Int J Clin Exp Med 2020;13(2):823-829
www.ijcem.com /ISSN:1940-5901/IJCEM0104011

Original Article

Analysis of early imaging prediction signs of type 2 avascular necrosis of the femoral head after DDH treatment

Yihua Ge, Zhigang Wang

Department of Orthopaedics, Shanghai Children's Medical Center, Shanghai Jiao Tong University School of Medicine, Shanghai, China

Received October 25, 2019; Accepted November 21, 2019; Epub February 15, 2020; Published February 28, 2020

Abstract: Objective: This study aimed to analyze the early imaging predictive signs of type 2 avascular necrosis of the femoral head after treatment of developmental dysplasia of hip (DDH). Methods: The 5-year follow-up data of DDH patients treated in our hospital were collected. Among them, 25 cases with type 2 avascular necrosis of the femoral head were taken as the case group, and 30 cases diagnosed with DDH without type 2 avascular necrosis of the femoral head were taken as the control group. The differences in early imaging signs between the two groups were analyzed. Results: The incidence of early influencing signs in the case group was significantly higher than that in the control group (P<0.001), and the incidence of the two types of early imaging signs (distortion of lateral epiphyseal plate and formation of osteopontia) in the case group was significantly higher than that in the control group (P<0.001). Further study on the 25 cases of children in the case group found that the incidence rate of type 2 signs was the highest and had differences with type 3 (P<0.001). The incidence rate of the type 4 signs was the lowest and had differences with type 1 and 2 (P<0.05). The binary logistic regression analysis showed that there was a significant correlation of avascular necrosis of the femoral head in children with distortion of the lateral epiphyseal plate and formation of a bone bridge (OR=9.821, P=0.002). Conclusion: The incidence rate of early imaging signs of avascular necrosis of the femoral head in DDH in children is high, of which the distortion of the lateral epiphyseal plate and the formation and change of a bone bridge are the most common, suggesting that early imaging changes canalert to the occurrence of type 2 avascular necrosis of the femoral head.

Keywords: DDH, type 2 avascular necrosis of femoral head, imaging signs, predictive value

Introduction

Developmental dysplasia of the hip (DDH) is common in children's orthopaedic diseases. Clinically, it is seen that there is a dynamic change with an aggravating or improving trend of growth and development, and the clinical incidence rate is 1-10‰ [1, 2]. At present, research on DDH shows that early diagnosis and treatment can significantly reduce the operation rates and disability rates [3-5]. Imaging plays an extremely important role in early screening and dynamic observation of DDH [6]. Once confirmed, appropriate observation and regular follow-ups should be actively carried out. DDH treatment is usually different according to age. Conservative treatment schemes are often used within 18 months of age. However, patients older than 18 months often have surgical treatment for intervention [7, 8]. However, due to the lack of specific clinical signs in early DDH, most children often only see a doctor only after gait abnormalities are found. Delayed diagnosis results in children missing the optimal conservative intervention treatment time [3], and some children's families give up early intervention treatment, resulting in irreversible degeneration of the hip joint and changes in anatomical position of hip joint. Finally, DDH treatment is more difficult and increases the incidence of disability [9, 10]. There are many surgical treatment methods for DDH in children [11, 12], but the incidence of postoperative hemorrhage, infection, avascular necrosis of the femoral head and subluxation is significantly increased due to the large damage of soft tissue and surrounding tissues of the hip joint by surgery [13]. Among them, avascular

necrosis of the femoral head is the most serious, and avascular necrosis of the femoral head is an unavoidable complication in DDH treatment process, which will seriously affect the prognosis of the children [14]. Among the various types of avascular necrosis of the femoral head, kalamchi-macewen type 2 has the highest incidence rate. This type of avascular necrosis of the femoral head involves the lateral epiphyseal plate of the femoral head, causing growth a disorder of the proximal femur. As the children get older, it will eventually lead to coxa varus deformity, poor coverage of the head socket and early degeneration of joints. Unfortunately, this has the highest incidence and a huge impact on complications, but it is often not found early after DDH treatment [15]. In Kalamchi & MacEwen's literature [16] some imaging early signs of type 2 avascular necrosis of the femoral head were proposed, including widening and calcification of the lateral epiphysis, distortion and deformation of the lateral epiphyseal plate, lateral defect of epiphyseal, lateral cystic degeneration of metaphyseal, etc., but no detailed retrospective analysis was made on the specific occurrence of these early signs. Based on this, this study conducted a long-term treatment and follow-up of DDH children for more than 5 years to observe whether the above-mentioned early signs are related to the occurrence of avascular necrosis of the femoral head. The study is reported as follows.

Materials and methods

Materials

A case-control study method was used to collect the imaging data of DDH-diagnosed [17] cases treated in our hospital from January 2010 to June 2014. Inclusion criteria were as follows: (1) Unilateral DDH in children; (2) The research data were complete, including pelvic orthographic films before and at the last follow-up; (3) The follow-up period was not less than 5 years. Exclusion criteria were as follows: DDH in children caused by related diseases, such as cerebral palsy, spinal cord herniation, tethered cord syndrome, systemic multi-joint contracture and other neuromuscular system diseases. Twenty-five patients with type 2 Kalamchi & MacEwen avascular necrosis of the femoral head after closed reduction or simple open reduction or Salter pelvic osteotomy + open reduction were collected as the case group, and 30 patients diagnosed with DDH5 with the same treatment plan as the case group but without type 2 avascular necrosis of the femoral head were taken as the control group, including 19 males and 36 females, 36 cases on the left and 19 cases on the right. The age of first visit was 15.1±4.6 months, with an average follow-up of 84.3±24.8 months.

Methods

The basic information of this group of cases was collected, including sex, age, hip joint involvement, occurrence of ossification at the center of the femoral head, initial treatment and classification by International Hip Dysplasia Institute (IHDI) [18]. The imaging data of 12-24 months after closed reduction or simple open reduction or Salter's pelvic osteotomy + open reduction were reviewed to find the early imaging signs of avascular necrosis of the femoral head proposed by kalamchi & MacEwen's classic literature, including: 1) widening and calcification of the lateral epiphysis, 2) distortion of the lateral epiphyseal plate, bone bridge formation, 3) lateral epiphyseal defect, and 4) lateral cystic change of the metaphyseal end. The occurrence of these imaging early signs in each case, the total occurrence rate of these early signs, and the occurrence rate of type 4 of early signs were recorded and statistically analyzed.

Statistical methods

SPSS 22.0 statistical software was used. Continuous variables were expressed by mean ± standard deviation (X±SD). Paired t-test was used for data with normal distribution and variance homogeneity, and rank sum test was used for non-consistency with normal distribution and variance homogeneity. Counting data was expressed as % and Pearson chi-square test was applied. The risk factors of femoral head necrosis were analyzed by binary logistic regression. A P value less than 0.05 was regarded as statistical significance.

Results

Analysis of general data of children

There was no statistical difference between the two groups in gender, age at first visit, average follow-up time, treatment method after diagnosis, lesion site, occurrence of ossification at the center of the femoral head at the first visit, and

Table 1. Analysis of general data of children

Term	Case group (n=25)	Control group (n=30)	X^2/t	P
Gender (male:female)	7:18	12:18	0.868	0.351
Age at first visit (months)	15.0±4.6	15.2±4.8	0.163	0.871
Treatment methodafter diagnosis			0.005	0.997
Closed reduction	10 (40.00)	12 (40.00)		
Simple open reduction	11 (44.00)	13 (43.33)		
Salter pelvic osteotomy + open reduction	4 (16.00)	5 (16.67)		
Lesion site			0.868	0.351
Left side	18 (72.00)	18 (60.00)		
Right side	7 (28.00)	12 (40.00)		
Occurrence of ossification center of femoral head at first visit	21/25 (84.00)	22/30 (73.33)	1.833	0.176
IHDI classification			2.984	0.394
I	1 (4.00)	4 (4.00)		
II	3 (12.00)	6 (12.00)		
III	8 (32.00)	10 (32.00)		
IV	13 (52.00)	10 (52.00)		

Table 2. Occurrence of early imaging signs

Term	Case group (n=25)	Control group (n=30)	X^2/t	P
Number of children	23/25 (92.00)	12/30 (40.00)	15.934	<0.001
Early imaging signs				
Type 1 (widening and calcification of lateral epiphysis)	7/25 (28.00)***,#	4/30 (13.33)	1.833	0.176
Type 2 (distortion of lateral epiphyseal plate and formation of bone bridge)	21/25 (84.00)###	9/30 (30.00)	16.038	<0.001
Type 3 (lateral epiphyseal defect)	3/25 (12.00)***	2/30 (6.67)	0.469	0.493
Type 4 (lateral cystic change of metaphysis)	1/25 (4.00)***	1/30 (3.33)	0.017	0.895

Note: Compared with Type 2, ***P<0.001; compared with Type 4, #P<0.05, ###P<0.001.

IHDI classification before treatment (P>0.05), as shown in **Table 1**.

Occurrence of early imaging signs

The incidence of early influencing signs in the case group was significantly higher than that in the control group (P<0.001), and the incidence of type 2 early imaging signs (distortion of the lateral epiphyseal plate and formation of osteopontia) in the case group was significantly higher than that in the control group (P<0.001). Further study on the 25 cases of children found that 23 of them showed early imaging signs in the 12-24 months of follow-up, with an incidence rate of 92.00%. A total of 32 early imaging signs were found in 23 children, of which 2 children had 3 signs at the same time, 4 children had 2 signs at the same time, and the remaining 18 children had 1 sign. Among the type 4 imaging signs, the incidence of type 2 signs (distortion of the lateral epiphyseal plate and formation of a bone bridge) was the highest, accounting for 84% (21/25 hips), the incidence of type 1 signs (widening and calcification of the lateral epiphysis) was 28% (7/25 hips), the incidence of type 3 signs (lateral epiphyseal defect) was 12% (3/25 hips), and the incidence of type 4 signs (lateral cystic change of the metaphysis) was the lowest, accounting for 4% (1/25 hips). Among them, the incidence rate of the type 2 signs was the highest and the incidence rates of the other 3 types of signs were statistically different (P<0.001). The incidence rate of the type 4 signs was the lowest and the incidence rates of the type 1 and 2 were statistically different (P<0.05). See **Table 2**; **Figures 1-3**.

Risk factor analysis of avascular necrosis of the femoral head

The data of 55 children with DDH were analyzed. Avascular necrosis of the femoral head was taken as the dependent variable (no occurrence =0, occurrence =1). The related factors

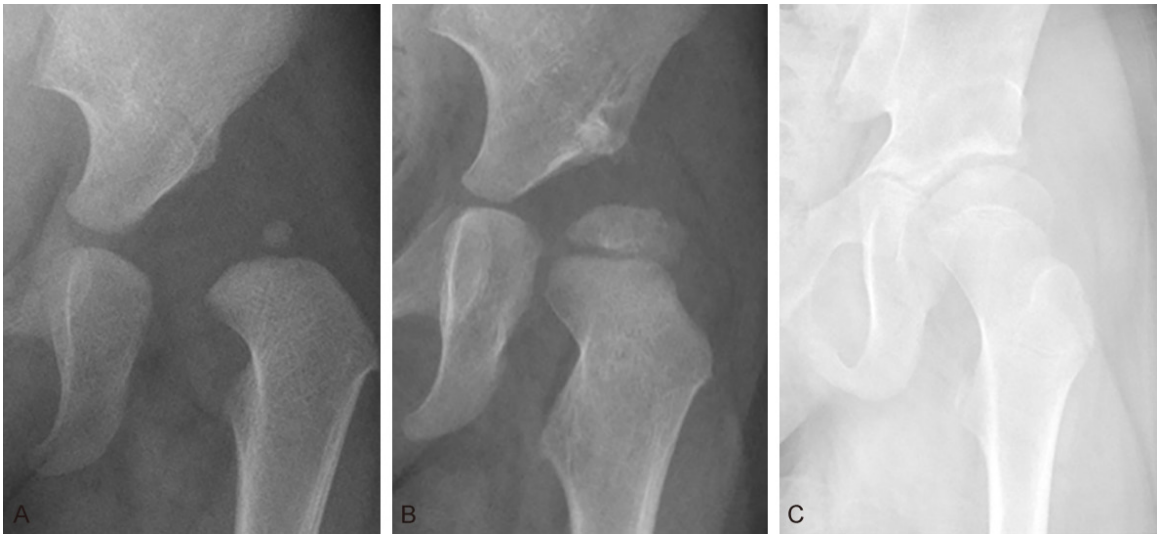

Figure 1. Imaging images of children with type 1 (widening and calcification of lateral epiphysis).

Figure 2. Imaging images of children with type 2 (distortion of lateral epiphyseal plate and formation of bone bridge).

included widening calcification of the lateral epiphysis (no occurrence =0, occurrence =1), distortion of the lateral epiphysis plate, formation of a bone bridge (no occurrence =0, occurrence =1), lateral defect of the epiphysis (no occurrence =0, occurrence =1), lateral cystic degeneration of the metaphysis (no occurrence =0, occurrence =1) as independent variables. The binary logistic regression analysis showed that the dependent variable avascular necrosis of the femoral head in children was significantly correlated with the distortion of the lateral epiphyseal plate and the formation of osteopontia (P=0.002) in independent variable patients. There was no significant correlation among widening calcification of the lateral epiphyseal, lateral epiphyseal defect and lateral epiphyseal cyst (P>0.05) (**Table 3**).

Discussion

Previous studies have found that DDH occurs in 3-4 of 1,000 live births, and the severity of the disease is closely related to the natural progression of DDH. Early detection of mild dysplasia of the hip may lead to clinical symptoms later in adulthood as the disease progresses slowly, while early findings of severe hip dysplasia may show symptoms in infancy. In addition, differences in limb length may occur for chil-

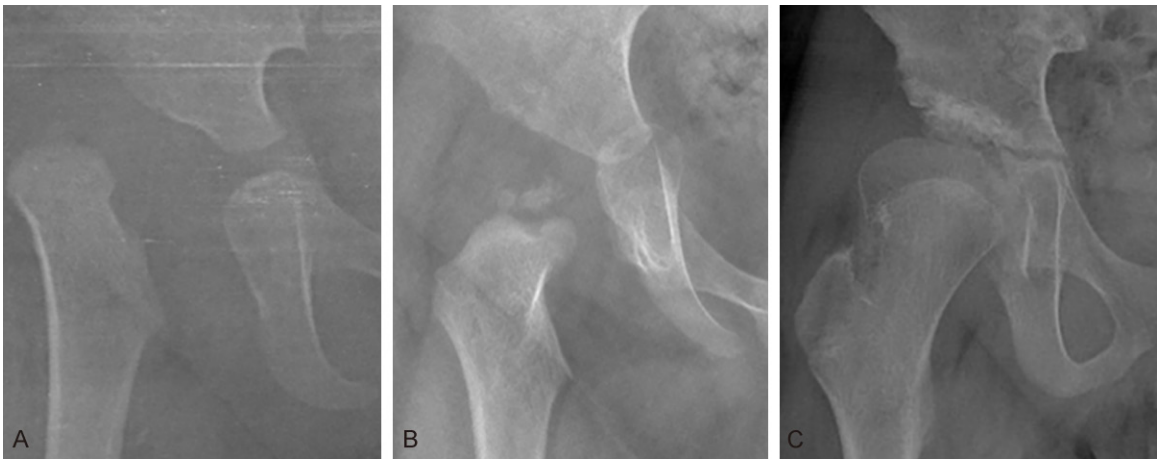

Figure 3. Among them, (A and B) are types 3 (lateral epiphyseal defect), and (C) is an imaging picture of a child with type 4 (lateral cystic change of metaphysis).

Table 3. Analysis of risk factors for avascular necrosis of the femoral head

Variable (B)	B	Standard error	Degree of freedom	Wald	EXP (B)	P
Widening and calcification of lateral epiphysis	0.812	0.835	1	0.945	2.252	0.331
Distortion of lateral epiphyseal plate and formation of bone bridge	2.282	0.729	1	9.828	9.821	0.002
Lateral epiphyseal defect	1.059	1.193	1	0.788	2.883	0.375
Lateral cystic change of metaphysis	1.901	1.558	1	1.488	6.691	0.223

dren who have not been identidied in time for delayed treatment, and secondary issues of the spine and knee joint may occur [19]. If children with positive clinical symptoms are not treated in time, the hip may deteriorate, and finally surgery is needed or serious sequelae may occur [20]. Among all complications, avascular necrosis of the femoral head is the most serious. Avascular necrosis of the femoral head is an unavoidable complication during DDH treatment, and once it occurs, it will seriously affect the prognosis of the children [14].

Among various types of avascular necrosis of the femoral head, Kalamchi-Macewen type 2 has the highest incidence rate. This type of avascular necrosis of the femoral head involves the lateral epiphyseal plate of the femoral head, causing growth disorder of the proximal femur. As the children get older, it will eventually lead to coxa varus deformity, poor coverage of the head socket and early degeneration of joints. Unfortunately, this has the highest incidence and a huge impact on complications, but it is not easily found early after DDH treatment [15]. Therefore, imaging plays an extremely important role in early screening and dynamic observation of DDH [6], and patients need long-term follow-up after treatment and intervention. The epiphyseal plate is the birthplace of bone growth and development, and the connection of the epiphyseal plate is much weaker than other ligament parts, which is more prone to injury. The local gap after the epiphyseal plate injury causes barrier function to blood vessels, which leads to the formation of fibrovascular bridges at the epiphyseal and metaphyseal ends, and eventually leads to the formation of bone bridges. The formation of bone bridges will lead to angulation and shortening of affected limb parts [21]. Clinically, it is considered that the formation of bone bridges is the main cause of limb deformities, so various treatment schemes are often used to prevent the formation of bone bridges for such patients [22-24]. In this study, the exploration of early imaging signs found that the incidence of early influencing signs in the case group was significantly higher than that in the control group ($P<0.001$), and the incidence of type 2 of early imaging signs (distortion of the lateral epiphyseal plate, formation of a bone bridge) in the case group was significantly higher than that in the control group ($P<0.001$).

Previous studies believed that the relevant factors of avascular necrosis of the femoral head in DDH in children include being female, primary delivery, DDH family history and left hip joint lesion [25, 26]. In this study, it was found that distortion of the lateral epiphyseal plate and formation of a bone bridge are independent risk factors for avascular necrosis of the femoral head in DDH in children. It was suggested that if the lateral epiphyseal plate is distorted early DDH in children, the risk of avascular necrosis of the femoral head should be indicated when a bone bridge is formed.

The sample size of this study is relatively small, which can be expanded in further study of the early imaging signs of avascular necrosis of the femoral head in DDH.

To sum up, the incidence rate of early imaging signs of avascular necrosis of the femoral head in DDH in children is high, of which the distortion of the lateral epiphyseal plate and the formation and change of a bone bridge are the most common, suggesting that early imaging changes should be indications to the occurrence of type 2 avascular necrosis of the femoral head.

Disclosure of conflict of interest

None.

Address correspondence to: Zhigang Wang, Department of Orthopaedics, Shanghai Children's Medical Center, Shanghai Jiao Tong University School of Medicine, No. 1678 Dongfang Road, Pudong New District, Shanghai 200127, China. Tel: +86-17721249250; E-mail: wangzhigang35wzg@163.com

References

[1] Pollet V, Percy V and Prior HJ. Relative risk and incidence for developmental dysplasia of the hip. J Pediatr 2017; 181: 202-207.

[2] Mace J and Paton RW. Neonatal clinical screening of the hip in the diagnosis of developmental dysplasia of the hip: a 15-year prospective longitudinal observational study. Bone Joint J 2015; 97-b: 265-269.

[3] Department of Orthopaedics, Pediatric Surgery Branch, Chinese Medical Association, Pediatric Trauma and Orthopaedics Section, Chinese Medical Association Orthopaedic Branch. Clinical diagnosis and treatment guidelines for developmental hip dysplasia (0 to 2 years old). Chin J Orthop 2017; 37: 641-650.

[4] Williams D, Protopapa E, Stohr K, Hunter JB and Roposch A. The most relevant diagnostic criteria for developmental dysplasia of the hip: a study of British specialists. BMC Musculoskelet Disord 2016; 17: 38.

[5] Zhang J, Yan M, Zhang Y, Yang H and Sun Y. Association analysis on polymorphisms in WISP3 gene and developmental dysplasia of the hip in Han Chinese population: a case-control study. Gene 2018; 664: 192-195.

[6] Hareendranathan AR, Mabee M, Punithakumar K, Noga M and Jaremko JL. Toward automated classification of acetabular shape in ultrasound for diagnosis of DDH: contour alpha angle and the rounding index. Comput Methods Programs Biomed 2016; 129: 89-98.

[7] Clohisy JC. Developmental dysplasia of the hip: contemporary concepts and treatment innovations. J Arthroplasty 2017; 32: S18-S19.

[8] Shin CH, Yoo WJ, Park MS, Kim JH, Choi IH, Cho TJ, Shin CH, Yoo WJ and Park MS. Acetabular remodeling and role of osteotomy after closed reduction of developmental dysplasia of the hip. J Bone Joint Surg Am 2016; 98: 952-957.

[9] Cicekli O and Dogan M. Evaluation of surgical outcome in advanced age patients with developmental hip dysplasia. Int J Surg 2018; 52: 44-49.

[10] Karachalios T and Hartofilakidis G. Congenital hip disease in adults: terminology, classification, pre-operative planning and management. J Bone Joint Surg Br 2010; 92: 914-921.

[11] Lerch TD, Steppacher SD, Liechti EF, Tannast M and Siebenrock KA. One-third of hips after periacetabular osteotomy survive 30 years with good clinical results, no progression of arthritis, or conversion to THA. Clin Orthop Relat Res 2017; 475: 1154-1168.

[12] van Bosse H, Wedge JH and Babyn P. How are dysplastic hips different? A three-dimensional CT study. Clin Orthop Relat Res 2015; 473: 1712-1723.

[13] Schwend RM, Shaw BA and Segal LS. Evaluation and treatment of developmental hip dysplasia in the newborn and infant. Pediatr Clin North Am 2014; 61: 1095-1107.

[14] Mahan ST and Kasser JR. Does swaddling influence developmental dysplasia of the hip? Pediatrics 2008; 121: 177-178.

[15] Kalamchi A and MacEwen GD. Avascular necrosis following treatment of congenital dislocation of the hip. J Bone Joint Surg Am 1980; 62: 876-888.

[16] Kalamchi A, Schmidt TL and MacEwen GD. Congenital dislocation of the hip. Open reduction by the medial approach. Clin Orthop Relat Res 1982; 127-132.

[17] Dezateux C and Rosendahl K. Developmental dysplasia of the hip. Lancet 2007; 369: 1541-1552.
[18] Narayanan U, Mulpuri K, Sankar WN, Clarke NM, Hosalkar H and Price CT. Reliability of a new radiographic classification for developmental dysplasia of the hip. J Pediatr Orthop 2015; 35: 478-484.
[19] Schaeffer EK, Study Group I and Mulpuri K. Developmental dysplasia of the hip: addressing evidence gaps with a multicentre prospective international study. Med J Aust 2018; 208: 359-364.
[20] Riccabona M, Schweintzger G, Grill F and Graf R. Screening for developmental hip dysplasia (DDH)-clinically or sonographically? Comments to the current discussion and proposals. Pediatr Radiol 2013; 43: 637-640.
[21] Nguyen JC, Markhardt BK, Merrow AC and Dwek JR. Imaging of pediatric growth plate disturbances. Radiographics 2017; 37: 1791-1812.
[22] Lee SU, Lee JY, Joo SY, Lee YS and Jeong C. Transplantation of a scaffold-free cartilage tissue analogue for the treatment of physeal cartilage injury of the proximal tibia in rabbits. Yonsei Med J 2016; 57: 441-448.
[23] Asen AK, Goebel L, Rey-Rico A, Sohier J, Zurakowski D, Cucchiarini M and Madry H. Sustained spatiotemporal release of TGF-beta1 confers enhanced very early chondrogenic differentiation during osteochondral repair in specific topographic patterns. FASEB J 2018; 32: 5298-5311.
[24] Fischer J, Knoch N, Sims T, Rosshirt N and Richter W. Time-dependent contribution of BMP, FGF, IGF, and HH signaling to the proliferation of mesenchymal stroma cells during chondrogenesis. J Cell Physiol 2018; 233: 8962-8970.
[25] Ortiz-Neira CL, Paolucci EO and Donnon T. A meta-analysis of common risk factors associated with the diagnosis of developmental dysplasia of the hip in newborns. Eur J Radiol 2012; 81: e344-351.
[26] Talbot CL and Paton RW. Screening of selected risk factors in developmental dysplasia of the hip: an observational study. Arch Dis Child 2013; 98: 692-696.