ALTERED SERUM ENZYMES AND BIOCHEMICAL LEVELS IN ARABIAN RACING CAMELS WITH BONE FRACTURES

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ABSTRACT

Analysis of blood serum enzymes is an important and routine diagnostic tool commonly used to assess the well-being and metabolic status of racing camels. Unfortunately there are no well defined gender-specific published reference ranges for serum enzymes for healthy, lame and fractured camels. In this study, we report baseline reference range values for Glucose, Alkaline phosphatase (ALP), Aspartate aminotransferase (AST), Lactate Dehydrogenase (LDH), Creatine Kinase (CK), Gamma-glutamyltransferase (GGT), Albumin (ALB), Creatinine, Blood Urea Nitrogen (BUN), Intact Parathyroid Hormone (N-PTH), Osteocalcin (OC), Undercarboxylated Osteocalcin (UCOC) and Total Iron binding capacity (TIBC) in healthy (n=60), lame (n=31) and bone-fractured (n=20) racing camels. In addition to documenting gender-specific reference ranges for various biochemical parameters, we report for the first time significant differences in these parameters between healthy, lame and bone-fractured camels. Our results show that of the 15 parameters analyzed, 13 of the hematological parameters in females and 11 parameters in male camels showed significant differences between at least one of the groups – healthy vs. lame, lame vs. fractured, or healthy vs. fractured. In conclusion, the present study clearly shows that some serum enzymes and biochemical levels are altered in racing camels with bone impairment and fractures.

Key words:

INTRODUCTION

Racing camels are culturally and economically important animals, especially in the Middle East (Charles 1982). Unfortunately, until recently, not much is known and published about the various factors that can affect bone health in these animals. We had previously carried out a detailed hematological analysis of healthy, lame and bone-fractured camels and published statistical analysis (gender-based) of differences in 13 parameters between these three racing camel groups (Alshamisi, Ksiksi, and Ashraf 2013). Our results showed that a statistical model could be derived from the routine hematological analysis of camel blood which could help in predicting ‘at-risk’ camels, before they develop bone fractures. Another routine laboratory analysis that can be carried out on blood samples are serum enzyme levels as well as various other biochemicals. There are several groups that have reported reference values for diagnostic enzymes in camel blood, but they are mostly more than 25 years old studies, they are not very detailed and most importantly, they are only carried out on healthy camels. In this report, we present data from 15 different enzyme and biochemicals from serum of a large number of healthy (n=60), lame (n=31) and bone-fractured (n=20) racing camels. The parameters that we report here and their significance (in brief) are described below:

Glucose: Glucose is a measured parameter that is used to evaluate carbohydrate metabolism (Washington and Hoosier 2012). It is also known to affect bone metabolism in both mice and humans (Harris et al. 2003). There is extensive evidence which links diabetes with low bone formation in humans (Hofbauer et al. 2007; Leidig-Bruckner, Ziegler, and others 2001; Bouillon et al. 1995; Krakauer et al. 1995) and animals (Shying, Devlin, and Sloan 2001; Verhaeghe et al. 1990) alike.

Alkaline phosphatase (ALP): Blood serum alkaline phosphatase (ALP) is a hydrolytic enzyme which removes phosphate from different molecules. It is mainly secreted by osteoblasts which play a vital role in the metastatic process of bone destruction (Roodman 2004). Alkaline phosphatase is considered a marker for osteoblastic activity (Dimai et al. 1998). It has been shown that ALP serum levels increase in bone metabolism (Alataş et al. 2002).

Aspartate aminotransferase (AST): Aspartate aminotransferase (AST), catalyzes the reversible transfer of an α-amino group between aspartate and glutamate (Washington and Hoosier 2012). It is a vital enzyme in amino acid metabolism. AST is commonly measured clinically as a marker for liver disease. Both the gender and age of racing horses are known to significantly affect creatine kinase (CK) and AST activities (Harris et al. 1990). Moreover, a high AST/ALT ratio and a low PLT
count can predict liver cirrhosis in chronic HCV infection (Pohl et al. 2001).

**Lactate Dehydrogenase (LDH):** Lactate dehydrogenase (LDH) is an enzyme known to catalyze the conversion of pyruvate to lactate, with the conversion of NADH to NAD⁺ (Washington and Hoosier 2012). A high LDH level is an indicator of organ infarction and significant cell death, which includes cellular cytoplasmic loss. There are several disease conditions which are linked with elevated levels of LDH such as hepatitis, shock, hypoxia, extreme hypothermia, and meningitis (Washington and Hoosier 2012).

**Creatine Kinase (CK):** Creatine kinase (CK) is an enzyme known to catalyze the phosphorylation of creatine (Washington and Hoosier 2012). Creatine kinase levels increase subsequent to exercise in humans (Noakes and others 1987) and animals (Shumate et al. 1979). Creatine kinase is also recommended for use as an indicator for lameness via carpal osteochondral fragmentation in horses (Chaney et al. 2004).

**Gamma-glutamyltransferase (GGT):** Gamma (γ)-glutamyltransferase (GGT) is an enzyme known to contribute to the extracellular catabolism of glutathione (GSH) (Grundy 2007) and is an important tool for the diagnosis of liver disorders. The measurement of urine GGT levels may be regarded as a useful marker for individuals who may have increased bone turnover, which increases the risk for bone fractures (Asaba et al. 2006).

**Albumin (ALB):** Albumin (ALB) is a protein with the highest concentration levels found in blood plasma (Soni and Margarson 2004). Albumin is formed exclusively in the liver and serves as a transport and binding protein for calcium, fatty acids, bilirubin, hormones, trace elements, and drugs (Soni and Margarson 2004). Decreased serum albumin concentrations can be a consequence of liver disease. Decreased serum albumin may also be a result of malnutrition or of a low protein diet.

**Creatinine:** Creatinine is a product of the break-down of creatine phosphate in muscle (Washington and Hoosier 2012). It is used as an indicator of kidney function. Serum creatinine is increased by deficient glomerular filtration. It also increases by necrosis or atrophy of the skeletal muscle, hyperthyroidism, infections, burns, or fractures.

**Blood Urea Nitrogen (BUN):** Urea is a break-down product of protein metabolism (Washington and Hoosier 2012). Concentrations of blood urea nitrogen are used to test for kidney function. The concentration level of BUN can be affected by circadian rhythm, diet, liver function, hydration, and intestinal absorption (Blood Urea Nitrogen (BUN) 2012). A low concentration of blood urea nitrogen (BUN) may be due to low protein intake, liver failure, or treatment with anabolic steroids.

**Calcitonin:** Calcitonin is a hormone known to control calcium metabolism by inhibiting bone resorption and thus has been shown to increase bone quality in various animal models used within the studies (Wallach et al. 1999). In human studies, calcitonin is shown to increase lumbar vertebral cortical thickness (Mosekilde, Danielsen, and Gasser 1994). Postmenopausal women have been shown to have lower calcitonin levels in comparison to premenopausal females (Reginster et al. 1990).

**Intact Parathyroid Hormone (N-PTH):** Parathyroid Hormone is one of the most important hormones in both calcium and phosphate metabolism. Many studies have shown that the parathyroid hormone improves bone growth (Pettway et al. 2008; Dempster et al. 1993). One of the ways in which it stimulates an increased blood calcium concentration is by stimulating bone resorption. An alternative way is by reducing renal calcium excretion and increasing renal phosphate excretion. It also promotes bioactive vitamin D production by kidneys, which in turn, enhances calcium and phosphate absorption by the small intestine. Parathyroid hormone has many fragments. Intact PTH is mainly known as the biologically active form of PTH (Murray et al. 2005), while carboxyl-terminal or C-terminal PTH is known as the inactive form of PTH. The inactive C-terminal PTH is, however, present in the circulation in large amounts. PTH (1–34), which is one of the intact forms of PTH, is now approved by the Food and Drug Administration (FDA) as a primary anabolic therapy used in the treatment of severe osteoporosis in both women and men (Tashjian Jr and Gagel 2006). A multitude of studies have demonstrated that the “Intact PTH” therapy does in fact improve bone formation in humans (Noakes and others 1987; Shumate et al. 1979; Chaney et al. 2004; Grundy 2007; Whitfield 2001; Asaba et al. 2006; Dempster et al. 1993; Thomas 2006; Neer et al. 2001; Jiang et al. 2003; Ejersted et al. 1995; Orwell et al. 2003) and animals (Dobnig and Turner 1997). The C-terminal to intact PTH ratio is important in Ca²⁺ ion metabolism (Nguyen-Yamamoto et al. 2001).

**Osteocalcin (OC) and Undercarboxylated Osteocalcin (UCOC):** Osteocalcin (OC) is a calcium-binding protein which is known to be an important protein in bone mineralization (Ivaska et al. 2005). It is a 49 amino acid polypeptide found exclusively in bone tissue that undergoes vitamin-K dependent gamma-carboxylation of three glutamic acid residues (Ng et al. 2004). In case of vitamin-K deficiency, Osteocalcin remains as undercarboxylated Osteocalcin (UCOC), which is not functional in binding calcium. A number of studies have shown that vitamin-K deficiency leads to the impairment in the carboxylation of Osteocalcin, the increase of under
carboxylated Osteocalcin content, and consequently an increase in bone fracture risk (Kim et al., 2010). Furthermore, undercarboxylated Osteocalcin (UCOC) is negatively correlated with femoral neck bone mineral density and bone fractures in the elderly (Fewtrell et al. 2008). The expression of the OC gene (and hence OC protein) depends on vitamin D, which is considered a major regulator of calcium metabolism (Vergnaud et al. 1997). To date, only one investigation has reported on osteocalcin in camels (Al-Sobayil 2010).

**Total Iron binding capacity (TIBC):** Iron is an essential dietary element as it is involved in various metabolic activities within the body such as oxygen sensing and transport, electron transfer, and catalysis (Papanikolaou and Pantopoulos 2005). Iron plays a significant role in bone metabolism (Katsumata et al. 2006), as iron deficiency decreases bone formation and resorption (Medeiros et al. 2002; Katsumata et al. 2009). Iron deficiency is also negatively associated with bone mineralization of the osteoblast cells (Paremal et al. 2006). Iron functions as a cofactor for many enzymes and is involved mainly in collagen synthesis (Ilich and Kerstetter 2000). Some studies have demonstrated that an overload of iron ingestion or intake may accumulate in bones, which may result in the development of bone diseases, such as osteoporosis in rats (Isomura et al. 2004). The dietary intake of iron is also associated with bone mineral density in postmenopausal women (Harris et al. 2003; Maurer et al. 2005).

**MATERIALS AND METHODS**

The study was conducted on racing camels, both females and males, ranging in age between one and four years. The camels were selected from different racing camps owned by the Presidential Affairs in Abu Dhabi Emirate, UAE.

Animals were fed on a ration of fresh clover, dry clover, barley, camel milk, pre-mix feeds, multi- vitamins and mineral. All camels had free access to drinking water. Three groups of camels were identified: healthy racing camels, racing camels with lameness, and racing camels with bone fractures. The three groups were referred to as normal, lame and fractured; respectively thereafter. Bone fracture was diagnosed by a veterinary doctor through a field examination and when necessary, an X-ray. Lameness was diagnosed by visual observations, if camels were limping while walking; they were classified as limping camels. Camels in the control groups consisted of apparently healthy animals. All groups were similar in age and gender (95% were between 1-4 years). Camels were split into three gender specific groups with a total of 111 individuals as outlined in Table1.

**Sample collection and storage:** Blood samples were collected in the morning hours, except in cases of fractures, when blood samples were collected immediately after the fracture was diagnosed.

| Table 1. The three groups of racing camels used in this study. |
|---------------------------------|--------|--------|
| Number of animal | Gender |
| Healthy | 35 | Females |
| 25 | Males |
| Lame | 19 | Females |
| 12 | Males |
| Fractured | 14 | Females |
| 6 | Males |

BD Vacutainer Serum Separation Tubes or SST (Becton Dickinson, USA) were used to collect blood samples. Serum was obtained from SST tubes after centrifugation at 3,000 rpm for 10 minutes. Routine clinical chemistry tests were performed on these serum samples the same day, and remaining serum was aliquoted and kept at -80°C.

**Measurements of various parameters:**

**Enzymes and biochemicals:** For determination of clinical biochemistry parameters, the standard laboratory instrument, Dimension RxL Max (Siemens, USA) was used (Mendu et al 1007). It is a discrete, random access, microprocessor-controlled, integrated instrument, which is routinely used to measure a variety of analytes, including enzyme activities in body fluids (Dimension RxL Max Integrated Chemistry System n.d.). All reagents, calibrators and controls were obtained from Siemens. Calibration for each test was performed as per instrument schedule and two level controls were run daily for each test.

The parameters measured using the Dimension RxL system were:

A. Proteins: albumin, total protein (TP)
B. Enzymes: alkaline phosphatase (ALP) (EC 3.1.3.1), aspartate aminotransferase (AST) (EC 2.6.1.1), creatine kinase (CK)(EC 2.7.3.2), Gamma-glutamyltransferase (GGT)(EC. 2.3.2.9), lactate dehydrogenase (LDH) (EC. 1.1.1.27)
C. Other tests: blood urea nitrogen (BUN), creatinine, and glucose.

**Calcitonin and Intact Pyrethroid Hormone (N-PTH) Measurement:** Calcitonin and intact PTH were analyzed by Liaison instrument (DiaSorin, Germany). The Liaison instrument is a fully automated system which is commercially available to determine various bone markers. All reagents (magnetic particles, luminescence-labeled tracer, calibrators, diluents, and assay buffer)
were obtained from the manufacturer and used as per the supplied instruction. Data reduction was based on a master curve with a two-point recalibration method.

**Measurement of Carboxylated (OC) and Undercarboxylated Osteocalcin (UCOC):** Carboxylated (OC) and Undercarboxylated (UCOC) Osteocalcin were analyzed using enzyme-linked immunoassay kit (EIA) (Takara, Japan). The OC and UCOC Kits are a solid phase EIA based on a sandwich method that utilizes two mouse monoclonal antibodies to detect OC or UCOC two-step procedure. One of the mouse monoclonal antibody is immobilized onto the micro titer plate and blocked against non-specific binding. Samples and standards are incubated in the micro titer plate. After one hour incubation, the plate is washed and second anti-OC or anti-UCOC labeled with peroxidase (POD). The bound POD-linked second OC or UCOC antibody is detected by the reaction between POD and substrate (H$_2$O$_2$ and tetramethylbenzidine) which results in color development with intensity proportional to the amount of OC or UCOC in the samples and standards. The amount of OC or UCOC can be quantitated by measuring the absorbance using an EIA plate reader. Accurate sample concentrations of osteocalcin can be determined by comparing their specific absorbance values with those obtained for the standards plotted on a standard curve.

The software package SPSS (v. 19) was used for all statistical analyses reported here.

**Controls used in the analyses:** In Dimension RxL Max instrument; two levels of controls were used daily - DADE Dimension RxL Max instrument chemistry controls (TRU-Liquid moni-trol level 1B5150-1 and TRU-Liquid moni-trol level 2 B5150-2 – both from Siemens, USA). The absorbance was also checked daily by absorbent test (DF73). A mixture of homogenization of blood samples serum were used for routine internal quality control. Two levels of standard spiking (at 0.25mg/l and 0.5mg/l) were also routinely used. In addition, proficiency testing from Center de Toxicologie du Quebec, Inter laboratory comparison program for metals in biological materials, PC-S-E was also carried out. Calcitonin II-Gen chemiluminescent immunoassay was used as a means of checking reliability of assay runs of Calcitonin (DiaSorin, 310651). For carboxylated (OC) and undercarboxylated Osteocalcin (UCOC) determination, immunoassay kits were used and samples were analyzed in duplicates.

**RESULTS AND DISCUSSION**

In spite of the importance and prevalence of racing camels in the Middle East, detailed biochemistry parameters are not widely published for these animals. In fact, only a handful of published reports can be found, and even they are fairly dated, not detailed and not performed on bone-health compromised camels. Therefore, we carried out a systematic analysis of various biochemical parameters in the serum of a relatively large numbers of healthy, lame and fractured racing camels (both genders). The reference values of various enzymes and biochemistry parameters in the three groups (healthy, lame, and fractured) of female and male racing camels shown in Tables 2 and 3. A comparison of the various parameters of the healthy camels show that for the most part, our reference values agree with those previously published by other groups. For example, the glucose and GGT values in our healthy racing camel groups were very similar to the values in Kuwaiti racing camels (Mohamed and Hussein 1999) and Sudan camels (El-dirdiri, Suliman, and Shommein 1987). Similarly, the results of BUN and TP match those of the Kuwaiti racing camels and Arabian camels in Iran (Mohamed and Hussein 1999; Alsaad 2009). AST values in our camels were also similar to Arabian camels in Iran (Alsaad 2009). However, breeding camels in Algeria (Aichouni et al. 2010), and Kuwaiti racing camels had less AST (Mohamed and Hussein 1999). AST values in our normal female and male camels were 99.9±21.4 IU/I and 100.7±16.2 IU/I; respectively, whereas, AST level were 74.5±15 IU/I and 69.8±23.11 IU/I in breeding camels in Algeria, and Kuwaiti racing camels; respectively. The LDH values in females and males were similar to the values which is published in Sudan (El-dirdiri, Suliman, and Shommein 1987). On the other hand, the mean value of creatinine in our healthy racing females and males were 139.3±28.9 µmol/l and 124.8±16.7 µmol/l; respectively, which is higher than the mean value of Kuwaiti racing camels and lower than the average values of breeding camels in Algeria. Kuwaiti racing camels had 174.15 ±29.17 µmol/l of creatinine. While breeding camels in Algeria had 96.05±2.51 µmol/l of creatinine. The mean value of ALP and CK was also higher in Kuwaiti racing camels than our mean values in healthy racing camels. Kuwaiti racing camels had mean value of 95.12±36.8 IU/I and 143.634±79.63 IU/I for ALP and CK; respectively. Our mean value of ALP and CK in normal female racing camels group was 190.9±89.2 IU/I and 105.1±113.1 IU/I; respectively. While in males, ALP and CK mean vales were 193.3±29.2 IU/I and 84.2±96.2 IU/I; respectively. On the other hand, the average CK values were lower in breeding camels (96.05±2.51 IU/I) than our results. Some studies show a seasonal variation in chemical parameters (Salman and Afzal 2004; Kataria and Bhatia 1991). TP, albumin, CK and creatinine values are stable during summer and winter(Salman and Afzal 2004). However, BUN and iron are higher in winter. Some papers conclude that LDH activity is higher in summer (Salman and Afzal 2004) while other papers reported that LDH activity is higher in winter (Kataria and Bhatia 1991). The activity of AST, and ALP in
higher during hot conditions (Kataria and Bhatia 1991). The activity of AST and ALP in males is higher than females (Kataria and Bhatia 1991). In this study we have very similar results for AST and ALP in females and males racing camels.

Statistical (t-test) analyses of significant differences in these biochemistry parameters (enzymes and biochemistry) showed that there were significant in 13 out of 15 parameters between at least one of the groups - healthy vs. lame, lame vs. fractured, or healthy vs. fractured in female racing camels (Table 2). While in males, 10 out of 15 parameters were significantly different between at least one of the three groups (Table 3). This is a very interesting and novel finding, as such a comparative study has not been previously carried out and clearly shows that even camels that have not yet developed bone fractures (i.e. lame camels) have significant biological and metabolic differences as compared to healthy animals. And of course, some of these differences in enzyme levels and other biochemicals are even more pronounced in the bone-fractured racing camels. At this point, we are unable to assess the “cause and effect” relationship, i.e. whether these observed differences in biochemical parameters in the three groups are due to compromised bone health (lameness or fracture) or if the poor bone conditions in these animals cause the observed biochemical disturbances. Nevertheless, this preliminary study clearly shows that there are several factors that need to be carefully studied in the future in the hope of possibly developing diagnostic tools to identify racing camels at risk of developing bone fractures.

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Table 2. Mean, standard deviations and statistical analyses in various biochemistry parameters in the three female camel groups.

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Normal</th>
<th>Lame</th>
<th>Fractured</th>
<th>N. vs. L</th>
<th>N. vs. F</th>
<th>L. vs. F</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glucose (mmol/l)</td>
<td>6.2±1.6</td>
<td>6.0±0.9</td>
<td>10.6±4.1</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>BUN (mmol/l)</td>
<td>5.8±1.4</td>
<td>5.8±2.4</td>
<td>6.8±2.1</td>
<td>No</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Creatinine (µmol/l)</td>
<td>139.3±28.9</td>
<td>126.5±18.2</td>
<td>150.0±37.6</td>
<td>No</td>
<td>Nu</td>
<td>Yes</td>
</tr>
<tr>
<td>TP (g/l)</td>
<td>6.5±1.5</td>
<td>6.4±1.4</td>
<td>6.7±1.1</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Albumin (g/l)</td>
<td>29.2±12.2</td>
<td>29.0±12.4</td>
<td>31.1±13.3</td>
<td>No</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>AST (IU/l)</td>
<td>17.2±2.9</td>
<td>20.1±3.2</td>
<td>17.3±3.3</td>
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<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>ALT (IU/l)</td>
<td>199.9±88.2</td>
<td>138.0±66.4</td>
<td>111.5±51.9</td>
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<td>Yes</td>
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<td>CK (IU/l)</td>
<td>105.1±131.1</td>
<td>55.1±123.4</td>
<td>1513.9±1307.6</td>
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<td>Yes</td>
<td>Yes</td>
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<td>LDH (IU/l)</td>
<td>452.1±52.6</td>
<td>424.1±199.5</td>
<td>67.9±199.2</td>
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<td>Yes</td>
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</tr>
<tr>
<td>triglycerides (µg/dl)</td>
<td>213.1±189.4</td>
<td>201.6±71.4</td>
<td>205.6±58.7</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Calcium II (mg/l)</td>
<td>2.4±1.1</td>
<td>3.0±2.7</td>
<td>4.2±3.5</td>
<td>No</td>
<td>No</td>
<td>No</td>
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<tr>
<td>Pi (µmol/l)</td>
<td>12.8±7.9</td>
<td>29.9±23.1</td>
<td>18.2±18.2</td>
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<tr>
<td>Ntcalc (µmol/l)</td>
<td>1.6±1.1</td>
<td>1.9±0.8</td>
<td>0.7±0.5</td>
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<td>Yes</td>
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<tr>
<td>Uric (mg/ml)</td>
<td>4.2±1.2</td>
<td>3.9±1.0</td>
<td>1.4±1.4</td>
<td>No</td>
<td>Yes</td>
<td>No</td>
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</table>
Table 3. Mean, standard deviation sand statistical analyses in various biochemistry parameters in the three female camel groups.

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Mean ± SD</th>
<th>Significance [p&lt;0.05]</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Normal</td>
<td>Lame</td>
</tr>
<tr>
<td>Glucose (nmol/l)</td>
<td>6.1±0.5</td>
<td>6.1±1.0</td>
</tr>
<tr>
<td>BUN (mmol/l)</td>
<td>6.7±1.1</td>
<td>7.3±1.4</td>
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<tr>
<td>Creatinine (μmol/l)</td>
<td>124.8±15.7</td>
<td>127.6±37.5</td>
</tr>
<tr>
<td>TP (g/l)</td>
<td>63.7±3.0</td>
<td>67.3±3.0</td>
</tr>
<tr>
<td>Albumin (g/l)</td>
<td>29.2±1.9</td>
<td>28.8±2.0</td>
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<td>GGT (IU/l)</td>
<td>20.5±3.2</td>
<td>19.5±2.7</td>
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<tr>
<td>ALP (IU/l)</td>
<td>193.3±39.9</td>
<td>137.5±39.9</td>
</tr>
<tr>
<td>AST (IU/l)</td>
<td>100.7±15.2</td>
<td>103.5±25.5</td>
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<tr>
<td>CK (IU/l)</td>
<td>84.2±96.4</td>
<td>55.8±17.5</td>
</tr>
<tr>
<td>LDH (IU/l)</td>
<td>407.0±43.8</td>
<td>358.4±45.9</td>
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<tr>
<td>TIBC (μg/dL)</td>
<td>201.6±18.1</td>
<td>192.5±27.2</td>
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<tr>
<td>Calcitonin II (pg/ml)</td>
<td>1.6±0.5</td>
<td>2.2±1.2</td>
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<tr>
<td>N-Term PTH (pg/ml)</td>
<td>32.1±22.8</td>
<td>30.3±19.2</td>
</tr>
<tr>
<td>OC (ng/ml)</td>
<td>2.1±2.2</td>
<td>1.0±1.0</td>
</tr>
<tr>
<td>UCOC (μg/ml)</td>
<td>4.5±1.7</td>
<td>2.3±0.9</td>
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REFERENCES


