Research Square

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Age at onset of hip osteoarthritis and joint survival in developmental dysplasia of the hip: a 20-year longitudinal study stratified by lateral center-edge angle

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Abstract

Background

Developmental dysplasia of the hip (DDH) is a major cause of hip osteoarthritis (OA). However, few studies have evaluated OA onset age and joint preservation in relation to DDH severity. Here, we stratified DDH cases by lateral center-edge angle (LCEA) and analyzed age at radiographic OA onset and 20-year joint survival rates according to LCEA magnitude.

Methods

DDH cases were classified into six groups by LCEA: A, ≤ 5°; B, > 5° to ≤ 10°; C, > 10° to ≤ 15°; D, > 15° to ≤ 20°; E, > 20° to ≤ 25°; and F, > 25°. Age at OA onset was compared using the Kruskal-Wallis test. The endpoint was radiographic OA onset, and joint survival rates were calculated using the Kaplan-Meier method.

Results

In total, 151 hips were followed for a mean of 24.8 years. The distribution was 15, 17, 48, 35, 25, and 11 hips in groups A to F, respectively. Age at OA onset was in the 50s to 60s in all groups, with no significant differences (p = 0.577). The 20-year joint survival rates were 40.0%, 77.8%, 71.6%, 81.4%, 79.2%, and 90.9%, respectively, with no significant difference (p = 0.138).

Conclusions

Radiographic OA onset in DDH occurred in the 50s to 60s regardless of LCEA magnitude. Although survival was lowest in group A and highest in group F, the overall comparison was not statistically significant. Prognosis is difficult to predict using LCEA alone, and further investigation incorporating other morphological parameters and patient background is warranted.

Trial registration

Not applicable.

Background

Developmental dysplasia of the hip (DDH) is a major cause of secondary hip osteoarthritis (OA). Patients with DDH are at higher risk of developing OA than those with normal hips [1–3]. In Japan, 80% of OA cases are attributable to DDH [4], meaning that predicting the long-term course of DDH is very important.

Estimating the future risk of OA onset is essential for determining appropriate treatment strategies for DDH and may also contribute to the prevention of OA development. OA is an important public health issue in aging societies [5], and although there is an urgent need for its prevention and appropriate treatment selection [6], epidemiological data on OA remain limited.

DDH is evaluated using the lateral center-edge angle (LCEA) proposed by Wiberg [7]. Several studies have demonstrated an association between LCEA and the risk of OA development [3, 8]. A previous study [9] also identified LCEA as an independent risk factor for OA among several indicators of DDH. A low LCEA increases stress on the joint surface [10], induces joint instability [11], and leads to cartilage degeneration, which can progress further to OA.

While several studies have investigated the incidence of OA within a certain period [12] and the risk of OA development [1, 2], the age at which OA develops remains unclear. A multicenter study in Japan [4] reported that the most common age at initial consultation for OA was in the 50s, followed by the 60s; however, very few reports clearly define OA onset and specifically examine the radiographic onset of OA.

In daily clinical practice, clinicians are frequently asked questions such as, “At what age is my hip likely to deteriorate?” and “What is the likelihood developing OA if I do not undergo surgery?” Answering these questions requires more than simply presenting the risk of OA onset. It is essential to evaluate temporal information, such as when OA is likely to develop and how long the joint can be preserved depending on the degree of acetabular coverage, in conjunction with the severity of DDH. However, there are very few longitudinal studies that have simultaneously evaluated OA onset and joint survival over time according to the severity of DDH. Many studies have concluded that DDH with LCEA < 25° or < 20° is associated with a higher risk of OA development compared with normal hips [13, 14]. However, because the severity of DDH varies widely from severe to mild, treating it as a homogeneous population has limitations.

In the present study, as in previous studies [9, 15], we followed the contralateral non-operated hips of patients with bilateral DDH who underwent unilateral rotational acetabular osteotomy (RAO) for at least 20 years to determine when and with what probability OA develops according to the magnitude of LCEA. The objective of this study was to stratify DDH cases based on LCEA and compare the age at OA onset and joint survival rates according to LCEA magnitude.

Methods

Study design and ethical considerations

This study was a retrospective study approved by the ethics committee at our hospital. Informed consent was obtained using an opt-out approach.

Patient selection

The study included patients who had undergone unilateral RAO for bilateral DDH at our hospital before 2005, with the contralateral non-operated hip used as the study hip. The inclusion criteria were based on previous studies [9, 15], including (1) no evidence of OA (i.e., asymptomatic at the start of observation [at time of unilateral RAO], with a Kellgren and Lawrence [KL] grade of 0), and (2) availability for continuous follow-up for at least 20 years after surgery. The exclusion criteria were as follows: (1) a history of hip surgery on the study hip during childhood; (2) a history of prior RAO or total hip arthroplasty (THA) before RAO; (3) a history of trauma, Perthes disease, skeletal disease, cerebral palsy, or other related conditions; (4) cases in which additional procedures such as greater trochanteric advancement or femoral osteotomy were performed on the operated side concurrently with RAO, or cases in which muscle release procedures were performed on the study hip; and (5) cases that required bilateral RAO within one year.

Radiographic evaluation and outcomes

The LCEA of the study hip was measured on a supine anteroposterior pelvic radiograph obtained immediately before unilateral RAO. Based on LCEA values, hips were classified into six groups as follows: A, LCEA ≤ 5°; B, 5° < LCEA ≤ 10°; C, 10° < LCEA ≤ 15°; group D, 15° < LCEA ≤ 20°; E, 20° < LCEA ≤ 25°; and F, 25° < LCEA. Postoperative radiographs were evaluated longitudinally at intervals of one to two years, and the timing of OA onset were identified. OA onset was defined as KL grade ≥ 1. The cumulative joint survival rate, with endpoint defined as OA onset, was also determined.

Reproducibility assessment

To assess reproducibility, LCEA measurements were performed by two authors (HA and AA). Data were analyzed for within- and between-measurer variation, and the coefficient of variation was calculated to be less than 5%, indicating acceptable measurement reproducibility.

Statistical analysis

Age at onset was expressed as the median for each group, and between-group comparisons were performed using the Kruskal-Wallis test. Joint survival rates were analyzed using the Kaplan-Meier method, and comparisons between groups were performed using the log-rank test. The significance level was set at less than 5%. Statistical analyses were performed using JMP version 17.2 software (SAS Institute, Cary, NC).

Results

Among the 1,050 hips that underwent RAO at our hospital before 2005, 380 hips met the inclusion criteria. After applying the exclusion criteria, a total of 151 hips were ultimately included in the analysis (Fig. 1). Of these, 149 hips were in female patients and 2 in male patients. Mean age at the time of RAO

(± standard deviation [SD]) was 42.3 ± 9.28 years, and the mean follow-up period (± SD) was 24.8 ± 4.58 years.

The distribution of hips in each group was as follows: 15 in group A, 17 in group B, 48 in group C, 35 in group D, 25 in group E, and 11 in group F. Table 1 shows the baseline characteristics of each group. The number of hips with OA onset among the respective groups was 6, 4, 10, 9, 6, and 1 (Table 2). Median age at OA onset was 50.2 years in group A, 64.9 years in group B, 62.5 years in group C, 59.4 years in group D, 62.3 years in group E, and 62.5 years in group F (Table 2). No significant differences were found among the 6 groups (p = 0.577, Kruskal-Wallis test).

Table 1
Baseline characteristics by LCEA group

Group	No. of hips	Age at baseline (mean ± SD)	LCEA (mean ± SD)
Total	151 hips	42.3 ± 9.28 years	15.0 ± 7.87 degrees
A	15 hips	36.3 ± 11.2 years	1.20 ± 6.32 degrees
B	17 hips	45.1 ± 7.80 years	8.12 ± 1.45 degrees
C	48 hips	43.6 ± 9.30 years	12.6 ± 1.36 degrees
D	35 hips	41.6 ± 9.58 years	17.8 ± 1.26 degrees
E	25 hips	41.4 ± 8.69 years	22.8 ± 1.19 degrees
F	11 hips	45.0 ± 5.50 years	29.4 ± 3.11 degrees
SD, standard deviation; LCEA, lateral center-edge angle			

Table 2
Number of cases and age at onset of OA in each group

Group	OA onset	Age at OA onset (median [IQR])
A	**6 hips**	**50.2 (46.9–59.4) years**
B	**4 hips**	**64.9 (62.3–66.7) years**
C	**10 hips**	**62.5 (57.4–67.5) years**
D	**9 hips**	**59.4 (55.0-65.8) years**
E	**6 hips**	**62.3 (57.5–67.0) years**
F	**1 hip**	**62.5 (62.5–62.5) years**
OA, osteoarthritis; IQR, interquartile range		

The 20-year joint survival rates were 40.0% (95% confidence interval [CI], 0.095-0.700), 77.8% (95% CI, 0.455–0.923), 71.6% (95% CI, 0.535–0.836), 81.4% (95% CI, 0.632–0.912), 79.2% (95% CI, 0.570–0.908),

and 90.9% (95% CI, 0.508–0.987) in groups A, B, C, D, E, and F, respectively (Fig. 2). The overall log-rank test demonstrated no significant difference among the six groups ($\chi^2 = 8.356$, $p = 0.138$).

Discussion

This study stratified DDH cases based on LCEA values and examined the age at OA onset and joint survival rates according to LCEA magnitude. Based on the results for age at OA onset and joint survival rates, we further discuss appropriate treatment strategies for DDH.

Radiographic age at OA onset

In this study, the radiographic age at OA onset was about 50 years in group A and around 60 years in the other groups, with no significant differences among the groups. In a previous study [9], the radiographic onset of OA was estimated to occur at about 55 years of age, and a prior literature review [16] found that up to 50% of DDH cases develop radiographic OA by a mean age of 50.3 years. Our findings are consistent with the peak age reported in these other studies. Thus, in patients with DDH, OA onset seems most likely to occur in their 50s or 60s regardless of LCEA magnitude. However, though no statistically significant difference was observed, the age at onset in group A was approximately 50 years, about 10 years younger than in the other groups. Therefore, the possibility that extremely severe DDH is associated with earlier onset of OA cannot be ruled out.

Joint survival rate

The 20-year joint survival rate was 40% in group A, 70 to 80% in groups B to E, and 90% in group F. Although the survival rate was lowest in group A and highest in group F, the overall comparison among the 6 groups showed no statistically significant difference.

In DDH, abnormal load distribution is considered to cause early cartilage damage, increase stress on the surrounding soft tissues, and ultimately lead to the development of OA [17, 18]. Murphy et al. [3] also reported that many patients with moderate to severe DDH with an LCEA of < 15° develop OA by their 70s, and another study reported that DDH with an LCEA of < 5° is prone to developing OA even at a young age [19]. While our findings were directionally consistent with these reports, the present study did not demonstrate a statistically significant difference among the 6 groups. This may be explained by insufficient statistical power due to the small sample size, as well as the possibility that OA onset is influenced by factors other than LCEA. A study on borderline DDH also reported no statistically significant differences in the timing of OA onset or joint survival compared with normal hips [15]. Therefore, more detailed evaluation incorporating additional parameters such as acetabular roof obliquity and anterior center-edge angle, as well as three-dimensional imaging using computed tomography [20, 21] and assessment of joint instability [22], is warranted.

Treatment strategy for DDH

In group A (LCEA ≤ 5°), the 20-year joint survival rate was the lowest, at 40%, indicating that this group requires particular attention regarding OA onset. In such cases, it is important to carefully assess symptoms and patient background during follow-up and, when necessary, to consider therapeutic interventions, including joint-preserving surgery. Although group F showed the highest 20-year joint survival rate, some hips still developed OA, so caution is therefore required. However, because the number of hips in this study was limited, these findings should be interpreted with caution.

In groups B to E, the age at OA onset was approximately in the 60s, and the joint survival rate was approximately 70% to 80%. Because these groups included a mixture of cases with and without OA onset, determining the optimal treatment strategy remains challenging. Since prognosis is difficult to predict using LCEA alone, further investigation of risk factors incorporating other morphological parameters and patient background is warranted.

Limitations

This study has several limitations. First, the sample size was limited, which may have reduced the statistical power to detect significant differences. In particular, the numbers of hips in groups A and F were small, and therefore the between-group comparisons should be interpreted with caution. Accordingly, the absence of statistical significance should not be interpreted as evidence of no association between LCEA and the risk of OA onset. Second, stratification was performed using LCEA alone. Because DDH cannot be fully evaluated using LCEA alone, future studies should include additional radiographic parameters. Third, this study was based on a Japanese cohort in which most patients were female, which may limit generalizability to other populations. Fourth, patients who underwent bilateral RAO within 1 year from the start of observation were excluded. These patients were likely to have already been symptomatic at the study hip [5] and were therefore judged not to meet the inclusion criterion of asymptomatic DDH. As a result, more severe cases may have been excluded, and the risk of OA onset may have been underestimated.

Despite these limitations, this was a rare longitudinal study that simultaneously evaluated the radiographic age at OA onset and joint survival rates by following patients with DDH for at least 20 years. The findings of this study may provide useful information for explaining prognosis to patients and guiding treatment decisions.

Conclusion

In this longitudinal study, the contralateral non-operated hips of patients with bilateral DDH who had undergone unilateral RAO were followed for more than 20 years. Study hips were stratified based on LCEA and analyzed for age at OA onset and joint survival. Radiographic OA onset did not differ significantly among the groups and occurred generally in the 50s to 60s. Although joint survival rates were not statistically different among the groups, they were 40% in group A, approximately 70% to 80% in groups B to E, and approximately 90% in group F. Because the timing of OA onset and the development

of OA within 20 years are difficult to evaluate using LCEA alone, further investigation of more detailed risk factors is warranted.

Abbreviations

DDH
Developmental dysplasia of the hip
OA
hip osteoarthritis
LCEA
lateral center-edge angle
RAO
rotational acetabular osteotomy
KL
Kellgren and Lawrence
THA
total hip arthroplasty
SD
standard deviation
CI
confidence interval
IQR
interquartile range

Declarations

Ethics approval and consent to participate

The study was conducted in accordance with the Declaration of Helsinki. This study was approved by the ethics committee at Kanagawa Rehabilitation Hospital (approval number: krh-2020-23). Informed consent was obtained by an opt-out approach.

Consent for publication

Not applicable.

Competing interests

The authors declare no competing interests.

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Author Contribution

A. A.: Conceptualization, Resources, Investigation, Writing – original draft, Writing – review & editing.H. S.: Resources, Project administration, Supervision, writing – review & editing.M. S.: Formal analysis, Supervision.

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Availability of data and materials

The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request.

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References

1. Jacobsen S, Sonne-Holm S. Hip dysplasia: a significant risk factor for the development of hip osteoarthritis. A cross-sectional survey. Rheumatology (Oxford). 2005;44:211–8.

https://doi.org/10.1093/rheumatology/keh436.

2. Reijman M, Hazes JMW, Pols HAP, Koes BW, Bierma-Zeinstra SMA. Acetabular dysplasia predicts incident osteoarthritis of the hip. Arthritis Rheum. 2005;52:787–93. https://doi.org/10.1002/art.20886.
3. Murphy SB, Ganz R, Müller ME. The prognosis in untreated dysplasia of the hip. A study of radiographic factors that predict the outcome. J Bone Joint Surg Am. 1995;77:985–89. https://doi.org/10.2106/00004623-199507000-00002.
4. Jingushi S, Ohfuji S, Sofue M, et al. A multi-institutional epidemiologic study regarding osteoarthritis of the hip in Japan. J Orthop Sci. 2010;15:626–31. 10.1007/s00776-010-1507-8.
5. Liebe J, Schlappi M, Meier C, Wahl P. Systematic review of the literature and meta-analysis of the long-term likelihood of contralateral total hip arthroplasty. EFORT Open Rev. 2025;10:267–76. https://doi.org/10.1530/EOR-2024-0026.
6. Iidaka T, Muraki S, Oka H, et al. Incidence rate and risk factors for radiographic hip osteoarthritis in Japanese men and women: a 10-year follow-up of the ROAD study. Osteoarthritis Cartilage. 2020;28:182–8. https://doi.org/10.1016/j.joca.2019.09.006.
7. Wiberg G. Studies on dysplastic acetabula and congenital subluxation of the hip joint: with special reference to the complication of osteo-arthritis. Acta Chir Scand. 1939;83:5–135.
8. Wyles CC, Heidenreich MJ, Jeng J, et al. The John Charnley award: Redefining the natural history of osteoarthritis in patients with hip dysplasia and impingement. Clin Orthop Relat Res. 2017;475:336–50. https://doi.org/10.1007/s11999-016-4815-2.
9. Amagami A, Sugiyama H, Tonotsuka H, Saito M. Long-term course of developmental dysplasia of the hip: follow-up of the non-operated hips of patients undergoing unilateral rotational acetabular osteotomy for twenty-four years. Arch Orthop Trauma Surg. 2024;144:997–1004. https://doi.org/10.1007/s00402-023-05140-1.
10. Pompe B, Daniel M, Sochor M, Vengust R, Kralj-Iglič V, Iglič A. Gradient of contact stress in normal and dysplastic human hips. Med Eng Phys. 2003;25:379–85. 10.1016/S1350-4533(03)00014 – 6.
11. Sato T, Tanino H, Nishida Y, Ito H, Matsuno T, Banks SA. Dynamic femoral head translations in dysplastic hips. Clin Biomech. 2017;46:40–5. https://doi.org/10.1016/j.clinbiomech.2017.05.003.
12. Lane NE, Lin P, Christiansen L, et al. Association of mild acetabular dysplasia with an increased risk of incident hip osteoarthritis in elderly white women: the study of osteoporotic fractures. Arthritis Rheum. 2000;43:400–4. https://doi.org/10.1002/1529-0131(200002)43. :2%3C400::AID-ANR21%3E3.0.CO;2-D.
13. Riedstra NS, Boel F, van Buuren MMA, et al. Acetabular dysplasia and the risk of developing hip osteoarthritis within 4–8 years: an individual participant data meta-analysis of 18,807 hips from the World COACH consortium. Osteoarthritis Cartilage. 2025;33:373–82. https://doi.org/10.1016/j.joca.2024.12.001.
14. Vinge R, Riedstra N, Tiderius CJ, Bierma-Zeinstra S, Agricola R, Runhaar J. Hip dysplasia as risk factor for clinically relevant and radiographic hip osteoarthritis: 10-year results from the CHECK

cohort. Rheumatology. 2025. https://doi.org/10.1093/rheumatology/kead650. 64;149 – 55.

15. Amagami A, Sugiyama H, Saito M. Long-term course of borderline dysplasia: a 26-year follow-up of nonoperative hips in patients undergoing unilateral acetabular rotational osteotomy. Bone Jt Open. 2025;6:419–24. https://doi.org/10.1302/2633-1462.64.BJO-2024-0276.R1.
16. Gala L, Clohisy JC, Beaulé PE. Hip dysplasia in the young adult. J Bone Joint Surg Am. 2016;98:63–73. 10.2106/JBJS.O.00109.
17. Ganz R, Leunig M, Leunig-Ganz K, Harris WH. The etiology of osteoarthritis of the hip. Clin Orthop Relat Res. 2008;466:264–72. https://doi.org/10.1007/s11999-007-0060-z.
18. Harris MD, Thapa S, Lieberman EG, et al. Identifying risk factors for disease progression in developmental dysplasia of the hip using a contralateral hip model. J Bone Joint Surg Am. 2024;106:2322–9. https://doi.org/10.2106/JBJS.24.00308.
19. Jessel RH, Zurakowski D, Zilkens C, Burstein D, Gray ML, Kim YJ. Radiographic and patient factors associated with pre-radiographic osteoarthritis in Hip dysplasia. J Bone Joint Surg. 2009;91:1120–9. 10.2106/JBJS.G.00144.
20. Graesser EA, Schwabe MT, Clohisy JC, Nepple JJ. Defining the borderline dysplastic hip: high variability in acetabular coverage and femoral morphology on low-dose computed tomography. Am J Sports Med. 2023;51:2551–8. https://doi.org/10.1177/03635465231184395.
21. Ito H, Matsuno T, Hirayama T, Tanino H, Yamanaka Y, Minami A. Three-dimensional computed tomography analysis of non-osteoarthritic adult acetabular dysplasia. Skeletal Radiol. 2009;38:131–9. https://doi.org/10.1007/s00256-008-0601-x.
22. Wyatt M, Weidner J, Pfluger D, Beck M. The Femoro-Epiphyseal Acetabular Roof (FEAR) index: a new measurement associated with instability in borderline hip dysplasia? Clin Orthop Relat Res. 2017;475:861–9. https://doi.org/10.1007/s11999-016-5137-0.

Figures

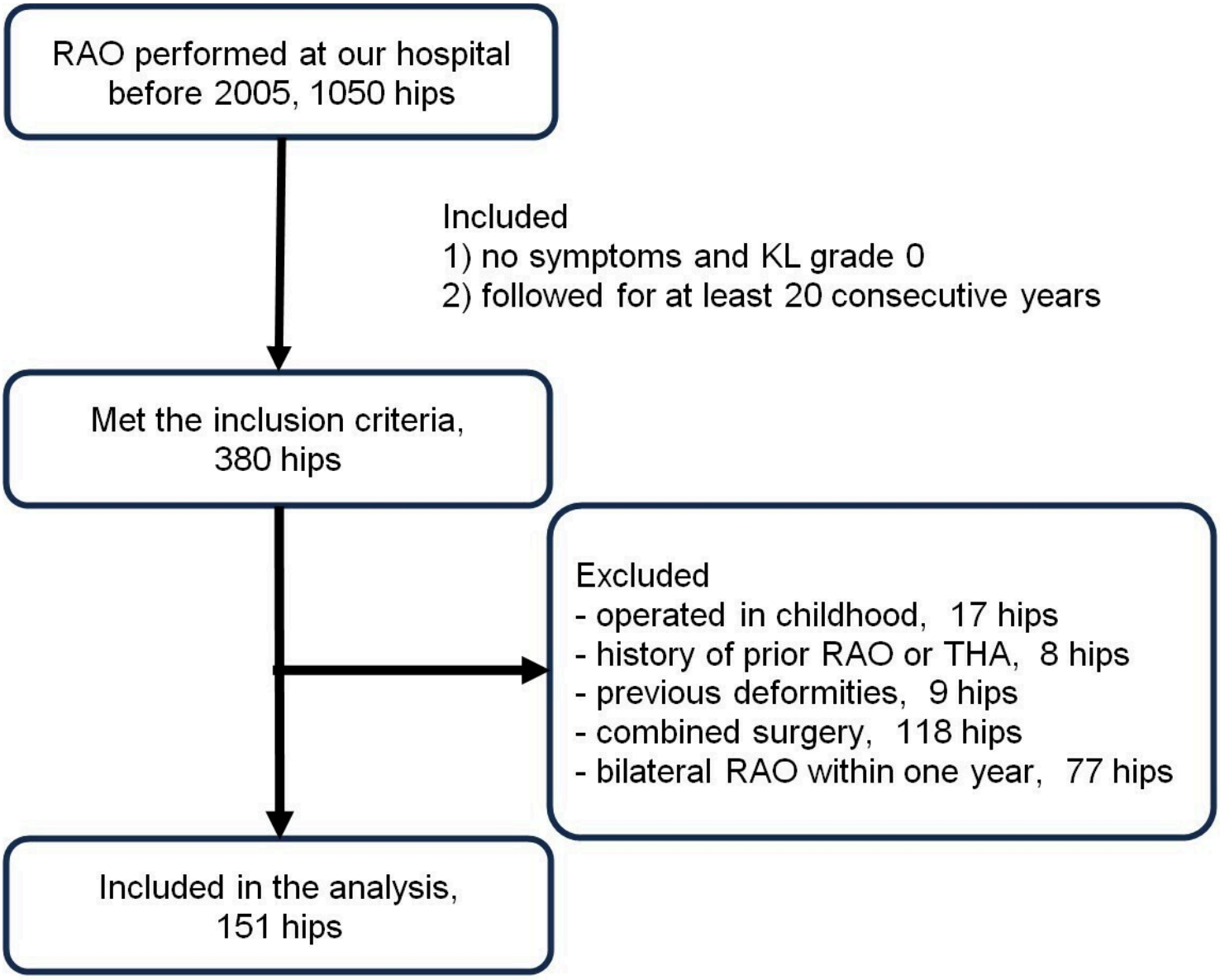


Figure 1

Flowchart of patient selection and exclusion criteria

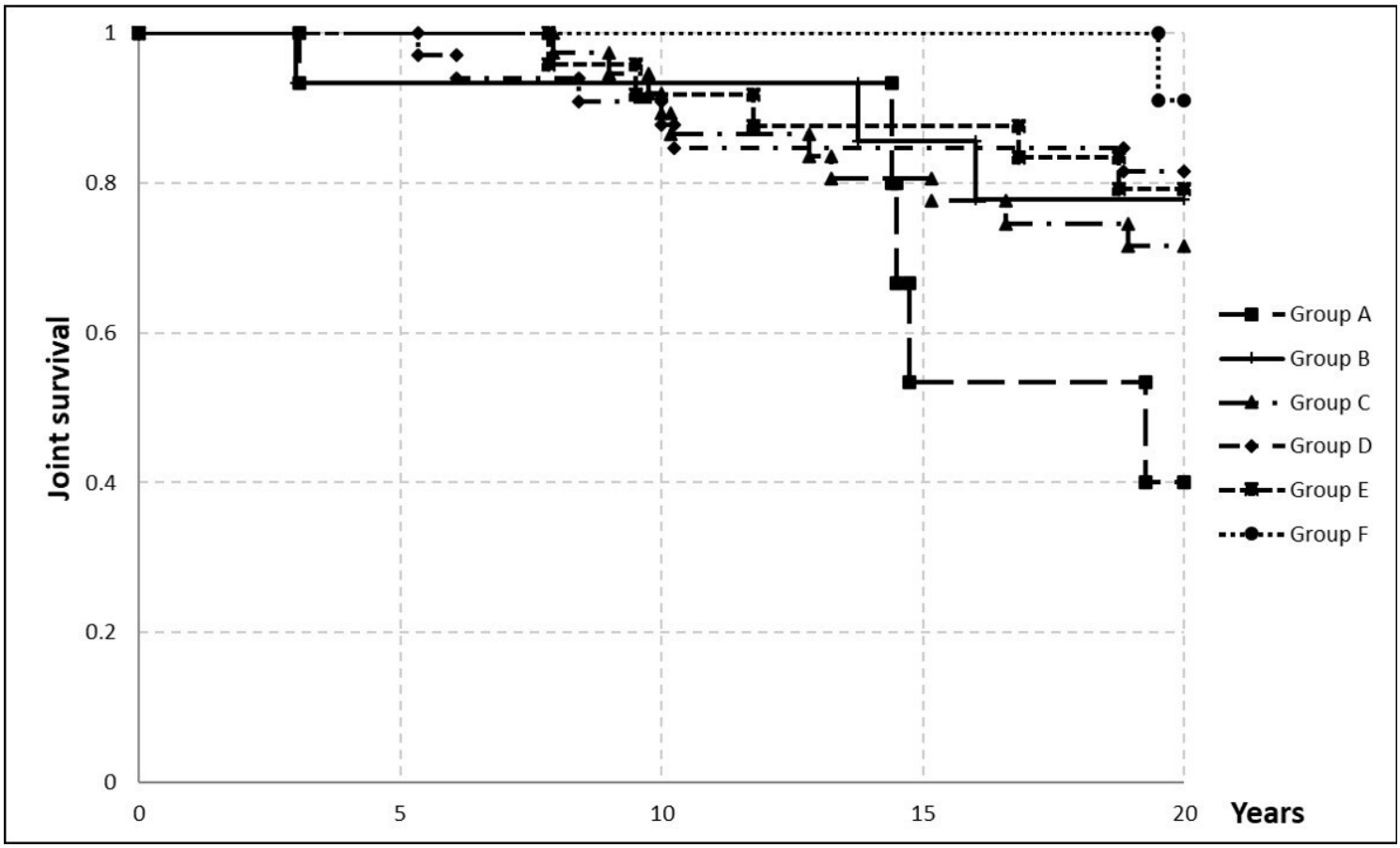


Figure 2

Kaplan-Meier survival curves with OA onset as endpoint