Index Analysis of Osteonecrosis and Related Causes

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Research

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Abstract

Background: The pathogenesis of non-traumatic osteonecrosis of the femoral head (ONFH) is related to the interruption of blood supply caused by lipid metabolism and hypercoagulability. The purpose of this study was to explore the relationship between clinical biochemical parameters and non-traumatic ONFH.

Methods: The basic information and biochemical indexes of 1292 patients with non- traumatic ONFH and 1880 healthy controls were collected. SPSS software (version 22.0) was used to process and analyze the data. T-test was used for quantitative analysis. Chi-square test was used for categorical variables.$p< 0.05$ were the index with statistical significance.

Results: In the population sample, TC ($p= 0.00004$), LDL ($p= 0.014$) and PLT ($p= 0.000005$) levels were statistically significant between the two groups.In men, levels of TC ($p = 0.004$), LDL ($p= 0.011$), and PLT ($p= 0.00005$) were statistically significant between the two groups.In women, TC ($p= 0.001$) and PLT ($p= 0.048$) levels were statistically significant between the two groups.There were differences in TC ($p= 0.00001$) and PLT ($p= 0.031$) levels between the case group and the control group in samples aged less than 45 years.There were differences in LDL ($p= 0.00002$) and PLT ($p= 0.022$) levels between the two groups in samples older than 45 years.Compared with the control group, patients with alcohol-induced ONFH had HDL ($p = 0.002$).LDL ($p= 0.00002$);The level of PLT ($p= 0.0001$) was significantly different.HDL ($p = 0.005$) was found in alcohol-induced ONFH patients younger than 45 years of age.The PLT level ($p=0.045$) was different from that of the control group.There was a difference in LDL ($p= 0.000003$) levels between control and alcohol-induced ONFH patients older than 45 years.The older the onset age, the TC in vivo;HDL;LDL;ApoA1;ApoB;The lower the PLT level.With the prolongation of the onset time, the PLT level in the patient's body is decreasing continuously.

Conclusion: The changes of biochemical indexes are closely related to the occurrence of non-traumatic ONFH. Our research can provide a new direction for the prevention of ONFH.

1. Introduction

Osteonecrosis of the femoral head (ONFH) is a destructive bone disease caused by many factors. Because of its special anatomical structure, the interruption of bone blood supply is the most important cause[1, 2]. At present, there are about 8.12 million patients in China[3]. In recent years, with the abuse of alcohol and hormone, the proportion of patients with non-traumatic ONFH has increased significantly. An epidemiological study of osteonecrosis in China showed that steroid-induced and alcoholic-induced ONFH accounted for 54.8%[4]. The prompt onset of non-traumatic ONFH is not easy to detect, and the progress is rapid, so a large number of late patients can only accept hip replacement surgery[5, 6]. Therefore, the study of the etiology and pathogenesis of ONFH can provide the basis for timely diagnosis.

About its pathogenesis, scholars at home and abroad have put forward many theories，of which lipid metabolism abnormalities and thrombosis have been recognized by most scholars[7-9]. Studies have found that heavy drinking and excessive use of corticosteroids may lead to abnormal lipid metabolism in the general population[10]. Therefore, we usually think that the osteonecrosis caused by the two has the same pathogenesis. Alcoholism and hormone use lead to disorder of lipid metabolism, increase of blood viscosity, accumulation of fat in bone cells, steatosis of bone cells, contraction of minute blood vessels and damage of endothelial cells. These changes eventually lead to slow down of blood flow in blood vessels of nutritional bone cells, blood coagulation, increase of intraosseous pressure and decrease or blockage of short blood vessels in the femoral head, And sometimes lead to vascular necrosis of bone[10, 11].Through literature review, it was found that some biochemical changes increased the risk of ONFH[12]. The purpose of this study is to understand the role of biochemical indicators in the occurrence of non-traumatic ONFH by analyzing the clinical biochemical indexes of patients with alcoholic and steroid induced ONFH and healthy people, to provide a basis for the prevention of early osteonecrosis.

2. Materials And Methods

2.1 Research object

From 2015 to 2019, a total of 1292 patients with non traumatic ONFH were enrolled in the study. These included patients with steroid-induced ONFH and patients with alcoholic-induced ONFH. Steroid-induced ONFH was diagnosed as steroid use for better than two months, with an average daily dose of > 20 mg, or a cumulative dose of > 2000 mg prednisone. Alcoholic-induced ONFH was diagnosed as drink greater than 400 milliliters (or 320 grams) a week for a year[4]. All patients were examined before and after and

frog type X-ray examination. MRI confirmed the diagnosis of ONFH.
At the same time, 1880 healthy people without kinship in Zhengzhou traditional Chinese's medicine trauma hospitals in recent years were taken as control. All subjects were Han's nationality. We excluded ONFH caused by steroid induced and alcoholism, traumatic hip dislocation or other hip diseases, and obvious familial genetic diseases. Subjects with chronic diseases and diseases involving the brain, heart, liver, lung and other important organs were excluded from the study.
In this study, the ethics committee of Zhengzhou trauma hospital of traditional Chinese's medicine agreed and signed a written informed consent.

2.2 Method

In the case group, peripheral venous blood samples were collected from patients with fasting for more than 12 hours in a quiet state in the morning. The elemental information and biochemical indexes of patients were collected: total cholesterol (TC), triglyceride (TG), high-density lipoprotein protein (HDL), low-density lipoprotein (LDL), apolipoprotein protein A1 (ApoA1), apolipoprotein protein B (ApoB), plasma hemoglobin time (PT); activatedpartialthromboplastintime（APTT）;erythrocytesedimentationrate（ESR）;C-reactionprotein（CRP）;bloodplatelet（PLT） Blood lipid index was detected by Roche cobas8000-701 biochemical analyzer, and coagulation index was detected by leatherneck CA-7000 coagulation instrument. In the control group, peripheral venous blood was collected for basic information and five biochemical indexes (TC; TG; HDL; LDL; PLT). In the end, two people collected the required data by setting up an Excel sheet for two people and collating the data through. software.

2.3 Statistical analysis

SPSS software (version 22.0) was used to process and analyze the data. All continuous data were expressed as mean ± standard deviation (SD); quantitative variables were analyzed by double tailed T-test. Chi-square test was used for categorical variables. Fisher's exact test is used to analyze the situation that the expected frequency is not reached. Determine the indicators with statistical significance. Double tail p value < 0.05 were considered to be statistically significant.

3. Result

3.1 Demographic characteristics

A total of 1292 patients (233 females and 1059 males) and 1880 controls (350 females and 1530 males) were enrolled in the study. The average age of the patients was 43.09 ± 11.51 years aged and 48.33 ± 10.66 years elderly in the control group. There was no difference in gender (p> 0.05) between the patients and the control group, as shown in Figure. 1.

3.2 Analysis of blood biochemical indexes in case group and control

Group

Five biochemical indexes (TC; TG; HDL; LDL; PLT) were selected to compare the differences between the osteonecrosis group and the control group, as shown in Table 1. In the overall sample, TC (p = 0.00004), LDL (p = 0.014) and PLT (p = 0.000005) levels were statistically significant between the two groups In men, TC (p= 0.004), LDL (p= 0.011) and PLT (p= 0.00005) levels were statistically significant between the two groups In women, TC (p= 0.001) and PLT (p= 0.048) levels were statistically significant between the two groups. There were differences in TC (p= 0.00001) and PLT (p= 0.031) levels between the case group, and the control group in the samples aged < 45 years. There were differences in LDL (p= 0.00002) and PLT (p= 0.022) levels between the two groups in the samples aged > 45 years. Compared with the control group, the levels of TC, LDL and PLT were significantly different. In patients with avascular necrosis of the femoral head, the levels of TC decreased, while the levels of LDL and PLT increased. (Table 1)

3.3 Analysis of blood biochemical indexes in patients with ONFH

We stratified the osteonecrosis samples (gender, age, hip lesions, course of disease, and osteonecrosis stage). Eleven clinical biochemical indexes, including TC; TG; HDL; LDL; ApoA1; ApoB; PT; APTT; ESR; CRP; PLT was selected for analysis in each layer, as shown in Table 2. The levels of TG, HDL, LDL, ApoA1, ApoB, APTT, ESR and PLT were different between male and female. The levels of TG, LDL, ApoB, APTT and PLT in male were much taller than that in female, while HDL, ApoA1 and ESR were taller in female than in mail. TC; TG; HDL; LDL; ApoA1; ApoB; APTT; ESR; CRP and PLT were statistically significant in different age groups. The older the age of onset, the lower the level of TC; HDL; LDL; ApoA1; ApoB; PLT. However, with the extension of the onset time, the level of PLT in

patients decreased continuously. Through the study of unilateral and bilateral osteonecrosis, there were differences in HDL and ApoA1 levels. The levels of LDL, ApoA1, apoB and APTT were significantly unlike in distinctive stages of osteonecrosis. (Table 2)
We further compared the stages of osteonecrosis with the levels of APTT and found that the levels of APTT were unusual in unusual stages, as shown in Table 3. In the study of hip lesions, we found that CRP;ApoB;ApoA1;HDL levels were statistically significant, as shown in Table 4. The levels of CRP and PLT were also different in the different course of disease, as shown in Table 5.

3.4 Biochemical analysis between alcohol-induced ONFH group and control group

The difference analysis of indexes between the alcohol-induced ONFH group and the control group is shown in Table 6. Compared with the control group, the levels of HDL (p= 0.002), LDL (p= 0.000) and PLT (p= 0.000) were significantly different in patients with alcohol-induced ONFH There were significant differences in HDL (p= 0.005) and PLT (p= 0.045) levels between the two groups. LDL (p= 0.000) levels were different between the two groups. (Table 6)

3.5 Analysis of blood biochemical indexes in patients with alcohol-induced ONFH

The levels of TC, HDL, LDL, ApoB, ESR and PLT were different among different age groups. The older the onset age, the higher the levels of TC, HDL, LDL and ESR, the lower the level of PLT. In the study of biochemical indexes among distinctive courses of disease, it was found that the levels of ESR and PLT were significantly distinct among different courses. There were significant differences in the levels of ApoA1; ESR; CRP and PLT between unilateral and bilateral osteonecrosis. There were different stages of osteonecrosis, TG, LDL and ApoA1 levels. (Table 7)

3.6 Analysis of blood biochemical indexes in patients with steroid-induced ONFH

There were differences in HDL; ApoA1; APTT and ESR levels between male and female patients with ONFH. The level of APTT in male was much taller than that in female, HDL; ApoA1 and ESR were taller in female than in mail. TC; HDL; LDL; ApoA1; ApoB; APTT and ESR were statistically significant in different age groups. The older the age of onset, the lower the level of TC; HDL; LDL; ApoA1; ApoB. Through the study of unilateral and bilateral osteonecrosis, it was found that the levels of TG, HDL, LDL and ApoA1 were different. (Table 8)

4. Discussion

The etiology of non-traumatic ONFH is not clear, but the biochemical indicators in patients may play a certain role. Abnormal biochemical level in patients may cause lipid metabolism disorder and hypercoagulable state of blood, which may interrupt blood supply of femoral head and lead to osteonecrosis. In this study, we found that the levels of TC, LDL and PLT in ONFH patients were significantly changed compared with normal subjects.
The disorder of lipid metabolism refers to the increase or decrease of one or more lipid components, the change of lipoprotein and (or) quantity in plasma. The common lipid metabolism disorders in the clinic include hypertriglyceridemia, hypercholesterolemia, high and low-density lipoprotein, etc[13] The change of these indexes or the appearance of symptoms may lead to the accumulation of a large number of plump globules in the medullary cavity, resulting in ischemic changes of bone marrow. In the study of steroid-induced ONFH, it was found that after the injection of adrenocortical hormone into rabbits, a large number of plump globules were found in the blood vessels of medullary cavity under the microscope, while no-fat globules were found in the medullary cavity of the control group [14]. Studies have confirmed that excessive use of corticosteroids can lead to ONFH and dyslipidemia[15, 16]. Systemic lupus erythematosus is a disease that needs steroid therapy for a long time. In the study of biochemical indicators of osteonecrosis caused by the disease, it was found that the serum total cholesterol level increased rapidly and reached the peak within one month. The speedy increase of serum cholesterol level leads to the quick increase of adipocytes and / or vascular damage in the femoral head bone marrow[17]. Motomura et al.[12] found that plasma TG level and LDL increase were associated with increased risk of steroid-induced ONFH. Our previous study on alcohol-induced ONFH found that TC / HDL was statistically significant compared with the control group [18]. Our study found that decreased TC levels and increased LDL levels increased the risk of avascular ONFH. The levels of HDL and LDL affect the occurrence of alcohol-induced ONFH.
It is well known that endothelial cells in the inner layer of the vascular wall are essential for vascular homeostasis. Endothelial cell injury can lead to abnormal coagulation and thrombosis, leading to downstream ischemia [19, 20]. Under normal circumstances, vascular endothelial cells produce PGI2, and its metabolites can cause vasodilation and inhibit platelet aggregation [10]. Glucocorticoids can induce endothelial cell apoptosis through different signal pathways. Apoptotic endothelial cells stimulate the

adhesion of platelets to endothelial cells and activate platelets, which eventually leads to thrombosis [21, 22]. Thrombotic and fibrinolytic dysfunction are considered to be the factors leading to osteonecrosis, which are caused by the decreased ability of thrombolysis and the increased possibility of thrombosis[2, 23]. We found that PLT levels in ONFH patients were importantly higher than those in normal controls, while PLT levels were gradually decreased with the onset of ONFH. In patients, the level of manly platelet was meaningfully lower than that of female, and the number of manly patients was much more than that of female, indicating that platelet level had a greater impact on osteonecrosis in men.

ONFH is a refractory disease. Timely diagnosis and intervention are the key to the treatment when the reversible lesions of the femoral head occur. If the collapse of the femoral head occurs, even if active treatment measures are taken, the patients will eventually have to undergo joint replacement surgery. However, in the early stage of ONFH. The clinical manifestations are lacked of specificity, and the negative X-ray findings of the hip will lead to missed diagnosis of the disease. Early prevention is a good choice. It has been reported that warfarin, and lipid-lowering drugs have a certain preventive effect on steroid-induced ONFH [24, 25]. We can take the detection of biochemical indicators as a screening tool for ONFH high-risk population, and monitor the biochemical level of people who use hormones and long-term drinking, and achieve the purpose of early prevention through drug intervention.

The sample size of the cases in this study comes from one hospital. The coverage is not wide enough. The selection is limited, and there is no strong representative. Although the statistical method is used, there may be correlation bias. In the later stage, we need to further expand the sample size, conduct multi center, large sample survey, and get more objective and comprehensive results.

Conclusion

Our case-control study found significant changes in THE levels of TC, LDL, and PLT in patients with non-traumatic femoral head necrosis.We can monitor the biochemical level of people who use hormones and drink alcohol for a long time, and achieve the goal of early prevention through drug intervention.

Abbreviations

Osteonecrosis of the femoral head (ONFH)

Total cholesterol (TC)

Triglyceride (TG)

High-density lipoprotein protein (HDL)

Low-density lipoprotein (LDL)

Apolipoprotein protein A1 (ApoA1)

Apolipoprotein protein B (ApoB)

plasma hemoglobin time (PT)

activatedpartialthromboplastintime （APTT）

erythrocytesedimentationrate （ESR）

C-reactionprotein （CRP）

Bloodplatelet （PLT）

Declarations

Ethical Approval

Ethics committee approval was received for this study from Zhengzhou Traditional Chinese Medicine Traumatology Hospital.All participants provided written informed consent.

Conflicts of Interest

The authors declare there are no conflicts of interest.

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Author's contribution

- Tingting Liu and Yuju Cao performed the experiments, analyzed the data, prepared

figures and/or tables, authored or reviewed drafts of the paper, approved the final draft.

- Huiqiang Wu, Feimeng An, Tiantian Wang and Menghu Sun collected and counted the data.
- Changxu Han, Ye Tian conducted data analysis and approved the final draft.
- Kunzheng Wang conceived and designed the experiments, authored or reviewed drafts of the paper, approved the final draft.
  • Jianzhong Wang conceived and designed the experiments, contributed analysis tools, authored or reviewed drafts of the paper, approved the final draft.

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Tables

Table 1
Comparative study of biochemical indexes in the necrosis group and the control group

Variables	Type	Cases		Controls		p value
		N	Mean ± SD	N	Mean ± SD	
Total	TC	944	4.62 ± 10.93	1876	4.78 ± 1.00	0.00004
	TG	944	1.94 ± 1.48	1876	1.99 ± 1.57	0.405
	HDL	945	1.09 ± 0.28	1880	1.09 ± 0.40	0.977
	LDL	945	2.75 ± 0.77	1880	2.67 ± 0.79	0.014
	PLT	944	288.69 ± 62.69	1877	218.13 ± 55.16	0.000005
Male	TC	764	4.64 ± 0.93	1527	4.76 ± 1.00	0.004
	TG	764	2.02 ± 1.55	1526	2.09 ± 1.62	0.338
	HDL	764	1.04 ± 0.25	1530	1.06 ± 0.38	0.253
	LDL	764	2.79 ± 0.77	1530	2.71 ± 0.79	0.011
	PLT	763	225.82 ± 61.29	1528	215.65 ± 53.67	0.00005
female	TC	180	4.56 ± 0.94	349	4.87 ± 1.03	0.001
	TG	180	1.58 ± 1.08	350	1.55 ± 1.22	0.776
	HDL	181	1.28 ± 0.30	350	1.22 ± 0.43	0.069
	LDL	181	2.56 ± 0.72	350	2.53 ± 0.81	0.617
	PLT	181	240.78 ± 67.11	349	228 ± 60.14	0.048
Age(yr)<45	TC	535	4.49 ± 0.94	632	4.74 ± 0.98	0.00001
	TG	536	2.02 ± 1.64	631	2.10 ± 1.58	0.410
	HDL	537	1.06 ± 0.27	633	1.06 ± 0.30	0.929
	LDL	537	2.66 ± 0.79	633	2.67 ± 0.80	0.926
	PLT	536	233.83 ± 62.23	631	226.37 ± 54.65	0.031
Age(yr)≥45	TC	409	4.79 ± 0.90	1244	4.80 ± 1.01	0.801
	TG	408	1.83 ± 1.24	1245	1.93 ± 1.56	0.224
	HDL	408	1.12 ± 0.28	1247	1.10 ± 0.44	0.472
	LDL	408	2.86 ± 0.73	1247	2.67 ± 0.79	0.00002
	PLT	408	221.94 ± 62.73	1246	213.95 ± 54.96	0.022

Table 2

Case group index analysis

Variables		Mean ± SD										
		TC	TG	HDL	LDL	ApoA1	ApoB	PT	APTT	ESR	CRP	PLT
Gender	Male	4.64 ± 0.93	2.02 ± 1.55	1.04 ± 0.25	2.79 ± 0.77	1.18 ± 0.20	0.98 ± 0.30	11.89 ± 2.42	33.81 ± 5.96	13.54 ± 12.90	7.85 ± 13.18	225.82 ± 61.29
	female	4.56 ± 0.94	1.58 ± 1.08	1.28 ± 0.30	2.56 ± 0.72	1.29 ± 0.20	0.90 ± 0.50	11.54 ± 1.16	32.22 ± 5.10	23.19 ± 18.12	8.17 ± 12.42	240.78 ± 67.11
	p value	0.299	0.0003	0.000	0.0003	0.000	0.004	0.115	0.005	0.000	0.849	0.007
Age(yr)	≤30	4.19 ± 0.87	1.54 ± 1.19	1.12 ± 0.31	2.40 ± 0.72	1.19 ± 0.19	0.82 ± 0.24	12.14 ± 1.13	35.49 ± 7.20	13.45 ± 15.27	8.16 ± 12.47	245.14 ± 66.25
	30–40	4.44 ± 0.92	2.08 ± 1.81	1.04 ± 0.27	2.65 ± 0.75	1.17 ± 0.21	0.92 ± 0.26	11.95 ± 4.01	33.39 ± 5.74	11.47 ± 9.77	5.31 ± 7.37	231.16 ± 60.75
	40–50	4.79 ± 0.89	2.11 ± 1.43	1.05 ± 0.24	2.84 ± 0.78	1.20 ± 0.20	1.01 ± 0.31	11.70 ± 1.13	33.67 ± 5.74	15.21 ± 14.60	9.94 ± 17.99	230.43 ± 58.93
	50–60	4.75 ± 0.95	1.89 ± 1.37	1.13 ± 0.27	2.85 ± 0.70	1.24 ± 0.21	1.03 ± 0.51	11.79 ± 1.22	32.64 ± 5.39	17.26 ± 13.85	6.94 ± 8.13	217.44 ± 64.58
	≥ 60	4.97 ± 0.85	1.50 ± 0.86	1.20 ± 0.30	3.05 ± 0.78	1.28 ± 0.22	1.06 ± 0.34	11.75 ± 0.98	33.06 ± 5.01	20.91 ± 19.44	9.07 ± 11.64	213.77 ± 66.57
	p value	0.000	0.0002	0.00001	0.000	0.0001	0.000	0.461	0.003	0.000	0.049	0.001
Course, months	≤12	4.64 ± 0.97	2.02 ± 1.52	1.04 ± 0.26	2.75 ± 0.81	1.21 ± 0.22	0.98 ± 0.31	12.08 ± 1.12	34.34 ± 6.24	15.38 ± 13.82	8.50 ± 11.34	234.49 ± 60.31
	12–24	4.72 ± 0.93	2.07 ± 1.38	1.03 ± 0.25	2.86 ± 0.85	1.21 ± 0.24	1.05 ± 0.53	11.92 ± 1.11	33.64 ± 6.75	13.48 ± 12.16	7.46 ± 8.94	237.27 ± 61.44
	24–36	4.73 ± 0.92	2.01 ± 1.64	1.03 ± 0.23	2.83 ± 0.76	1.19 ± 0.22	0.99 ± 0.27	12.38 ± 5.41	33.81 ± 5.62	14.57 ± 14.08	7.94 ± 13.26	233.30 ± 61.58
	≥ 36	4.65 ± 0.92	1.88 ± 1.14	1.07 ± 0.27	2.79 ± 0.78	1.23 ± 0.22	1.01 ± 0.37	11.82 ± 1.08	34.24 ± 5.83	14.52 ± 16.03	12.43 ± 23.63	212.14 ± 71.21
	p value	0.772	0.676	0.452	0.555	0.416	0.278	0.281	0.644	0.588	0.148	0.002
Hip lesions	Unilateral	4.79 ± 0.91	1.86 ± 1.23	1.09 ± 0.25	2.90 ± 0.83	1.27 ± 0.24	1.01 ± 0.26	11.92 ± 1.23	33.89 ± 5.74	13.12 ± 13.31	9.88 ± 19.90	224.62 ± 60.77
	Bilateral	4.65 ± 0.95	2.05 ± 1.49	1.03 ± 0.25	2.77 ± 0.80	1.20 ± 0.22	1.00 ± 0.41	12.07 ± 2.74	34.08 ± 6.32	15.00 ± 14.08	8.57 ± 12.27	232.18 ± 64.63
	p value	0.082	0.145	0.004	0.092	0.0002	0.923	0.520	0.732	0.150	0.483	0.195
Clinical stages	Stage Ⅰ	4.70 ± 1.00	2.19 ± 1.72	1.03 ± 0.27	2.70 ± 0.81	1.22 ± 0.23	0.97 ± 0.26	11.94 ± 1.08	34.69 ± 6.13	15.47 ± 14.63	8.29 ± 10.73	234.01 ± 61.49

Variables		Mean ± SD										
		TC	TG	HDL	LDL	ApoA1	ApoB	PT	APTT	ESR	CRP	PLT
	Stage Ⅱ	4.69 ± 0.91	1.96 ± 1.41	1.04 ± 0.25	2.90 ± 0.79	1.18 ± 0.21	0.98 ± 0.25	11.91 ± 1.13	33.20 ± 5.86	14.31 ± 13.48	9.88 ± 17.74	230.23 ± 61.84
	Stage Ⅲ	4.66 ± 0.95	1.86 ± 1.14	1.06 ± 0.26	2.78 ± 0.82	1.26 ± 0.24	1.07 ± 0.60	11.91 ± 1.08	34.11 ± 6.44	14.33 ± 14.32	7.92 ± 10.33	227.34 ± 63.20
	p value	0.895	0.069	0.498	0.026	0.001	0.015	0.946	0.032	0.652	0.490	0.594

Table 3
The stage of clinical was compared with APTT

		APTT			Total
		0	1	2	
Clinical stages	2	2	119	59	180
	3	2	220	66	288
	4	5	129	62	196
Total		9	468	187	664

Table 4
The pathological changes of hip joint were compared with biochemical indexes

		Hip lesions		P
		0	1	
CRP	0	108	336	0.038
	1	39	188	
ApoB	0	6	37	0.002
	1	85	356	
	2	65	149	
ApoA1	0	14	90	0.012
	1	127	425	
	2	15	28	
HDL	0	74	329	0.004
	1	82	215	

Table 5
The course was compared with biochemical indexes

		PLT			CRP	
		0	1	2	0	1
Course	0	7	225	10	143	100
	1	7	169	8	124	55
	2	1	120	4	82	32
	3	14	125	4	97	40
p		0.011			0.022	

Table 6
Comparison between alcoholic osteonecrosis group and control group

	Type	case/control	control	case
			Mean ± SD	Mean ± SD
Total	TC	496/1527	4.76 ± 0.95	4.75 ± 0.94
	TG	496/1526	2.10 ± 1.61	2.08 ± 1.43
	HDL	496/1530	1.09 ± 0.38	1.03 ± 0.25*
	LDL	496/1530	2.70 ± 0.73	2.86 ± 0.82*
	PLT	487/1528	215.65 ± 53.67	226.75 ± 58.75*
Age(yr)<45	TC	285/532	4.79 ± 0.94	4.67 ± 0.96
	TG	286/531	2.24 ± 1.64	2.18 ± 1.50
	HDL	286/533	1.07 ± 0.28	1.01 ± 0.23*
	LDL	286/533	2.74 ± 0.73	2.81 ± 0.84
	PLT	281/531	224.64 ± 54.49	232.71 ± 54.35*
Age(yr)≥45	TC	211/995	4.75 ± 0.96	4.84 ± 0.90
	TG	210/995	2.02 ± 1.59	1.95 ± 1.31
	HDL	210/997	1.10 ± 0.42	1.05 ± 0.26
	LDL	210/997	2.67 ± 0.73	2.94 ± 0.77*
	PLT	206/997	210.86 ± 52.63	218.62 ± 63.51

Table 7

Alcohol-induced osteonecrosis of femoral head group index analysis

Variables		Mean ± SD										
		TC	TG	HDL	LDL	ApoA1	ApoB	PT	APTT	ESR	CRP	PLT
Age(yr)	□30	4.48 ± 1.00	1.93 ± 1.63	1.01 ± 0.23	2.60 ± 0.87	1.16 ± 0.22	0.92 ± 0.29	12.19 ± 1.17	34.97 ± 8.19	9.14 ± 6.60	6.85 ± 8.00	241.31 ± 55.42
	30–40	4.52 ± 0.94	2.12 ± 1.42	0.99 ± 0.22	2.73 ± 0.80	1.17 ± 0.23	0.95 ± 0.24	11.94 ± 1.06	33.84 ± 6.11	10.63 ± 9.09	6.12 ± 8.87	234.30 ± 54.55
	40–50	4.91 ± 0.87	2.20 ± 1.37	1.03 ± 0.25	2.96 ± 0.83	1.20 ± 0.22	1.07 ± 0.35	11.85 ± 1.10	34.10 ± 5.69	12.47 ± 10.97	9.35 ± 13.08	230.27 ± 55.57
	50–60	4.81 ± 1.01	2.06 ± 1.60	1.06 ± 0.27	2.94 ± 0.76	1.23 ± 0.21	1.06 ± 0.34	11.94 ± 1.10	33.24 ± 5.84	14.84 ± 12.04	6.74 ± 6.76	208.87 ± 61.56
	≥ 60	4.92 ± 0.82	1.60 ± 0.78	1.13 ± 0.31	3.02 ± 0.79	1.26 ± 0.20	1.05 ± 0.23	12.05 ± 0.93	33.78 ± 5.34	20.19 ± 16.92	9.77 ± 12.52	211.33 ± 74.56
	p value	0.001	0.241	0.039	0.011	0.129	0.003	0.429	0.623	0.00002	0.260	0.002
Course, months	□12	4.66 ± 0.97	2.14 ± 1.59	1.02 ± 0.25	2.75 ± 0.82	1.20 ± 0.22	0.97 ± 0.28	12.03 ± 1.00	34.31 ± 6.23	14.18 ± 12.02	8.31 ± 11.39	231.06 ± 56.67
	12–24	4.79 ± 0.98	2.11 ± 1.39	1.02 ± 0.26	2.95 ± 0.88	1.19 ± 0.24	1.04 ± 0.29	11.94 ± 1.16	33.43 ± 6.45	11.97 ± 10.41	6.72 ± 7.77	232.91 ± 54.86
	24–36	4.85 ± 0.89	2.10 ± 1.52	1.01 ± 0.21	2.97 ± 0.72	1.16 ± 0.19	1.04 ± 0.27	11.81 ± 1.10	33.55 ± 5.50	13.19 ± 12.76	6.34 ± 6.35	226.58 ± 59.74
	≥ 36	4.76 ± 0.87	1.91 ± 1.00	1.07 ± 0.27	2.89 ± 0.78	1.23 ± 0.22	1.06 ± 0.42	11.82 ± 1.14	34.02 ± 5.57	9.85 ± 8.04	9.41 ± 15.00	209.42 ± 58.75
	p value	0.417	0.609	0.356	0.071	0.190	0.120	0.321	0.585	0.022	0.460	0.015
Hip lesions	Unilateral	4.88 ± 0.91	2.05 ± 1.36	1.06 ± 0.23	2.93 ± 0.86	1.26 ± 0.24	1.04 ± 0.26	11.86 ± 1.04	33.89 ± 5.35	10.44 ± 7.79	5.04 ± 4.21	215.53 ± 49.18
	Bilateral	4.71 ± 0.95	2.09 ± 1.45	1.02 ± 0.25	2.85 ± 0.81	1.18 ± 0.21	1.02 ± 0.32	11.95 ± 1.10	33.91 ± 6.23	13.22 ± 11.81	8.31 ± 11.29	229.66 ± 60.68
	p value	0.111	0.777	0.218	0.372	0.001	0.551	0.508	0.981	0.007	0.001	0.035
Clinical stages	Stage □	4.81 ± 1.01	2.40 ± 1.78	1.03 ± 0.27	2.74 ± 0.82	1.22 ± 0.24	0.99 ± 0.26	11.85 ± 0.97	34.28 ± 5.91	13.32 ± 11.12	8.01 ± 10.85	229.86 ± 56.98
	Stage □	4.74 ± 0.89	1.98 ± 1.31	1.02 ± 0.22	2.99 ± 0.83	1.15 ± 0.18	1.01 ± 0.26	11.88 ± 1.06	33.20 ± 6.04	12.84 ± 11.20	8.11 ± 11.42	227.92 ± 52.02
	Stage □	4.71 ± 0.96	1.94 ± 1.20	1.06 ± 0.27	2.83 ± 0.80	1.25 ± 0.24	1.06 ± 0.42	12.00 ± 1.13	33.97 ± 6.05	11.71 ± 11.18	7.12 ± 8.97	223.58 ± 66.00
	p value	0.686	0.011	0.437	0.021	0.00008	0.167	0.478	0.243	0.484	0.832	0.655

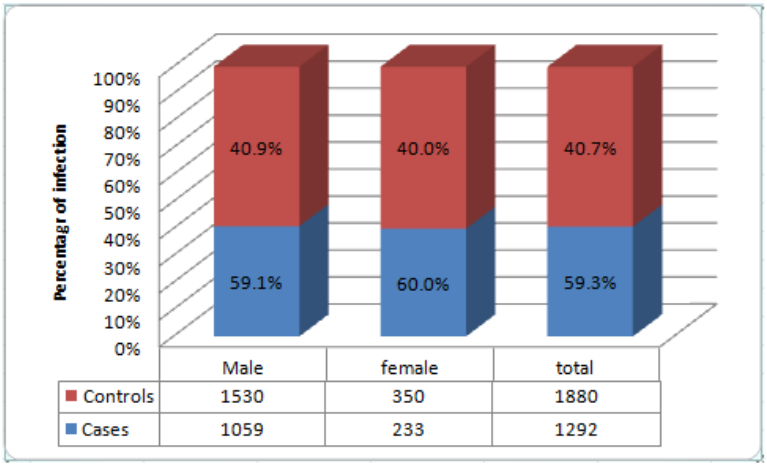
Table 8

Steroid-induced osteonecrosis of the femoral head group index analysis

Variables		Mean ± SD										
		TC	TG	HDL	LDL	ApoA1	ApoB	PT	APTT	ESR	CRP	PLT
Age(yr)	□30	4.05 ± 0.76	1.49 ± 0.89	1.04 ± 0.25	2.32 ± 0.65	1.21 ± 0.19	0.79 ± 0.19	12.47 ± 1.11	37.30 ± 6.93	18.05 ± 18.04	9.44 ± 10.93	250.44 ± 81.21
	30–40	4.38 ± 0.97	2.08 ± 2.01	1.01 ± 0.25	2.56 ± 0.79	1.18 ± 0.23	0.92 ± 0.39	12.91 ± 8.39	33.91 ± 5.75	12.33 ± 11.56	4.94 ± 4.12	234.71 ± 72.65
	40–50	4.63 ± 0.90	1.97 ± 1.46	1.03 ± 0.23	2.69 ± 0.73	1.24 ± 0.23	0.96 ± 0.24	11.90 ± 1.33	33.77 ± 7.24	22.54 ± 21.48	17.63 ± 31.52	238.88 ± 73.96
	50–60	4.75 ± 0.86	1.89 ± 1.04	1.13 ± 0.33	2.86 ± 0.64	1.30 ± 0.27	1.20 ± 1.09	12.20 ± 1.53	33.21 ± 5.46	23.30 ± 18.88	8.52 ± 10.00	234.22 ± 72.90
	≥ 60	5.03 ± 0.88	1.48 ± 1.20	1.24 ± 0.27	3.12 ± 0.86	1.38 ± 0.25	1.14 ± 0.53	11.65 ± 1.02	33.95 ± 5.43	18.10 ± 16.32	7.87 ± 10.50	227.41 ± 65.81
	p value	0.0002	0.224	0.006	0.0004	0.008	0.007	0.716	0.020	0.039	0.177	0.760
Course, months	□12	4.55 ± 1.01	1.60 ± 1.18	1.07 ± 0.29	2.75 ± 0.80	1.23 ± 0.24	0.97 ± 0.40	12.24 ± 1.46	34.44 ± 6.41	19.29 ± 18.32	9.07 ± 11.48	244.55 ± 70.40
	12–24	4.48 ± 0.75	1.98 ± 1.39	1.05 ± 0.25	2.59 ± 0.70	1.25 ± 0.23	1.08 ± 0.90	11.85 ± 0.97	34.24 ± 7.55	16.56 ± 15.03	7.96 ± 9.19	249.12 ± 75.41
	24–36	4.51 ± 0.93	1.89 ± 1.85	1.07 ± 0.26	2.60 ± 0.78	1.24 ± 0.25	0.89 ± 0.25	13.36 ± 8.77	34.23 ± 5.90	16.10 ± 15.09	11.29 ± 20.94	243.63 ± 64.04
	≥ 36	4.47 ± 0.98	1.86 ± 1.37	1.07 ± 0.27	2.62 ± 0.76	1.24 ± 0.24	0.91 ± 0.24	11.84 ± 0.95	34.67 ± 6.31	22.73 ± 22.26	16.78 ± 32.17	217.04 ± 82.07
	p value	0.961	0.583	0.963	0.708	0.991	0.276	0.263	0.985	0.267	0.360	0.114
Hip lesions	Unilateral	4.64 ± 0.92	1.54 ± 0.87	1.15 ± 0.26	2.83 ± 0.79	1.29 ± 0.26	0.96 ± 0.24	12.04 ± 1.49	33.93 ± 6.47	17.14 ± 18.39	17.25 ± 30.25	238.26 ± 74.14
	Bilateral	4.45 ± 0.92	1.94 ± 1.60	1.03 ± 0.26	2.57 ± 0.74	1.22 ± 0.22	0.97 ± 0.60	12.37 ± 4.92	34.58 ± 6.59	19.47 ± 18.03	8.92 ± 14.17	238.70 ± 74.18
	p value	0.186	0.026	0.006	0.031	0.061	0.894	0.613	0.526	0.430	0.177	0.970
Clinical stages	Stage □	4.38 ± 0.92	1.59 ± 1.44	1.04 ± 0.25	2.55 ± 0.79	1.23 ± 0.21	0.89 ± 0.24	12.23 ± 1.37	36.06 ± 6.73	21.20 ± 21.10	9.13 ± 10.71	264.14 ± 72.36
	Stage □	4.55 ± 0.94	1.96 ± 1.66	1.08 ± 0.30	2.69 ± 0.68	1.23 ± 0.24	0.92 ± 0.23	11.98 ± 1.24	33.22 ± 5.57	17.12 ± 16.91	13.24 ± 26.42	234.33 ± 79.93
	Stage □	4.53 ± 0.91	1.67 ± 0.99	1.08 ± 0.25	2.66 ± 0.84	1.26 ± 0.25	1.10 ± 0.89	11.73 ± 0.94	34.43 ± 7.28	20.48 ± 18.55	9.50 ± 12.64	235.53 ± 74.61
	p value	0.593	0.280	0.675	0.612	0.707	0.083	0.103	0.056	0.409	0.599	0.681

Variables		Mean ± SD										
		TC	TG	HDL	LDL	ApoA1	ApoB	PT	APTT	ESR	CRP	PLT
Gender	Male	4.40 ± 0.85	1.82 ± 1.58	1.00 ± 0.24	2.64 ± 0.72	1.16 ± 0.20	0.95 ± 0.36	12.63 ± 5.41	35.32 ± 7.01	14.98 ± 20.37	11.12 ± 22.93	230.26 ± 73.88
	female	4.64 ± 1.00	1.84 ± 1.25	1.16 ± 0.28	2.65 ± 0.82	1.34 ± 0.25	0.99 ± 0.69	11.78 ± 1.22	33.06 ± 5.56	24.46 ± 20.37	10.71 ± 13.79	250.60 ± 72.91
	p value	0.067	0.942	0.00001	0.915	0.000	0.573	0.163	0.015	0.001	0.914	0.054

Figures



***p*=0.709**

Figure 1

Figure 1