Risk factors for the development of knee osteoarthritis

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ABSTRACT

Objective. This article presents case control study conducted at the Rheumatology Outpatient Unit, Department of Internal Medicine, Cipto Mangunkusumo Hospital. The aim of this study was to determine several risk factors for the development of knee osteoarthritis (OA) at Rheumatology out-patient unit Department of Internal Medicine, Cipto Mangunkusumo Hospital.

Method. This study used a case control design. Subjects were divided into two groups, case group and control group. The case group included all patients who had knee complaints that fulfilled the 1990 American College of Radiology criteria while the control group included patients randomly chosen from visitors of the Rheumatology Outpatient Unit of Cipto Mangunkusumo Hospital who did not complain of knee pain and had been proven of not suffering OA by physical and radiological examinations. A similar questionnaire was designed for both groups. After filling out the questionnaire, both groups were examined physically and radiologically. The risk factors were analyzed using logistic regression.

Results. We included 127 patients in the case group and 102 in the control group. In the case group, there were 95 women (74.80 %) and 32 men (25.20%) while the control group consisted of 69 women (67.65%) and 33 men (32.35%). From the analysis of several risk factors, there were significant differences between the case and control groups such as weight ($p < 0.0001$ *df* 3), prior history of overweight or obesity ($x^2 = 21.255$, *df* 1, $p < 0.0001$), knee trauma ($p = 0.0002$), and kneel down activity ($p < 0.0001$). There was also a significant difference of smoking habit between the case group and the control group ($p < 0.0001$). Duration of smoking cessation was also significantly different between the case group and control group ($t = 2.315$, *df* 45, $p = 0.0252$). From the multivariate analysis, it was found that age, kneel down activity, prior obesity condition, interval between knee trauma and onset of OA, smoking habit, and duration of smoking cessation had a significant correlation with knee OA.

Conclusion. Age, prior history of overweight or obesity, kneeling, and interval between knee trauma and onset of OA were risk factors of knee OA. Smoking was a negative risk factor for knee OA. The protective effect of smoking was influenced by the duration of smoking habit and the duration of smoking cessation.

Osteoarthritis (OA) is a slowly progressive degenerative disease of the articular cartilage of which cause and pathogenesis is still debatable.[1,2] Adebajo and Moskowitz reported that besides the hip and finger, the knee joint is the most common joint attacked by OA, usually causing more severe case and worse disability.[3,4] The prevalence of OA obtained from patients visiting the Rheumatology Outpatient Unit of the Cipto Mangunkusumo Hospital/ Faculty of Medicine University of Indonesia was high (56.7%).

The etiopathogenesis of knee OA or OA in general is still unclear until today.[5] There are two main mechanisms in the development of knee OA: biomechanical disorder and biochemical disorder.[6,7] In the first mechanism, the body weight along with friction and the capability of the articular cartilage as absorber of mechanical stress play an important role. The latter mechanism - biochemical changes - is more acceptable in explaining the development of OA in joints that do not bear the body weight.

Some published epidemiological studies showed that various factors play a role in the development of knee OA as seen in table 1.[1,7-21]

Table 1 Risk factors for development of knee osteoarthritis

Researchers	Year	Risk Factors
Chander CL et al.	1991	Hormonal (estrogen)
Felson DT et al.		Obesity
Cushnagan J et al.	1991	Gender, age
Altman	1987	Gender, age, race, genetic, physical activity, obesity
Doherty M et al.	1983	Trauma (menisectomy)
Hannan MT et al.	1993	Physical activity
Kohatsu ND et al.	1990	Physical activity
Hadi S et al.	1993	Physical activity
Knight SM et al.	1992	Bone mass density
Schalwijk J et al.	1989	Hormonal/ Insulin like factor
Hochberg et al.	1994	Hormonal/ Insulin like factor
Felson DT et al.	1989	Smoking habit, age, gender, obesity
Brown RA et al.	1988	Endothelial cell stimulating factor
Niethard FU	1986	Diabetes (animal experiment)

Age is the main factor in the development of knee OA. This is proven by the low prevalence (less than 5%) of knee OA in ages below 45 years old. The steep rise in the prevalence is found at the age of above 65 years old and shows obvious clinical and radiological signs.[20] Women, especially who are above the age of 55 years old, have a higher prevalence of knee OA than that of men with a ratio of 2:1.[22,23]

The First National Health and Nutritional Examination (HANES I) survey showed that there was a significant correlation between recent obesity and knee OA. This conclusion was supported by the study conducted by Cushnagan and Dieppe.[9]

In the Framingham study, Felson et al unexpectedly found that cigarette smoking had a protective effect against knee OA. Although adjustments were made for age, gender, body weight, severity of the disease, history of knee trauma, exercising, physical activity, alcohol consumption, and coffee consumption; smoking was still found to be statistically significant in being a protective factor in the development of knee OA. The correlation between occupation and activity requiring repetitive involvement of the knee joint was proven by Cooper et al as one of the risk factors for the development of knee OA.[24]

The primary objective of our study was thus to determine the various risk factors associated with the development of knee OA in arthritis patients visiting the Rheumatology Outpatient Unit of the Department of Internal Medicine in Jakarta, Indonesia. The factors were age, obesity, knee trauma, squatting position during defecation, smoking, and physical activity requiring involvement of the knee. We hope this study would be beneficial in early detection of the disease and improve the methods of knee OA treatment.

METHODS

Patients

This case control study was carried out from January 1995 until November 1995 at the Rheumatology Outpatient Unit and other Outpatient Units of the Department of Internal Medicine, Faculty of Medicine University of Indonesia/ Cipto Mangunkusumo Hospital, Jakarta. The subjects in the case group were those that fulfilled the 1990 American College of Rheumatology criteria: knee pain, osteophyte (at least Grade II of the Kellgren- Lawrence Index), and one of the following: age above 50 years old, crepitation, or joint stiffness of less than 15 minutes. The subjects in the control group were randomly selected from the patients visiting the Outpatient Unit of the Department of Internal Medicine of the Cipto Mangunkusumo Hospital who presented no knee pain and were proven by physical and radiological examination not suffering from OA.

The instrument used in this study was a questionnaire similarly designed for both case and control group and contained information of the subject's identity, history of knee pain and knee trauma, smoking habit, physical activity requiring involvement of the knees, type of latrine**,** and physical examination findings: body height and weight, crepitation, bone enlargement, pressure pain of the bone, and warmness of the knee on palpation. Radiological examination based on the Kellgren- Lawrence Index was also performed.

Methods

Routine anamnesis and physical examination were performed to determine the diagnosis of knee OA. Explanation about the study was conveyed by the physician examining the case subjects or control subjects. If the patients were willing to participate in the study and sign a form of informed consent, radiological examination would be performed. The patients would be included in the case group if they fulfilled the operational definition of knee OA. Because there was no strong correlation between symptoms and objective signs of knee OA and to make sure that the case group consisted of only knee OA patients, patients having symptoms of knee OA but could not be proven by radiological examination were excluded in the study.

The control group were selected from patients visiting the Department of Internal Medicine Outpatient Unit and complaining of knee pain. Similar to the case group, the patients had to fulfil the inclusion and exclusion criteria and underwent radiological examination of the knee. To make sure that the control group only consisted of patients without knee OA, the control subjects who had knee OA of Grade II of the Kellgren- Lawrence Index on radiology examination were excluded in the study.

Statistical analyses

The sample size was calculated based on relative frequency on target population (p0), hypothesis odds ratio (R), α and β value. A prior study conducted by Daud et al found that subjects of over 55 years old had 2.1 times the risk of developing knee OA and obese subjects had also 2.1 times the risk of developing knee OA. It is assumed that the relative frequency in the elderly and the obese in the control group ranged from 20 to 30 % and we took the mean value of 25% as an estimate; therefore, Po was 0.25 and α specification was 0.05 (two-sided), β was 0.20 and R was 2.1. Finally, the estimated numbers of subject needed to be enrolled in this study was 144 subjects per group (with the estimated numbers of non responsive subject was 15%).

The risk factors were then analyzed by finding the odd ratio that was calculated using the logistic regression

RESULTS

We obtained 127 patients for the case group and 102 patients for the control group from data collected from January 1995 until November 1995.

Distribution of patients

In the control group, 95 subjects were women (74.80%) and 32 subjects were men (25.20%), while in the control group, 69 subjects were women (67.65%) and 33 subjects were men (32.35%) as shown in figure 1. The chi square test showed no significant difference between gender in the case group and control group in this study ($x^2 = 1.095$, *df* 1, $p = 0.2954$).

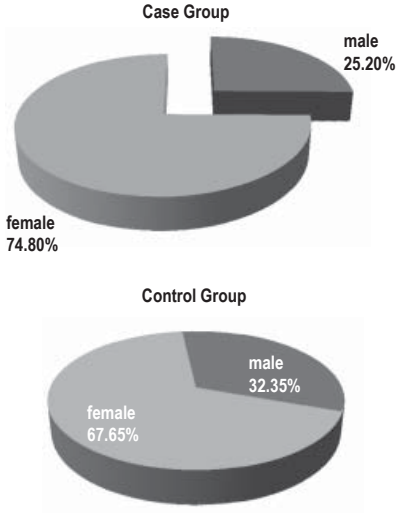

Figure 1 Distribution of gender in case and control groups

The age distribution in this study is shown in table 2. The independent sample t test of both groups showed statistical significance ($t = 7.277$, $p < 0.0001$).

Table 2 Distribution of age in case and control groups

Group	Range (year)	Mean of age (year)	Modes of age (year)
Case	37-87	59.93 ± 9.35	50-70
Control	30-83	50.10 ± 10.22	35-50

Risk Factors

Body Weight

Table 3 shows the distribution of body weight during examination of the case group and control group. Chi square test showed that there was a significant difference between the two groups based on the presence or absence of obesity ($x^2 = 17.343$, *df* 1, $p < 0.0001$) while based on overweight, there was also a significant difference between the two groups (x^2, *df* 1, $p = 0.0191$). In the analysis of the overall body weight (underweight, normal weight, overweight, and obesity), there was significant difference between the two groups ($p < 0.0001$ *df* 3) and there was a linear correlation between body weight and development of knee OA ($p < 0.0001$, *df* 1).

Table 3 Weight distribution at the time of enrollment in case and control groups

Group	Underweight	Normal weight	Overweight	Obesity	Total
Case	7 (5.51%)	35 (27.56%)	27 (21.25 %)	58 (45.67%)	127 (55.46%)
Control	40 (39.22%)	26 (25.49%)	17 (16.67%)	19 (18.63%)	102 (44.54%)
Total	47 (20.70%)	61 (26.87%)	44 (19.38%)	77 (33.92%)	229 (100%)

Table 4 contains prior history of obesity of the case group and control group. The chi square test showed a significant difference of prior history of obesity between the two groups ($x^2 = 21.255$, *df* 1, $p < 0.0001$). The Fisher's exact test showed statistical significance for the prior body weight between the case group and control group ($p < 0.0028$).

Table 4 Distribution of prior weight before enrollment in case and control groups

Group	Underweight	Normal weight	Overweight	Obesity	Total
Case	8 (6.30%)	34 (27.20%)	23 (18.11%)	62 (48.82 %)	127 (55.46%)
Control	34 (33.33%)	38 (37.25%)	11 (10.78%)	19 (18.63%)	102 (44.54%)
Total	42 (18.50%)	72 (31.72%)	34 (14.8%)	81 (35.68%)	229 (100%)

The duration of obesity of all 58 obese knee OA subjects ranged from one to 288 months with a mean value of 43.83 months. Fifty four subjects of the women group had duration of obesity ranging from one to 288 months with a mean value of 43.07 months. Four subjects of the men group had duration of obesity ranging from 12 to 120 months with a mean value of 54 months. Table 5 shows duration of obesity of both men and women in the knee OA group.

Table 5 Duration of obesity based on gender in case group

Gender	Duration	Average
Female	1-288 months	43.07 months
Male	12-120 months	54 months
Female + male	1-288 months	43.83 months

Twenty three subjects in the control group had duration of obesity ranging from 36 to 396 months with a mean value of 147.26 months. Twenty one subjects of the women control group had duration of obesity ranging from 36 to 396 months with a mean value of 148.71 months. In the men control group, two subjects had duration of obesity ranging from 120 to 144 months with a mean value of 132 months. Table 6 shows the duration of obesity based on gender.

Table 6 Duration of obesity based on gender in control group

Gender	Duration	Average
Female	36-396 months	148,71 months
Male	120-144 months	132 months
Female + male	36-396 months	147,26 months

The role of squatting position during defecation

The position during defecation can be seen in Table 7. The Fisher's exact test showed no significant difference between the two groups (p = 0.5143).

Table 7 Distribution of defecation position in case and control groups

Defecation position	Case	Control	Total
Squatting	98 (77.17%)	83 (81.37 %)	181 (79.04 %)
Sitting	29 (22.83%)	19 (18.36 %)	48 (20.96 %)
Total	127 (55.46%)	102 (44.54%)	229 (100%)

The role of knee trauma

Table 8 shows history of knee trauma before the onset of knee OA complaints. In the women case group, 15.79% had a history of knee trauma while in the men group, 25% had a history of knee trauma. In the women control group, there was no history of knee trauma among the subjects while in the men control group, 13.51% had a history of knee trauma. The Fisher's exact test showed a significant difference between the two groups (p = 0.0002).

Table 8 Distribution of prior history of knee trauma in case and control groups

Knee trauma	Case	Control	Total
Trauma +	23 (18.11%)	3 (2.94 %)	26 (11.35 %)
Trauma -	104 (81.89 %)	99 (97.06 %)	203 (88.65%)
Total	127 (55.46 %)	102 (44.54 %)	229 (100%)

The interval between incident of knee trauma and onset of knee OA symptoms in the knee OA group was between 12 and 576 months with a mean value of 220.70 months. Fifteen women in the knee OA group had a history of knee trauma ranging from 24 to 576 months with a mean value of 228.8 months prior to onset of knee OA symptoms while the men in the knee OA group had history of knee trauma ranging from 12 to 396 months with a mean value of 93 months prior to the onset of knee OA symptoms. In the control group, the knee trauma that did not result into knee OA occurred 12 to 360 months prior to examination with mean value of 141.6 months during collection of data. The logistic regression test did not show a significant difference of the interval between incident of knee trauma and onset of knee OA symptoms (r = 0.7361, p = 0.1562). The number of knee trauma experienced by the subjects in the knee OA group ranged from one to 10 times with a mean value of 1.96 times prior to the onset of knee OA symptoms. Fifteen women in the knee OA group experienced one to 10 times knee trauma with a mean value of 2.47 times prior to the onset of knee OA symptoms. Eight men in the knee OA group experienced only one knee trauma with a mean value of one time prior to onset of knee OA symptoms. The control group, who were all men and had knee trauma, had only experienced an average of one time knee trauma without knee OA complaints.

The role of smoking

The distribution of smokers in the case and control groups is shown in table 9. Based on the Fisher's exact test, we found a significant difference between the two groups (p = 0.0389).

Table 9 Distribution of smokers in case and control groups

	Case	Control	Total
Smoker	7 (5.51%)	14 (13.73 %)	21 (9.17 %)
Non-smoker	120 (94.49 %)	88 (86.27 %)	208 (90.83%)
Total	127 (55.46 %)	102 (44.54 %)	229 (100%)

Table 10 shows the distribution of prior smoking habit in the case and control groups. According to the Fisher's exact test, there was as significant difference between the case and control groups (p < 0.0001).

Table 10 Distribution of ex-smoker in case and control groups

	Case	Control	Total
Ex smoker	73 (10.24%)	31 (30.39 %)	44 (21.15 %)
Non-smoker	107 (89.76 %)	57 (69.61 %)	164 (78.85%)
Total	120 (57.69 %)	88 (42.31 %)	208 (100%)

Table 11 shows the distribution of duration of smoking habit in the case and control groups. Six men who were smokers had duration of smoking habit between 60 to 456 months with a mean value of 288 months. In the women case group, one person had duration of smoking habit of 24 months. In the control group, seven male smokers had smoked for 60 to 420 months with a mean value of 271.71 months. The female smokers in the control group had duration of smoking habit of two to 180 months with a mean value of 150.3 months. The independent-sample t test for duration of smoking habit showed no significant difference between the two groups (t = 1.153, *df* 19, p = 0.2632).

Table 11 Duration of smoking in case and control groups

Group	Duration of smoking	Average duration
Case	24-456 months	250,29 months
Control	2-420 months	150,63 months

The duration of smoking cessation in each group can be seen in table 12. The independent-sample t test for duration of smoking habit showed a significant difference between the two groups (t = 2.315, *df* 45, p = 0.0252).

Table 12 Duration of smoking cessation in case and control groups

Group	Duration of smoking cessation	Average
Case	1-446 months	207,92 months
Control	1-588 months	100,77 months

Physical activity involving the knee

Table 13 shows the association between activity involving the knee and knee OA.

Table 13 Physical activities correlation between case and control group

Physical activities	Case		Control group		Odds ratio	P
	n	%	n	%		
Squatting (>30 minutes menit/day)	65	51.18 %	68	66.67%	0.5420	0.0260
Kneeling (>30 minutes/day)	85	66.93 %	29	28.43%	5.0940	<0.0001
Climbing stairs (>10 step/day)	46	36.22 %	46	54.10%	0.6914	0.2201
Weight lifting (>25 kg/day)	7	5.51 %	21	20.59 %	0.2250	0.0009
Walking (>60 minutes/day)	91	71.65 %	75	73.53%	0.9100	0.8673
Standing (>120 minutes/day)	99	77.95 %	86	84.31%	0.6578	0.2420
Sitting (>120 minutes/day)	125	98.43%	99	94.12%	1.8940	0.6581

Multivariate analysis of potential factors

Table 14 shows the result of multivariate analysis in which eight variables had a strong association with the development of knee OA based on the bivariate analysis: age, kneeling, prior history of body weight, prior history of obesity, interval between incident of knee trauma and development of knee OA, duration of smoking habit, duration of current body weight and duration of smoking cessation.

Table 14 Risk factors for development of knee osteoarthritis based in multivariate analyses

Risk Factors	β	SE	P	OR (ajusted)
Kneeling	1.9278	0.4693	0.0000	6.8744
History of overweight	1.2460	0.6236	0.0457	3.4765
History of obesity	1.6342	0.4930	0.0009	5.1254
Interval of knee trauma	0.0669	0.0455	0.1418	1.0691
Duration of recent body weight	-0.1157	0.0298	0.0001	0.8908
Duration of smoking	-0.0075	0.0023	0.0013	0.9925
Duration of smoking cessation	-1.9888	0.6679	0.0029	0.1369
Age	0.1469	0.0259	0.0000	1.1582

DISCUSSION

The role of gender

Most of the OA subjects studied were women. The ratio of women to men was 2.98 to 1. This ratio is slightly higher than the result reported in HANES I.[22] However, this figure was lower than the one reported by Adebajo which is 3.5:1.[3] Other studies, such as conducted by Cushnagan et al,[9] Felson,[25] and Waldron[26] also showed that the female gender had a higher risk. Until today, the explanation for the difference in incidence between the genders is not yet clear. Statistically, the difference of the number of samples based on gender was not significant between the case group and control group so that during the analysis for other factors it was considered not different during calculation based on gender.

The role of age

The prevalence of 5.5 % was the lowest in the study and found in the age group of less than 45 years old. This is similar with a 1987 study conducted by Moskowitz.[27] The highest prevalence (85.82%) was found in the age group of above 50 years old for both genders. A study conducted by Felson et al found similar result, which was a steep increase in prevalence above the age of 60 years old (82%) and similarly for the age group of the seventh, eighth, ninth decade.[25,26]

The prevalence of knee OA radiologically was 80% in the age group of above 55 years old as reported by Altman in 1987 and our study also showed similar result.[20]

Another evidence of the correlation of knee OA with age was reported by Schautten et al who conducted a prospective study for eleven years to determine the effect of knee articular cartilage damage of subjects aged between 46 and 68 years old and found that age was still a main factor in the development of knee OA. Multivariate analysis showed that age was a risk factor for the development of knee OA which was 1.15 times a year. This factor which was already known to be one of the factors in contributing to the development of knee OA and the odd ratio which was not large showed that for the development of more severe or earlier onset of knee OA, interaction with other risk factors was needed.

The role of body weight

Obesity is a risk factor that is significantly correlated with the development of KNEE OA as reported by Cushnagan,[9] Moskowitz,[27] Anderson,[22] Hartz,[6] Spector,[29] and Hart[30]. In our study we found a significant correlation between the presence of obesity and development of knee OA. By analyzing further the prior history of obesity, it was found that there was a significant correlation between history of obesity and prevalence of knee OA. Unfortunately, the subjects in our study could not determine the duration of obesity prior to the development of knee OA because the subjects did not recall how long they had been obese. This analysis should have been necessary to find out whether the weight loss prior to the development of knee OA was a factor that slow down the process of knee OA in the future as reported by Felson.[19] The study conducted by Felson showed that a decrease of two units of body mass index in a 10 year span will reduce the risk by 50%. Evidence that support the duration of body weight as an important factor was reported by Hart in 1993. Hart found that an increase of 5 kg of body weight will increase the risk of developing knee OA by 35%.[30] Until today, it is still difficult to determine at what age the body weight should be manipulated in relation to the development of knee OA.

Analysis of correlation of prior history of overweight and the development of knee OA also showed statistically strong significance. This supported the study performed by Felson in 1988 that overweight or obesity was a risk factor and not as a result of knee OA.[7]

The duration of obesity seems to play a role in the process of the development of knee OA as it is obvious that the heavy biomechanical load/weight of the obese can cause a deviation of body weight force that pass through the knees. The force of the body load will fall toward the median so that it initiates various responses of the knees. In addition, a change of walking pattern occurs that pulls both knees toward each other to the median (bow legged or knocked knee) and thus worsen

the joint damage.

To determine the tendency whether the increase of body weight affects the incidence of knee OA, a logistic regression analysis was performed. We found a linear tendency – the heavier the body weight, the higher risk of developing knee OA ($p < 0.0001$, *df* 1). This was also found in subjects with current obesity or prior history of obesity.

Therefore, body weight is a positive risk factor that needs special attention so that weight reduction is the best method to detect knee OA at the early stage or to prevent knee OA from becoming more severe.

However**,** the multivariate analysis showed only prior history of overweight and prior history of obesity were risk factors for developing knee OA, which were 3 to 5 times. The duration of the current body weight was also one of the risk factors that effected the development of knee OA.

The role of knee trauma

Whether knee trauma is correlated to knee OA is still debatable. Moskowitz found that there was a positive correlation between repetitive knee usages for long periods in men and development of knee OA. The study conducted by Lane et al of female runners did not support the finding, although there was evidence of development of osteophyte or increased bone mass density in this group.[31, 32] Neyret [33] and Doherty [10] who conducted a study on post knee surgery subjects found similar result that there was a 50% increase in prevalence of knee OA. However, the Framingham OA Study did not find any correlation between habitual physical activity and the development of knee OA.[11] Panush et al found similar result.[34] Our study found that knee trauma was significantly correlated to the development of knee OA. If there was knee trauma, the risk for developing knee OA was seven times higher. This was in accordance to the result of study conducted by Kohatsu et al in which knee trauma increased the risk of developing knee OA by five times.[15]

The bivariate analysis showed no significant difference of interval between the incident of knee trauma and development of knee OA. The reason seems to be that usually the interval between the incident of knee trauma and the development of knee OA varied very much, the number of sample was too little, and there were interactions of other risk factors. The multivariate analysis showed this factor as a risk factor that was correlated for the development of knee OA.

The role of smoking

Smokers had lower prevalence or less severity of knee OA as reported by Anderson,[22] Felson,[14] Samanta[35]. The prevalence of knee OA in the study of the effect of smoking in the development of knee OA in 1989 by Felson et al was lower in smokers although adjustments were made for other risk factors such as age, gender, body weight, history of knee trauma or physical activity.[3] Other study that did not support this was reported by Hart et al in the study of generalized knee OA.[24] Unfortunately, the study did not include elderly patients.

In our study, we found a negative correlation or a protective effect of smoking in the development of knee OA. Even former smokers had a lower prevalence of knee OA. However, we should note the duration of smoking cessation of those former smokers prior to the study while for the current smokers, we should note the duration of smoking habit. The multivariate analysis showed that duration of smoking habit and duration of smoking cessation were protective factors in the development of knee OA.

The role of position during defecation

The position during defecation resulting in increased mechanical stress to the knees and its correlation with the prevalence of knee OA has not been studied yet. However, the squatting position and its correlation with the prevalence of knee OA had been studied by Cooper et al in 1994.[24] He found that the squatting position was one of the risk factors for the development of knee OA. In our study, we found no significant difference between the defecating position and the development of knee OA. It was still contentious whether this difference was caused by the duration of less than 30 minutes of squatting during defecation as reported by Cooper. Interaction with others factors also should be noted as the squatting position of more than 30 minutes daily in knee OA patients was shown to be statistically significant in the bivariate analysis although the multivariate analysis did not show any correlation.

The role of physical activity involving the knees

From the various activities involving the knees in knee OA subjects, those whose activities involve squatting of more than 30 minutes daily, kneeling of more than 30 minutes daily, and lifting objects of more than 25 kg daily were risk factors that were statistically significantly different for the development of OA. A study conducted by Cooper et al showed that besides climbing stairs, squatting was a risk factor for the development of knee OA.[24] Lifting heavy objects was a positive risk factor for the development of knee OA. This study was also in accordance to that of Hadi in Semarang.[36] This conclusion must also consider the interaction with other risk factors such as obesity, smoking, etc. After adjustments of other risk factors, only kneeling was a risk factor that was correlated with the development of knee OA and had seven times the risk of developing knee OA.

CONCLUSION

Age, prior history of overweight or prior history of obesity, kneeling, and duration between incident of knee trauma and development of knee OA were risk factors that were correlated with the development of knee OA. Smoking was a negative factor or protective factor for the development of knee OA. The protective effect of smoking was determined by the duration of smoking habit and duration of smoking cessation.

REFERENCES

1. Kalim H . Patogenesis dan diagnosis osteoartritis. In: Current issues and future prospects in osteoartritis. Jakarta: Ikatan Reumatologi Indonesia;1993. 1-22.
2. Mankin HJ. Clinical features of osteoarthritis. In: Kelly WN, Harris ED, Ruddy S, Sledge CB, editors. Textbook of rheumatology. 4th ed. Philadelphia: WB Saunders; 1993. p 1377-84.
3. Adebajo AO. Pattern of osteoarthritis in west african teaching hospital. Ann Rheum Dis. 1991;50:20-22.
4. Moskowitz RW. Osteoarthritis and traumatic condition. In: Katz WA, editor. Rheumatic disease. Diagnosis and management. Philadelphia: JB Lippincott; 1977. p 581-96.
5. Hutton CW. Osteoarthritis: the cause not results of joint failure? Ann Rheum Dis. 48:958-61.
6. Hartz AJ, Fischer ME, Bril G, Kelber S, Rupley Jr D, Oken B, et al. The association of obesity with joint pain and osteoarthritis in the HANES data. J Chron Dis. 1986;39(4):311-9.
7. Felson DT, Anderson JJ, Naimark A, Walker AM, Meenan RF. Obesity and knee osteoarthritis. Ann Int Med. 1988;109:18-24.
8. Chander CL, Spector TD. Oestrogen, joint disease and cartilage. Ann Rheum Dis. 1991;50:139-40.
9. Cushnagan J, Dieppe P. Study of 500 patients with limb joint osteoarthritis. I. Analysis by age, sex, and distribution of symptomatic joint sites. Ann Rheum Dis. 1991;50:8-13.
10. Doherty M, Dipeppe P, Watt I. Influence of primary generalized osteoarthritis on development of secondary osteoarthritis. Lancet. 1983; 2:8-11.
11. Hannan MT, Felson DT, Anderson JJ, naimark a. Habitual physical activity is not associated with knee osteoarthritis: the framingham study. J rheumatol. 1993;20(4):704-9.
12. Knight SM, Ring EFJ, Bahlla AK. Bone mineral density and osteoarthritis. Ann rheum dis.1992;51: 1025-6.
13. Schalwijk J, Joosten LAB, Van Den Berg WB, Van Wyjk JJ, Van De Putte LBA. Insulin like growth factor stimulation chondrocyte proteoglycan synthesis by human synovial fluid. Arthritis Rheum.1989;32:66-71.
14. Felson DT, Anderson JJ, Naimark A, Hannan MT, Kannel WB, Meenan RF. Does smoking protect against osteoarthritis? Arthritis Rheum 1989;32(2):166-72.
15. Kohatsu ND, Schurman DJ. Risk factors for the development of osteoarthritismof the knee. Clin Orthop. 1990;261:242-6.
16. Brown RA. Neovascularization and its role in the osteoarthritis process. Ann Rheum Dis. 1988;47:881-5.
17. Nietard FU. Morphologise und biochemische untersuchungen am knorpelgewebe der diabetischen ratte. Ein modell einer stoffweschelbedingten arthrose. Z orthop. 1986;124(2):125-39.
18. Hochberg MC, Cengju ML, Scott Jr WW, Relichle R, Plato CC, Tobin D. Serum level of insulin growth factor in subject with osteoarthritis in the knee. Arthritis Rheum. 1994;37(8):1177-80.
19. Felson DT, Shang Y, Anthony JM, Naimark A, Anderson JJ. Weight loss reduces the risk for symptomatic knee osteoarthritis in women. Ann Int Med. 1992;116:535-9.
20. Altman RD. Overview of osteoarthritis. Am J Med. 1987;83(suppl 4B):65-9.
21. Felson DT, Zhang Y, Anthony JM ,Naimark A, Andesron JJ. The incidence and natural hystory of knee osteoarthritis in the elderly. Arthritis Rheum. 1995;38:1500-05.
22. Anderson JJ, Felson DT. Factors associated with osteoarthritis of the knee in the first national health and nutrition survey examination (HANES I). J Epidemiol. 1987;128(1):179-89.
23. Van Saase JLCM, Romunde LKJ, Cats A, Vandenbroucke JP, Valkenburg HA. Epidemiology of osteoarthritis: zoetermeer survey. Comparison of radiological osteoarthritis in a dutch population with that in 10 other population. Ann Rheum Dis. 1989;48:271-80.
24. Cooper C, Mc Alindon T, Coggon D, Egger P, Dieppe P. Occupational activity and osteoarthritis of the knee. Ann Rheum Dis. 1994;59:90-3.
25. Felson DT, Naimark A, Anderson J, Kazis L, Castelli W, Meenan RF. The prevalence of the knee osteoarthritis in the elderly. Arthritis Rheum. 1987;30(8):914-7.
26. Waldron HA. Prevalence and distribution of osteoarthritis in a population from Georgian and Early Victorian London. Ann Rheum Dis. 1991;50:301-7.
27. Moskowitz RW. Primary osteoarthritis: epidemiology, clinical aspect, and general managenment. Am J Med. 1987;83(suppl 5A):5-10.
28. Forman MD, Malamet R, Kaplan D. A survey of osteoarthritis of the knee in elderly. J Rheumatol. 1983:10(2):282-7.
29. Spector TD. Epidemiology of the rheumatic disease. Curr Opin Rheumatol. 1993:5(2):132-7.
30. Hart DJ, Spector TD. The relationship of obesity, fat distribution and osteoarthritis in women in the general population: The Chingford Study. J Rheumatol. 1993;20(2):331-5.
31. Lane NE, Bloch DA, Hubert HB, Jones H, Simpson U, Fries JF. Running, osteoarthritis and bone density: initial 2-year longitudinal study. Am J Med. 1990;88:445-9.
32. Lane NE, Bloch DA, Jones HH, Marshal WH, Wood PD, Fries JF. Long distance running bone density and osteoarthritis. JAMA. 1986;255(9):1147-54.
33. Neyret P, Donnel ST, Dejour H. Osteoarthritis of the knees following mensectomy. Br J Rheum. 1994;33:267-8.
34. Panush RS, Schmidt C, Caldwell Jr, Edward NL, Longley S, Yonker R. Is running associated with degenerative joint diseases? JAMA 1986;255(9):1152-4.
35. Samanta A, Jones A, Regan M, Wilson S, Doherty M. Is osteoarthritis in women affected by hormonal change or smoking? Br J Rheumatol. 1993;32(5):366-70.
36. Hadi S, Soenarto, Nasution AR, Isbagio H, Daud R. Mechanical risk factors of knee osteoartthritis in Bandungan village, Semarang Indonesia. 1995.