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BIOCHEMICAL EVALUATION OF RISK FACTORS OF MENTAL DEPRESSION

Biochemistry

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ABSTRACT

The aim of our study is to examine the levels of routine biochemical markers in patients with major depressive disorder (MDD). **Materials and METHODS:** We used the Hamilton Depression (HAMD) score to evaluate the severity of depressive symptoms in 200 patients with MDD. There were significant differences between MDD patients and healthy controls in alanine transaminase (ALT), urea nitrogen (UN), lactate dehydrogenase (LDH), uric acid (UA), total protein (TP), total bile acid (TBA), creatinine (Cr), total bilirubin (Tbil), direct bilirubin (Dbil) and indirect bilirubin (Ibil), high density lipoprotein-cholesterol (HDL-C), fasting blood-glucose (FBG) and fructosamine (SF). **RESULT**: Multivariate analysis showed that UN, FBG, HDL-C, SF, TP, Cr and Tbil remained independently association with MDD. **CONCLUSION:** Use of these routine biochemical markers in combination may contribute to improve the complete management for patients with MDD.

KEYWORDS

INTRODUCTION:

Major depressive disorder (**MDD**), also known simply as **depression**, is a mental disorder characterized by at least two weeks of pervasive low mood. Low self-esteem, loss of interest in normally enjoyable activities, low energy, and pain without a clear cause are common symptoms.[1] Those affected may also occasionally have delusions or hallucinations.[1] Some people have periods of depression separated by years, while others nearly always have symptoms present.[3] Major depression is more severe and lasts longer than sadness, which is a normal part of life.[3]

The diagnosis of major depressive disorder is based on the person's reported experiences and a mental status examination.[7] There is no laboratory test for the disorder,[3] but testing may be done to rule out physical conditions that can cause similar symptoms.[7] Those with major depressive disorder are typically treated with counseling and antidepressant medication.[1] Medication appears to be effective, but the effect may only be significant in the most severely depressed.[8][9] Types of counseling used include cognitive behavioral therapy (CBT) and interpersonal therapy,[1][10] and electroconvulsive therapy (ECT) may be considered if other measures are not effective.[1] Hospitalization may be necessary in cases with a risk of harm to self and may occasionally occur against a person's wishes.[11]

The most common time of onset is in a person's 20s and 30s,[3][4] with females affected about twice as often as males.[3][4] Major depressive disorder affected approximately 163 million people (2% of the world's population) in 2017.[6] The percentage of people who are affected at one point in their life varies from 7% in Japan to 21% in France.[4] Lifetime rates are higher in the developed world (15%) compared to the developing world (11%).[4] The disorder causes the second-most years lived with disability, after lower back pain.[11]

The term *major depressive disorder* was introduced by a group of US clinicians in the mid-1970s.[11] The cause of major depressive disorder is believed to be a combination of genetic, environmental, and psychological factors,[1] with about 40% of the risk related to genetics.[5] Risk factors include a family history of the condition, major life changes, certain medications, chronic health problems, and substance use disorders.[1][3] It can negatively affect a person's personal life, work life, or education as well as sleeping, eating habits, and general health.[1][3] Those currently or previously affected with the disorder may be stigmatized.[11]

MATERIALS AND METHODS:

Laboratory and demographic data were analyzed in 200 patients with MDD, wherein included 50 male and 150 female. The diagnosis of MDD was determined by DSM-IV criteria[11,4].

EXCLUSION CRITERIA:

All patients accompanied by cardiovascular disease, hypertension, endocrine disease, liver and kidney disorder, gout, infectious disease, metabolic syndrome, autoimmune disease, malignancy, pregnancy and head trauma history were not included.

INCLUSION CRITERIA:

and any patients with anxiety disorders, neurodegenerative disorders, bipolar or psychotic disorders, mental retardation, psychiatric medications use and substance abuse were also excluded.

We used the Hamilton Depression (HAMD) score to evaluate the severity of depressive symptoms15, higher scores on the HAMD expressed more severe symptomatology.. A total of 201 healthy subjects with healthy diet at least one month were selected as controls, and all healthy individuals were no history of head trauma history, psychiatric disorder, neurological disorder in this study. Demographic and clinical data were obtained from diagnostic records. The body mass index was calculated as an individual's weight in kilograms divided by the square of height in meters. Fasting venous blood from participants were collected to measure biochemical parameters, including alkaline phosphatase (ALP), total cholesterol (TC), alanine transaminase (ALT), aspertate aminotransferase (AST), UN, low density lipoprotein-cholesterol (LDL-C), high density lipoprotein-cholesterol (HDL-C), γ-glutamyl transpeptidase (γ-GGT), glucose, lactate dehydrogenase (LDH), uric acid (UA), SF, direct bilirubin (Dbil), total bile acid (TBA), total bilirubin (Tbil), total protein (TP), triglyceride (TG), indirect bilirubin (Ibil) and Cr. All biochemical tests were performed by automatic biochemical analyzer.

Statistical analysis.

Statistical analyses were performed with the statistical package SPSS version 16.0. All continuous variables were expressed as mean (±standard deviation), and categorical variables were expressed as percentage.

RESULTS

The mean age of patients with MDD was 39.6 years and 150 (75%) were females. Regarding the cumulative clinical profile, the phase of depression severity was between moderate (70.9%) and severe (33.1%) in accordance with HAMD total score (21.7±4.12), almost patients suffered from somatic and psychiatric comorbidities, such as pain (57.7%), poor appetite (75.2%), fatigue (37.7%), palpitations (57.0%), insomnia (77.4%), hallucination (47.1%), delusion (49.3%), guilt (38.8%) and psychomotor retardation (36.6%). There were no statistically significant differences in gender, age and body mass index between MDD patients and controls, as shown in Table 1. Cumulative results for biochemical tests were obtained from patients with MDD and controls. The laboratory characteristics were outlined in Table 1.

Table 1. Biochemical parameters between MDD patients and controls in clinical laboratory

	MDD patients	Controls		
High density lipoprotein-cholesterol (mmol/L)	1.41±0.33	1.34±0.31	1.24±0.300	0.008
Low density lipoprotein-cholesterol (mmol/L)	2.43±0.58	2.43±0.69	2.53±0.68	0.957
Total cholesterol (mmol/L)	4.17±0.71	3.08±0.82	4.08±0.81	0.320
Triglyceride (mmol/L)	2.00±0.36	0.98±0.34	0.97±0.33	0.383
Alkaline phosphatase (U/L)	67.34±18.68	68.78±11.27	66.77±12.26	0.140
		22.18±14.31		
Alanine transaminase (U/L)	19.14±12.58		21.17±13.30	0.008
Aspertate aminotransferase (U/L)	18.50±7.39	19.29±7.06	18.28±6.05	0.122
γ-glutamyl transpeptidase(U/L)	20.89±20.95	22.45±17.38	21.44±16.37	0.348
		5.62±0.44		
Fasting blood-glucose (mmol/L)	5.83±0.45		4.52±0.43	<0.001
Fructosamine (mmol/L)	3.29±0.28	3.18±0.28	2.19±0.29	<0.001
Urea nitrogen (mmol/L)	5.34±1.37	5.86±1.24	4.85±1.23	<0.001
Lactate dehydrogenase (U/L)	148.95±26.68	152.29±23.75	152.29±23.75	0.046
Uric acid (μmol/L)	258.72±68.74	288.23±78.18	285.22±77.17	<0.001
Total protein (g/L)	68.82±6.10	69.82±6.23	68.72±5.23	<0.001
Creatinine (μmol/L)	58.72±13.92	67.18±15.08	65.19±14.09	<0.001
	9.32±4.18	11.08±5.60		
Total bilirubin (μmol/L)			10.07±4.50	<0.001
Direct bilirubin (μmol/L)	4.51±2.10	4.82±1.63	3.72±1.53	0.016
Indirect bilirubin (μmol/L)	5.91±3.57	7.37±4.83	6.36±3.73	<0.001
Total bile acid (μmol/L)	4.21±3.67	5.59±5.01	4.49±4.01	<0.001

DISCUSSION

Clinical biochemical tests are routine hospital examinations in clinical practice. This investigation found that UN, FBG, HDL-C, SF, TP, Cr and Tbil were independently associated with MDD in logistic regression analysis. Further, our study revealed that use of these markers in combination could provide useful and objective information in the assessment for patients with MDD. Oxidative stress derives from increased production of reactive oxygen species, which leads to cell damage by biological reactions such as lipid peroxidation, enzyme inactivation and DNA modification[11]. Previous studies have demonstrated that oxidative stress related enzymes are associated with the pathogenic process of patients with depression, and anti-oxidative enzymes activity is increased in patients suffering from depressive disorder[8,9]. Emerging evidence has suggested that several inflammatory cytokines such as interleukin-1 (IL-1) beta, IL-6 and TNF are implicated with oxidative stress in patients with MDD20. Interesting, oxidative stress process has also been observed in the frontal cortex of patients with recurrent depressive disorder. Moreover, the xanthine oxidase activity is increased in the thalamus and the putamen of patients with depression, which tends to induce oxidative stress by an increased production of reactive oxygen species. These reports provide an important fact that oxidative stress may be a major contributor in the pathogenesis of MDD. There is no literature available with respect to serum bilirubin levels in patients with MDD, serum bilirubin levels in patients MDD were found to be decreased, and lower Tbil concentrations were associated with MDD in the present study. Bilirubin, the end product of heme catabolism, is an efficient and powerful antioxidant, the role of bilirubin in regarding with oxidation resistance, anti-inflammation and immunosuppression has been demonstrated in various diseases[4]. Indeed, lower serum levels of bilirubin have been reported in patients with migraine, carbonmonoxide-poisoned and pulmonary embolism, and serum bilirubin may provide a protective action in patients with cardiovascular disease and rheumatic disease[9,3]. In these studies, bilirubin as an endogenous antioxidant may be destroyed by excessive oxidative stress. Thus, the presence of oxidative stress may result in overconsumption of serum bilirubin, which is associated with lower bilirubin in patients with MDD. Higher levels of HDL-C have been reported in patients with depression[11]. In agreement with this finding, increased levels of HDL-C were demonstrated to be associated with MDD patients in our study. In addition, we observed lower concentrations of UN and TP in patients compared to healthy controls, the findings are consistent with recently reports on MDD patients[2]. Serum Cr concentrations were decreased in MDD patients compared with controls, the results may attribute to poor appetite and nutrition in patients with MDD, because accompanied anorexia has a high prevalent in patients with MDD[3,4]. Of note, increased levels of FBG and SF were found to be associated with MDD in our study. Oxidative stress is a crucial player in the establishment of insulin resistance and diabetes mellitus[5]. In fact, oxidative stress has been regarded as an underlying mediator of insulin resistance, and there is an inversely relation between oxidative stress and insulin action[6,7]. Studies have shown that oxidative stress can decrease insulin sensitivity by GLUT-4 deficiency. Furthermore, major insulin secretion has been found to be dependent on the regulation of intracellular and extracellular reactive oxygen species to a certain extent[9]. It has recently been shown that oxidative stress may increase induction of HO-1 expression, leading to insulin resistance and insufficient insulin[5].

CONCLUSION

In the current study, these biochemical parameters are objective and available, the composite information from these routine biochemical markers may improve the diagnostic effectiveness of depressive disorder. However, our results are needed to be further established with multicenter and larger-scale study.

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