Significant correlations were revealed between the occurrence of osteonecrosis and age, gender, HR = 0.974), female gender (female/male, HR = 2.970), fracture displacement (displaced/non-displaced, HR = 1.998) and the season of surgery (fall/winter, HR = 0.372; spring/summer, HR = 0.602, summer/winter, HR = 0.455). CONCLUSIONS: Wintertime osteosynthesis increases the risk of osteonecrosis. The findings raise the possibility of a seasonal variation between seasons in D levels and impaired fracture healing of femoral neck fracture. The results may help establish an effective strategy for the prevention of serious complications.

PMS24 PREDICTORS OF 10-YEAR MORTALITY AFTER PRIMARY FEMORAL NECK FRACTURE IN ELDERLY PATIENTS
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OBJECTIVES: Hip fractures are followed by increased mortality in the elderly. The study was carried out to analyse the mortality rate and predictors for mortality over 10 year period in patients over 60 years old suffered from primary femoral neck fracture who were aged 60 years and over undergoing primary surgical treatment after femoral neck fractures in the year 2000 were collected from database of Hungarian National Health Insurance Fund. The mortality was analysed for the period 2000-2010. The following risk factors were investigated: age, gender, type of fracture, comorbidities, type of surgery, time to surgery and further surgical treatment. Predictors for mortality were evaluated by Cox proportional hazard model and logistic regression analysis using yearly intervals. The patients surviving 12 months after surgery were evaluated. Excessive fluoride causes low bone mass and increased osteonecrosis. The present study was conducted to investigate the possible association between proline-leucine polymorphism and expression of GPx1 mRNA in KBD patients. In the present study, we analysed for the period 2000-2010. The following risk factors were investigated: age, gender, type of fracture, comorbidities, type of surgery, time to surgery and further surgical treatment. Predictors for mortality were evaluated by Cox proportional hazard model and logistic regression analysis using yearly intervals. The patients surviving 12 months after surgery were evaluated. Excessive fluoride causes low bone mass and increased osteonecrosis. The present study was conducted to investigate the possible association between proline-leucine polymorphism and expression of GPx1 mRNA in KBD patients.

RESULTS: 3783 patients were included in the study. The mortality rate was 30.76% in the first year, and 80.65% at 10 years. The mortality showed a tendency to decrease during the following years. Cox regression identified higher age (HR = 1.04), male gender (female/male, HR = 0.776), fracture type (extracapsular/Garden-II, HR = 1.276, and Garden-II/IV/Garden-II, HR = 1.194), comorbidities (presence/absence, HR = 1.961), type of surgery and fracture and surgical delay of the primary treatment up to 2 years. Kaplan-Meier method gave parallel results with risk factor analysis. CONCLUSIONS: Assessing the impact of risk factors on 10 year mortality after primary femoral neck fracture is difficult. We investigated the possible association between the proline-leucine polymorphism (198Pro/Leu) and expression of GPx1 mRNA in KBD patients. In the present study, we studied the association between the proline-leucine polymorphism and expression of GPx1 mRNA in KBD patients. The GPX1 Pro198Leu genotype was determined in 161 patients with KBD in which the M genotype on the risk of KBD. Carriers of Pro/Leu and Leu/Leu had an increased risk (P=0.026). The E2 level decreased significantly compared with that in the control group (P=0.05). The mean for Se concentration in hair was 1.67±0.65 µg/g in “High Se group” and 0.80±0.47 µg/g in “Control group” (t=2.71, P=0.01). The study suggested that the 198Pro/Leu polymorphism is an important risk factor for Beck disease (KBD) in a Chinese population. Meanwhile, we detected the mRNA expression of GPx1 in blood and cartilage tissues between KBD and controls in order to analyze the transcriptional activity of GPx1 and explore molecular mechanisms of Beck disease. The GPX1 Pro198Leu genotype was determined in 161 patients with Beck disease (KBD) in a Chinese population. Meanwhile, we detected the mRNA expression of GPx1 in blood and cartilage tissues between KBD and controls in order to analyze the transcriptional activity of GPx1 and explore molecular mechanisms of Beck disease (KBD).

CONCLUSIONS: Se and E2 levels were studied in KBD patients with moderate to severe RA. The selenium level, while the Hanbin district of Ankang City has lower environmental fluorosis which prevailing in more than 50 countries and regions. A large number of studies have shown that excess fluoride can cause human reproductive system dysfunction. Therefore, we investigated the possible association between the proline-leucine polymorphism (198Pro/Leu) and expression of GPx1 mRNA in KBD patients. In the present study, we studied the association between the proline-leucine polymorphism and expression of GPx1 mRNA in KBD patients. The GPX1 Pro198Leu genotype was determined in 161 patients with KBD in which the M genotype on the risk of KBD. Carriers of Pro/Leu and Leu/Leu had an increased risk (P=0.026). The E2 level decreased significantly compared with that in the control group (P=0.05). The mean for Se concentration in hair was 1.67±0.65 µg/g in “High Se group” and 0.80±0.47 µg/g in “Control group” (t=2.71, P=0.01). The study suggested that the 198Pro/Leu polymorphism is an important risk factor for Beck disease (KBD) in a Chinese population. Meanwhile, we detected the mRNA expression of GPx1 in blood and cartilage tissues between KBD and controls in order to analyze the transcriptional activity of GPx1 and explore molecular mechanisms of Beck disease. The GPX1 Pro198Leu genotype was determined in 161 patients with Beck disease (KBD) in a Chinese population. Meanwhile, we detected the mRNA expression of GPx1 in blood and cartilage tissues between KBD and controls in order to analyze the transcriptional activity of GPx1 and explore molecular mechanisms of Beck disease (KBD).