



P-ISSN: 2789-1623  
E-ISSN: 2789-1631  
IJRP 2021; 1(1): 21-23  
Received: 22-12-2020  
Accepted: 13-02-2021

Afreenbanu A Khadirnavar  
Department of Psychiatry and  
Psychotherapy, Semmelweis  
University Medical School,  
1083 Budapest, Hungary

## Assessment of serum S100B level in patient with depression: A clinical study

Afreenbanu A Khadirnavar

### Abstract

**Background:** Depression is a common psychiatric condition that negatively affects feeling, thinking and acting. The present study was conducted to assess serum S100B level in patient with depression.

**Materials and Methods:** 86 patients of depression of both genders were enrolled. Group I comprised of depression patients and group II had control. The severity of depression was assessed using Beck's depression inventory-II (BDI), Hamilton depression rating scale (HAM- D) and stressful life event scale (SLES). Serum S100B levels were measured using Sandwich ELISA.

**Results:** The mean BDI scale was 28.4 in group I, 2.1 in group II. HAM- D was 20.4 in group I and 1.6 in group II and SLES was 5.7 in group I and 2.4 in group II. The difference was significant ( $P < 0.05$ ). The mean S100B level in group I males was 98.1 pg/ml and in females was 93.4 pg/ml and in group II males was 67.1 pg/ml and in females was 26.9 pg/ml. The difference was significant ( $P < 0.05$ ).

**Conclusion:** Serum S100B level was elevated in depression patients as compared to control subjects.

**Keywords:** Beck's depression inventory-II, depression, stressful life event scale

### Introduction

Depression is a common psychiatric condition that negatively affects feeling, thinking and acting. Depression causes feelings of sadness and/or a loss of interest in activities once enjoyed. Depression can lead to a multiple emotional and physical problems and can decrease a person's ability to function at work and at home. The young people in the age group of 10-24 are characterized by immense growth and development<sup>[1]</sup>.

Adoption and family studies have established that depression runs in families and that most of this familiarity occurs as a result of genetic rather than environmental influences<sup>[2]</sup>. Unipolar depression, as a heterogeneous disorder, is likely to include subgroups that represent more genetic forms of depressive illness. Recurrent, early onset depression, defined as two or more episodes before the age of 25, is associated with a strong family history of affective disorder and appears to follow a particularly malignant course, with frequent recurrence, poor response to treatment and high psychiatric and physical comorbidity<sup>[3]</sup>.

Depression is a complex, heterogeneous disorder. Many pathological mechanisms have been described for depression. Decrease in the levels of the neurotransmitter serotonin in the brain is the basis of serotonin hypothesis of depression<sup>[4]</sup>. Neurotrophin hypothesis involves the disruption of neuroplasticity that is a fundamental mechanism of neuronal growth and adaptation, which involves the neurotrophic factors having multiple functions at different stages of development and at different locations in and around the nervous system. Furthermore, depression is considered to be low-grade chronic neuroinflammation. There is the interaction of cytokines that are the mediators of inflammation with the brain through the blood-brain barrier<sup>[5]</sup>.

The role of S100B in neuroplastic mechanisms in neuronal pathways has also been studied in conditions other than depression such as Alzheimer's disease, schizophrenia and mania. This study was undertaken to look into the serum levels of S100B in participants of age 13-25 years with major depression as compared to healthy controls of comparable age and gender and also to analyze the correlation of the levels with clinical severity of depression assessed by psychometric evaluation scores<sup>[6]</sup>. The present study was conducted to assess serum S100B level in patient with depression.

### Correspondence

Afreenbanu A Khadirnavar  
Department of Psychiatry and  
Psychotherapy, Semmelweis  
University Medical School,  
1083 Budapest, Hungary

## Materials and Methods

The present study comprised of 86 patients of depression of both genders. All were informed regarding the study and their written consent was obtained. Equal number of age and gender matched control were also enrolled.

Demographic profile such as name, age, gender etc. was recorded. Group I comprised of depression patients and group II had control. The severity of depression was assessed using Beck's depression inventory-II (BDI), Hamilton depression rating scale (HAM-D) and stressful life event scale (SLES). Medical assessment and laboratory investigations were done. Serum S100B levels were measured using Sandwich ELISA. Results thus obtained were subjected to statistical analysis. P value less than 0.05 was considered significant.

## Results

**Table 1:** Distribution of patients

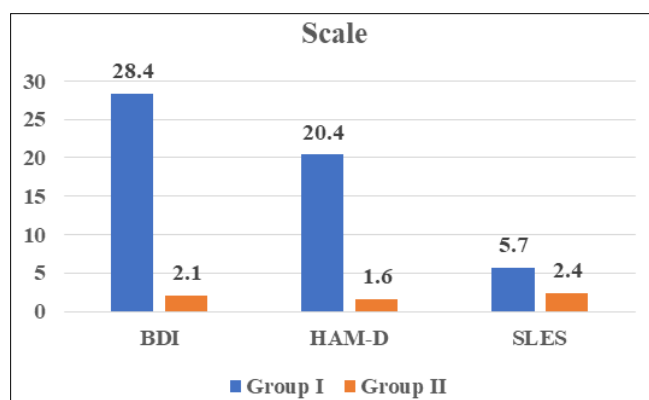
Total- 86		
Gender	Males	Females
Number	42	44

Table I shows that out of 86 patients, males were 42 and females were 44.

**Table 2:** Comparison of scale in both groups

Groups	Group I	Group II	P value
BDI	28.4	2.1	0.01
HAM-D	20.4	1.6	0.01
SLES	5.7	2.4	0.04

Table II, graph I shows that mean BDI scale was 28.4 in group I, 2.1 in group II. HAM-D was 20.4 in group I and 1.6 in group II and SLES was 5.7 in group I and 2.4 in group II. The difference was significant ( $P < 0.05$ ).



**Graph 1:** Comparison of scale in both groups

**Table 3:** Comparison of S100B levels in both groups

S100B (pg/ml)	Group I	Group II	P value
Male	98.2	67.1	0.82
Female	93.4	26.9	0.01

Table III shows that mean S100B level in group I males was 98.1 pg/ml and in females was 93.4 pg/ml and in group II males was 67.1 pg/ml and in females was 26.9 pg/ml. The difference was significant ( $P < 0.05$ ).

## Discussion

Depression can be described as a state of low mood or loss of interest in one's daily activities. Involvement of these several neurotransmitters, multiple neuroimmunological and neurohormonal pathways have been the basis of various experimental studies to find reliable blood-derived biomarkers to identify, diagnose, or subclassify the patients of depression and study the treatment response in them [7]. Many studies on depression are in accordance with the biochemical basis of depression that has been related to the state of blood-brain barrier (BBB) allowing/restricting a number of components to enter the brain milieu from the peripheral plasma milieu. The clinical presentation of depression at this stage of life can be atypical and is often complicated by personality difficulties and substance misuse [8]. A significant proportion of young people presenting with recurrent depression will go on to develop a bipolar disorder, with important implications for future pharmacological treatment choices [9]. Adolescents with sub-diagnostic levels of depressive symptoms show higher rates of early adulthood depression, substance misuse and adverse psychological and social functioning. When symptom severity reaches the threshold for diagnosis, there is a likelihood that depression will continue into early adult life [10].

S100B is produced by the astrocytes or can spill from injured astrocytic cells and enter the extracellular space or bloodstream [11]. It is also a neurotrophic factor involved in neuroplasticity which is disrupted in depression along with the decrease in levels of growth factors. Elevated S100B levels in serum or CSF can be correlated with the presence of neuropathological conditions, including neurodegenerative diseases or traumatic head injury. Effects of S100B depend on its concentration [12]. The present study was conducted to assess serum S100B level in patient with depression.

In present study, out of 86 patients, males were 42 and females were 44. Arora *et al.* [13] included forty-two confirmed cases of depression. Levels of serum S100B were significantly elevated in patients with major depression as compared to controls. Significantly higher levels of S100B were seen only in females as compared to their healthy counterparts. Serum S100B was higher in depressed participants with the recurrent disorder than those with single episode. No correlation of levels of this marker was seen with clinical severity of the patients. It was found that with increased duration of illness for which the patient was being treated with antidepressants, the patients had higher levels of S100B.

We observed that mean BDI scale was 28.4 in group I, 2.1 in group II. HAM-D was 20.4 in group I and 1.6 in group II and SLES was 5.7 in group I and 2.4 in group II. The mean S100B level in group I males was 98.1 pg/ml and in females was 93.4 pg/ml and in group II males was 67.1 pg/ml and in females was 26.9 pg/ml. Rothermundt *et al.* [14] reported that the serum S100B levels in melancholic-depressive patients were higher than those in normal controls. They suggested that since S100B is a marker for neuroplasticity, high level of serum S100B might correspond to neuron growth and synaptogenesis during the process of synaptic remodeling in depressive patients. Effects of S100B depend on its concentration.

The increase in S100B level signifies a breach in the integrity of the BBB; however, it is not a result of damage. When secreted in small nanomolar concentrations, it signifies the activation of growth and differentiation of neurons and astrocytes. Yang *et al.* [15] found that S100B levels in participants with the first episode were significantly lower than those with recurrent depression. Since S100B is a marker of neuroplasticity, in patients with recurrent depression, repeated damage, and regeneration of the neurons as a compensatory response could be the cause for increased S100B levels in the participants with recurrent depression.

### Conclusion

Authors found that serum S100B level was elevated in depression patients as compared to control subjects.

### References

1. Albert PR, Benkelfat C. The neurobiology of depression: Revisiting the serotonin hypothesis. II. Genetic, epigenetic and clinical studies. *Philos Trans R Soc Lond B Biol Sci* 2013;368:20120535.
2. Duman RS, Li N. A neurotrophic hypothesis of depression: Role of synaptogenesis in the actions of NMDA receptor antagonists. *Philos Trans R Soc Lond B Biol Sci* 2012;367:2475-84.
3. Felger JC, Lotrich FE. Inflammatory cytokines in depression: Neurobiological mechanisms and therapeutic implications. *Neuroscience* 2013;246:199-229.
4. Rothermundt M, Peters M, Prehn JH, Arolt V. S100B in brain damage and neurodegeneration. *Microsc Res Tech* 2003;60:614-32.
5. Karege F, Perret G, Bondolfi G, Schwald M, Bertschy G, Aubry JM *et al.* Decreased serum brain-derived neurotrophic factor levels in major depressed patients. *Psychiatry Res* 2002;109:143-8.
6. Schäfer BW, Heizmann CW. The S100 family of EF-hand calcium-binding proteins: Functions and pathology. *Trends Biochem Sci* 1996;21:134-40.
7. Rothermundt M, Arolt V, Wiesmann M, Missler U, Peters M, Rudolf S *et al.* S-100B is increased in melancholic but not in non-melancholic major depression. *J Affect Disord* 2001;66:89-93.
8. Missler U, Wiesmann M, Friedrich C, Kaps M. S-100 protein and neuron-specific enolase concentrations in blood as indicators of infarction volume and prognosis in acute ischemic stroke. *Stroke* 1997;28:1956-60.
9. Rothoerl RD, Woertgen C, Brawanski A. S-100 serum levels and outcome after severe head injury. *Acta Neurochir Suppl* 2000;76:97-100.
10. Arolt V, Peters M, Erfurth A, Wiesmann M, Missler U, Rudolf S *et al.* S100B and response to treatment in major depression: A pilot study. *Eur Neuropsychopharmacol* 2003;13:235-9.
11. Schroeter ML, Abdul-Khaliq H, Diefenbacher A, Blasig IE. S100B is increased in mood disorders and may be reduced by antidepressive treatment. *Neuroreport* 2002;13:1675-8.
12. Schroeter ML, Abdul-Khaliq H, Krebs M, Diefenbacher A, Blasig IE. Serum markers support disease-specific glial pathology in major depression. *J Affect Disord* 2008;111:271-80.
13. Arora P, Sagar R, Mehta M, Pallavi P, Sharma S, Mukhopadhyay AK. Serum S100B levels in patients with depression. *Indian J Psychiatry* 2019;61:70-6.
14. Rothermundt M, Arolt V, Wiesmann M, Missler U, Peters M, Rudolf S *et al.* S-100B is increased in melancholic but not in non-melancholic major depression. *J Affect Disord* 2001;66:89-93.
15. Yang K, Xie GR, Hu YQ, Mao FQ, Su LY. The effects of gender and numbers of depressive episodes on serum S100B levels in patients with major depression. *J Neural Transm (Vienna)* 2008;115:1687-94.