on age at reduction, underdevelopment of the ossific nucleus (ENR < 50%) was evenly distributed across the various age groups (67%, 49%, and 45%, respectively) ($P = 0.345$) (Table 3).

In addition, 1, 0, 55, and 13 hips developed AVN out of 11 (9%), 18 (0%), 86 (36%), and 25 (48%) hips with grades I, II, III, and IV dislocations, respectively. The incidence of AVN rises with the grade of dislocation. Upon comparison of the age at reduction by the grade of dislocation, there is no statistically significant difference; however, for Grade IV dislocations, the patients were all more than 1-year-old.

The incidence of underdevelopment of the ossific nucleus was also positively correlated with the dislocation grade ($P = 0.047$).

Secondary procedures for hip dysplasia were performed for 25 hips (18%), involving patients who were all older than 1 year at the time of closed reduction. Hips with AVN had a higher rate of secondary procedures (12/44, 27%) compared with nonAVN hips (13/96, 14%), there was a trend toward a significant difference ($P = 0.059$) (Fig. 5).

DISCUSSION

AVN is a major complication after closed reduction for DDH. Surgeon-related factors, such as immobilization position, adductor tenotomy, and preliminary traction, are well studied. This large case series study demonstrates 2 objective factors that predisposes patients to AVN.

The reported prevalence of avascular necrosis varies widely and may depend on the methods of treatment and the criteria used to describe AVN. The treatment method is a principal factor that predisposes to AVN (as an iatrogenic complication). Malvitz et al[1] reported a 60% AVN rate in their case series. In their cases series, patients were immobilised in more than 60 degree abduction. And 45 degree internal rotation spica casts were sometimes used. A 21% AVN rate was reported by Gregosiewicz.[2] In his study, the 'frog-leg' position after reduction was found to be a risk factor for iatrogenic necrosis. Similar findings were reported by Schur,[4] who recommended that abduction in spica casts should be limited to 50 degree for children younger than 6 months of age. Conversely, Luhmann reported a case series with a very low AVN rate of 6% if mild coxa magna was excluded as AVN.[12] Abduction was limited to <60 degree in all their cases. In our cases, extreme position was avoided, and hips were immobilized in <70 degree abduction. Fourteen hips (10%) developed Grade II or greater AVN, which is similar to that reported in the literature in the last 30 years.[13]

Some treatment factors, apart from the immobilization position, have been suggested to reduce the incidence of AVN, including preliminary traction, gentle reduction under general anesthesia, and adductor tenotomy. Traction is performed to reduce the tightness of the hip musculature; however, its role in the reduction of AVN remains controversial. We did not use preliminary traction for high dislocation or hips with increased muscle tension after reduction; instead, we performed adductor tenotomy and iliopsoas tendon release. The AVN rate is comparable in the nontenotomy group and tenotomy group.

Patient-related factors are objective factors that may make hips susceptible to AVN and includes the grade of dislocation, age at reduction, and development of the

FIGURE 2. Type II avascular necrosis with minimal residue deformity. A, Anteroposterior left hip radiograph of a 18-month-old girl with grade 3 dislocation, epiphyseal ossific nucleus diameter to neck diameter ratio smaller than 50%; B, 1 year after closed reduction, mild avascular necrosis showing as failed ossification of the lateral portion of the epiphysis; C, 9-year-old, lateral epiphysis notching with normal femoral neck length; D, 11-year-old, premature closure of the lateral physis was identified (arrow); E, At age of 12-year-old; F, 14-year-old, residue shorted femoral neck length with good acetabular coverage.

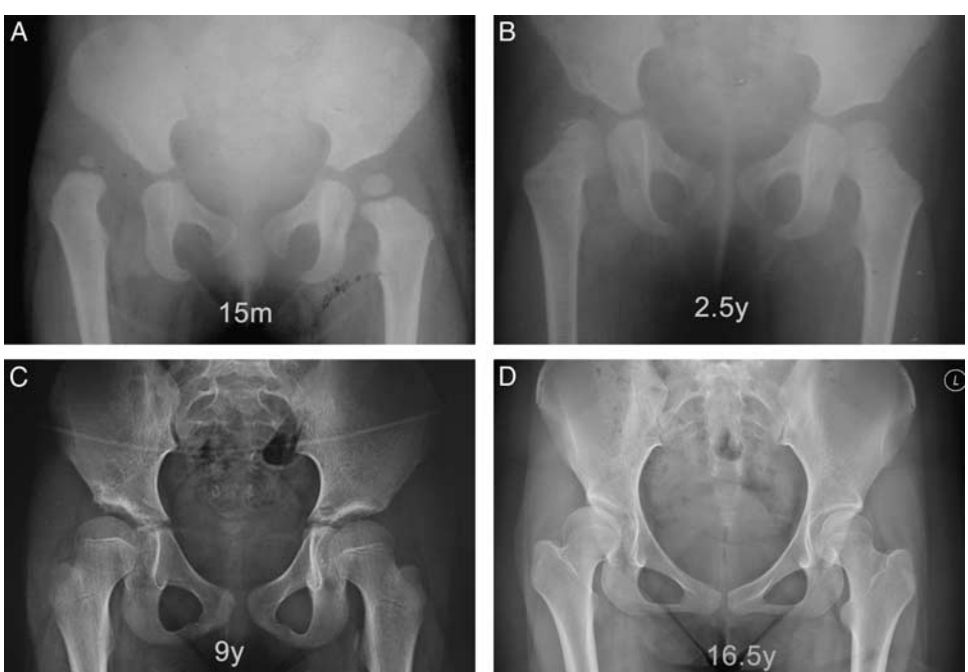

FIGURE 3. Type II avascular necrosis with residue deformity. A, Radiograph of the pelvis of a 15-month-old girl with right side developmental dysplasia of the hip, grade 3 dislocation, epiphyseal ossific nucleus diameter to neck diameter ratio smaller than 50%; B, 1 year after closed reduction, there was partial fragmentation of the lateral ossific nucleus; C, 9-year-old, type II avascular necrosis was confirmed with mild coxa valga deformity and acetabular coverage was inadequate; D, 16-year-old, there was coxa valga and coxa brevis deformity with acetabular dysplasia.

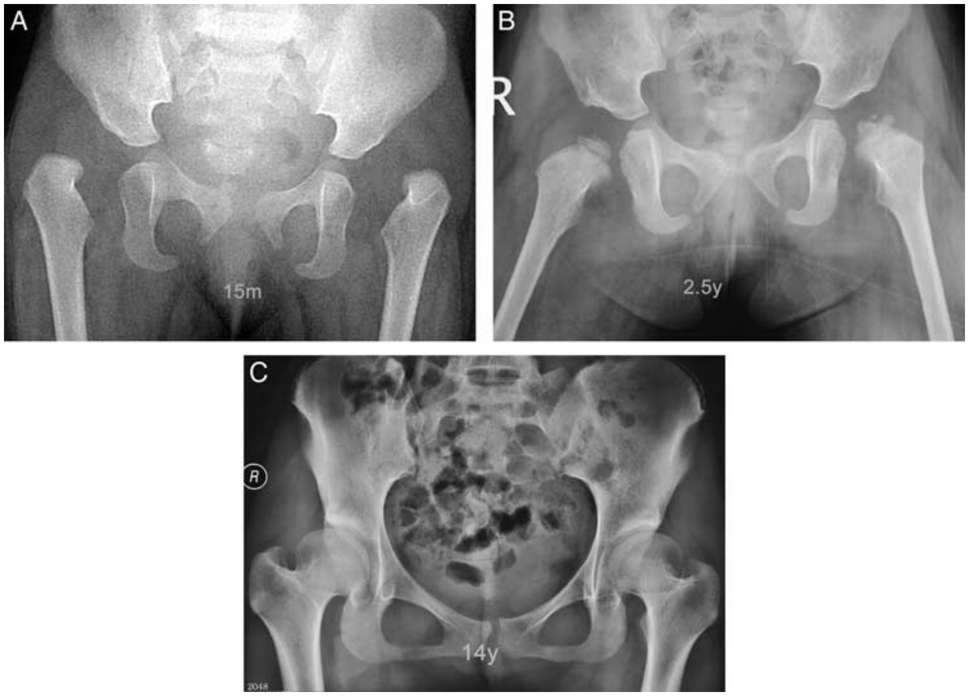

FIGURE 4. Type III avascular necrosis. A, Radiograph of the pelvis of a 15-month-old girl with bilateral developmental dysplasia of the hip with grade 3 dislocation, bilateral nuclei were absent; B, 1 year after closed reduction, there was bilateral fragmentation involving the entire ossific nucleus; C, 14-year-old, type III avascular necrosis with coxa magna and brevis, acetabular dysplasia.

ossific nucleus. Their roles in AVN development were controversial in previous studies.

Sibiński et al[8] found that the degree of initial dislocation was a significant risk factor for AVN. Similar findings were reported by works of Bozkurt et al, who found that higher preoperative dislocation grade (IHDI classification) was a significant risk factor for AVN in patients older than 10 months,[9] and by the work of Wu et al.[14] Preliminary traction was not used in both studies, which may magnify the role of the dislocation grade in AVN development. Our study shows similar results.

Results of previous studies on the effect of age at reduction and development of the ossific nucleus on AVN are more controversial. Some studies indicate that femoral heads without an ossific nucleus were susceptible to necrosis.[15] Anatomically, the microcirculation of the chondroepiphysis of the capital femoral epiphysis is supplied by a fragile end-arteriole. By the time the secondary center of ossification becomes radiographically visible at 5–7 months, major vascular changes would have occurred.[16] An effective collateral circulation is established as the ossific nucleus develops, providing further resistance of the capital femoral epiphysis to ischemic injury.[17] Kalamchi and MacEwen noted that the most severe forms of AVN are observed in children who were younger than 6 months old when treated.[5] Clarke proposed that treatment for DDH should be delayed until the appearance of the ossific nucleus to decrease the risk of compressive ischemic injury to the femoral head during reduction.[6] However, Luhmann did not detect any significant difference in the prevalence of AVN between patients who had an intact ossific nucleus at the time of

TABLE 2. Logistic Multivariate Regression Analysis of Avascular Necrosis Related Factors

Factors	AVN (−)	AVN (+)	AVN rate (%)	*P*-value
Age at reduction	—	—	—	> 0.99
< 1 y	8	4	33	—
1-1.5 y	50	24	32	—
1.5-2 y	38	16	30	—
Sex				0.945
Male	9	3	25	—
Female	87	41	32	—
Side				0.420
Left	57	25	30	—
Right	39	19	33	—
Development of ossific nucleus				< 0.001
ENR > 50%	56	2	3.4	—
ENR < 50%	33	35	51	—
Delayed ossified	7	7	50	—
Grade of dislocation	—	—	—	0.022
1	10	1	9.1	—
2	18	0	0	—
3	55	31	36	—
4	13	12	48	—
Adductor & iliopsoas tenotomy				0.330
Yes	89	40	31	—
No	7	4	36	—

AVN indicates avascular necrosis; ENR, epiphyseal ossific nucleus diameter to neck diameter ratio.

TABLE 3. Relationship Between Development of the Ossific Nucleus and Different Age Groups

	Number of hips	ENR > 50% (%)	ENR < 50% (%)	Delayed un-ossification (%)	Total (%)
< 1 y	12	4 (33)	8 (67)	0	100
1-1.5 y	74	28 (38)	36 (49)	10 (13)	100
1.5-2 y	54	26 (48)	24 (45)	4 (7)	100
P-value			$P = 0.345$		

reduction and those who did not.[12] Those results were also supported by the work of Roposch. He found the ossific nucleus did not protect for AVN,[18] only moderate evidence showed that the presence of the ossific nucleus was protective against the development of more severe forms of AVN in his meta analysis.[19] Absence of ossific nucleus can be part of a normal growth stage as an infant, but it may also signify growth retardancy, if it occurs in patients older than 6 months. Yilar S et al found that the presence of ossific nucleus is protective against AVN after the sixth month, however, the presence of ossific nucleus does not provide extra protection against AVN in before sixth month.[20]

Tonnis also supported the protective role of the normal development ossific nucleus. He found that necrosis occurred in 0.9% and 4.5% of cases with normally developed nucleus and missing nucleus (or small for age patients), respectively. The incidence rose to 12.5% when the appearance of the ossific nucleus was delayed (in patients older than 8 mo).[21] C Bozkurt et al found that the delayed un-ossification (older than 10 mo), other than absence of ossific nucleus, was a significant risk factor for AVN.[9] Similar findings were reported by our study and are consistent with Tonnis' and Bozkurt's studies. We measured the ENR and classified the patients into 3 groups: ENR > 50%, ENR < 50%, and delayed un-ossification (in patients older than 1 y). The incidence of AVN was 3.4% (2 hips in 58), 51% (35 hips in 68), 50% (7 hips in 14), respectively. These results indicate that the vulnerability of the femoral head is greater when it is underdeveloped. However, the age at reduction was not a significant factor for the occurrence of AVN. Moreover, upon analyzing the ossific nucleus development by age at reduction, underdevelopment of the ossific nucleus (ENR < 50%) was evenly distributed across age groups. These findings suggest a certain vulnerability of the immature ossific nucleus regardless of age.

Weicket suggests that a 'delayed nucleus development' may explain the retarded nucleus development, which is said to exist when the development of the femoral head is

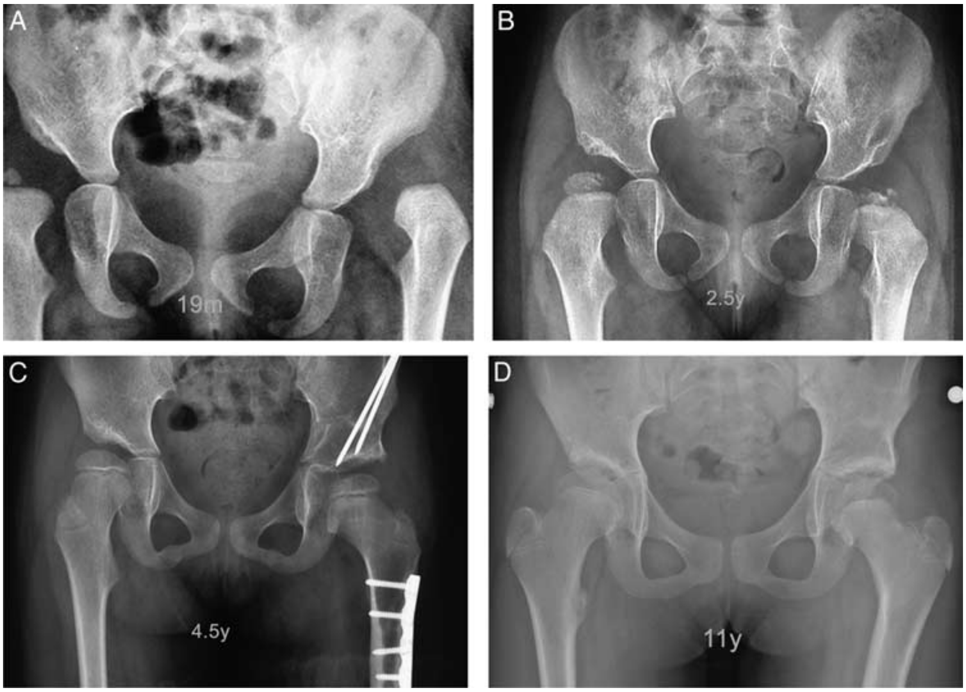

FIGURE 5. A, Radiograph of the pelvis of a 19-month-old girl with bilateral developmental dysplasia of the hip, left sided grade 4 dislocation with absent ossific nucleus, right sided grade 3 dislocation with small ossific nucleus, epiphyseal ossific nucleus diameter to neck diameter ratio smaller than 50%; B, 1 year after closed reduction, there was fragmentation of the bilateral ossific nuclei; C, 4.5-year-old, Salter osteotomy and femoral osteotomy were performed for hip dysplasia; D, 11-year-old, there was right sided type I avascular necrosis without femoral head deformity, and left sided type II avascular necrosis with mild coxa valga deformity, the hip was maintained with good coverage.

disturbed from the outset. It is an endogenous condition and constructive disturbance; here, the femoral head lacks an ossific nucleus and hence, becomes irregular in shape and structure or exhibits a great delay in appearance before reduction.[22] Furthermore, our study demonstrates one of the possible causes of underdevelopment of the ossific nucleus. In high dislocation, the femoral head bears less pressure, however, the blood vessels are under more tension. Both factors may lead to a retarded nucleus. The higher the dislocation, the smaller the ossific nucleus.

The limitations of our study are as follows: First, it was a retrospective study involving multiple surgeons. Differences in surgical technique may exist. Second, other objective factors related to AVN may exist. For example, we did not record if there was general ligamentous laxity in the patients, which may relate to AVN and therefore, needs further research.

In conclusion, this study demonstrates that patients with an underdeveloped ossific nucleus and high-grade dislocation are vulnerable to AVN. These 2 factors are also positively correlated. As AVN was not influenced by age at reduction, and the underdevelopment of the ossific nucleus was evenly distributed across age groups, it is not advisable to delay reduction, as it also increases the rate of secondary procedures.

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