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SOME BIOCHEMICAL CHANGES IN PATIENTS WITH BACTERIAL MENINGITIS

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ABSTRACT

Gamma glutamyl transferase (GGT) and true cholinesterase, as well as each of the amino acids (glutamine, aspartic acid, glutamic acid, glycine, tyrosine, phenylalanine, histidine and tryptophan), except for gamma amino butyric acid (GABA) activity were significantly increased P≤0.001 in cerebrospinal fluid (CSF) of patients with bacterial meningitis (B.M). No change was observed in CSF electrolytes such as Sodium, calcium and magnesium except for potassium which was significantly increased P≤0.01 in patients with B.M as compared to healthy controls. Serum GGT activity was significantly elevated P≤0.01 in patients while serum true cholinesterase activity was significantly decreased p≤0.05 in patients with B.M. No change was observed in serum amino acids of patients with B.M as compared to healthy controls.

INTRODUCTION

Recently, Lutsar et al.,[1] reported that GGT was significantly higher in patients with bacterial meningitis on admission as compared with those with aseptic meningitis and meningism (P<0.05 and P<0.005, respectively) and decreased with therapy.

GGT activity was significantly elevated (P<0.001) in CSF and serum in meningitis as compared with control subjects. Levels were significantly higher in pyogenic than in tuberculous meningitis (P<0.001) and in CSF than in serum (P<0.001).

Obviously, GGT rises during acute inflammation of the meninges and the rise is greater in pyogenic meningitis (PM) than in tuberculous meningitis (TBM). Since the activity was highest on the first day and declined there after in all the cases who survived, this would mean that during the first few days the pathological process is at its peak; thereafter once the patient is given treatment, the vascularity of the meninges decreases and enzymatic activity declines. However, in cases where the activity increased on subsequent estimations, disruption of the blood brain barrier (BBB) continued and the enzyme continued to leak into the CSF, suggesting that the vascularity in these cases stayed higher. This could be because of fulminant disease in these cases or because drug therapy was not sufficiently effective to control the pathological process[2].

Meanwhile, Henry[3] reported that low values of serum cholinesterase were found in acute infections. Tonel et al.,[4,5] found that acetylcholinesterase (AChE) activity in CSF was higher in patients with meningitis than in controls.

Amino acids are known to be directly or indirectly involved in synaptic transmission in nervous tissues and some amino acids act as neurotransmitters[6].

Guerra et al., [7] reported that CSF concentrations of glutamate, aspartate, glycine, taurine and alanine increased significantly in infected animals. Among the amino acids with known excitatory or inhibitory function, interstitial fluid concentrations of glutamate were significantly elevated (by 470 %). Alanine, a marker for anaerobic glycolysis, also increased in the cortex of infected rabbits. The elevated glutamate concentrations in the brain extracellular space suggest that excitotoxic neuronal injury may play a role in bacterial meningitis.

In recent study, Perry et al.[8] found significant elevation of the excitatory amino acids aspartate and glutamate, as well as of the inhibitory neurotransmitters, gamma amino butyric acid (GABA) and taurine, in the excessive extracellular fluid of animals injected with *E.coli* compared with control animals injected with saline. However concentrations of these excitatory and inhibitory amino acids rose late in the course of meningitis.

Awadein[9] found that there was no significant difference between the values of sodium ion of patients with meningitis while the potassium values were significantly increased in patient's groups on admission compared to the normal control.

Venkataraman et al.[10] reported that the concentration of calcium and magnesium were not altered significantly in meningitis.

MATERIAL AND METHODS

The study was carried out on 30 patients with

bacterial meningitis, aged 7-20 years. 10 patients were not suffering from bacterial meningitis with matched age and sex served as a control group.

Methods:

Gamma glutamyl transferase (GGT) was determined in CSF and serum of patients with bacterial meningitis and normal controls as previously reported [11].

True cholinesterase was determined in CSF and serum according to the method of Ellman et al., [12]. Amino acids analysis was carried out in CSF and serum according to the method of Heinrikson and Meredith[13] using reversed phase, high performance, Liquid chromatography. Concentration of sodium, potassium, calcium and magnesium were determined in CSF using an atomic absorption spectrophotometer according to the method of Bernhard Welz [14].

RESULTS

30 patients of B.M. were studied. The mean CSF and serum GGT levels in patients with B.M. were significantly elevated P≤0.001 as compared to controls (Table 1) (Fig. 1,3). The mean CSF true cholinesterase level in patients with B.M. was significantly elevated, P≤0.001 as compared to controls while serum true cholinesterase level in patients with B.M was significantly decreased P≤0.05 (Table 1), (Fig. 2,4).

In the present study, there was a marked increase, in CSF amino acid levels (glutamine, aspartic acid, glutamic acid, glycine, tyrosine, phenylalanine, histidine and tryptophan) P≤0.001 and unchange in gamma amino butyric acid (GABA), P≤0.1 (Table 2), (Fig. 5, 6, 7, 8.)

Among the serum amino acid levels, there was no significant change between healthy controls and B.M. patients (Table 3), (Fig. 9, 10). The mean CSF potassium Level in patients with B.M. was significantly elevated P≤0.001, as compared to controls, while sodium, calcium and magnesium levels in CSF of patients with B.M. were not significantly changed as compared to controls (Table 4), (Fig. 11, 12).

DISCUSSION

The mean value of CSF GGT of patients with B.M was increased by 96.2%, P≤0.001 as compared to the normal control (Table 1), (Fig. 1). These results are in accordance with the previously reported results[11,12] which showed that CSF GGT in patients with meningitis was increased, P≤0.001 as compared to the control value (Table 1), (Fig. 3).

While, Akimov et al.[15] reported that the GGT enzyme activity proved to be drastically high in patients with cerebral diseases.

Also in other study Akimov et al.[16] revealed a sharp elevation in CSF GGT which points to dystrophic changes in nerve cells, damage to their membranes and consequently, an increase in the permeability of the blood-brain barrier. GGT activity in CSF may be recommended as a supplemental method to the differential diagnosis of cerebral meningitis.

Table (1): Mean levels ± SD of CSF and serum gamma glutamyl transferase and true cholinesterase (U/L) in controls and patients with bacterial meningitis

Enzyme	Control	Patients
Gamma glutamyl transferase (CSF) (Mean ± SD) Change	7.05±3.11	13.89±3.69* 9.32%
True cholinesterase (CSF)Mean ± SD Change	19.1±3.14	70.52±30.2* 269.2%
Gamma glutamyl transferase U/L (Serum) Mean ± SD Change	11.84±2.29	15.76±2.7* 33%
True cholinesterase (Mean±SD) Change	3119.9±561	2606.7±768* 16.47%

Data are presented as means ± S.D. P <0.05 was calculated using student "t" test. *The values of significance represent the difference between mean values of the meningitis patients and corresponding control.

Table (2): Mean levels ± SD of amino acids (glutamine, aspartic acid, GABA, glutamic acid, glycine, tyrosine, phenylalanine, histidine and tryptophan) μmol/L in CSF of controls and patients with bacterial meningitis

Enzyme	Control	Patients
Glutamine (Mean ± SD) Change	548±31.64 82.7%	1001.7±170.1*
Aspartic acid (Mean ± SD) Change	8.03±0.6	12.66±1.44* 57.6%
GABA (Mean ± SD) Change	1.4±0.09	1.49±0.24* 6.4%
Glutamic acid (Mean ± SD) Change	151.2±7.08	220.2±23.64* 45.6%
Glycine (Mean ± SD) Change	7.18±1.19	42.17±4.19* 487%
Tyrosine (Mean ± SD) Change	9.84 ± 1.47	76 ± 13.44* 701.6%
Phenyl alanine (Mean ± SD) Change	9.87±1.09	56.5±14.56* 472%
Histidine (Mean ± SD) Change	12.29±1.14	33.73±5.05* 174%
Tryptophan (Mean ± SD) Change	2.33±0.66	11.02±1.33* 372%

Data are presented as means ± S.D. P <0.05 was calculated using student "t" test. *The values of significance represent the difference between mean values of the meningitis patients and corresponding controls.

While the patient mean value of serum GGT was increased by 33%, P≤0.001.

The obvious levels were significantly higher in the CSF than in the sera. This rise in serum can not be explained on the basis of disturbed blood brain barrier (BBB) because normally enzymatic activity is very low in the CSF as compared to the serum. Moreover, there seems to be no cause for a rise in serum, if the blood brain barrier (BBB) is intact. Several studies have shown that when the (BBB) is intact, CSF enzymatic activity is unaffected by serum changes.

Other workers observed significantly elevated activity of enzymes in the CSF and no correlation was observed between CSF and serum activity in patients with meningitis [17,18].

The mean value of CSF true cholinesterase of patients with bacterial meningitis was significantly increased by 269.2%, P≤0.001 as compared to the normal control (Table 1) , (Fig. 2). Our results are in accordance with many authors.

Tietz et al.,[19] found that CSF cholinesterase activity was often increased in meningitis patients. While recent reports[4,5] indicated that acetylcholinesterase (AChE) activity in CSF was higher in patients with meningitis than in controls.

On the other hand, the patient mean value of serum true cholinesterase showed a significant decrease by 16.472%, P≤0.05 as compared to the normal control (Table 2). (Fig. 4). These results are in good agreement with literture data[3] which indicated low values of serum cholinesterase in patients with acute infections.

Choline enters the CNS through a carrier mediated transport process that can be inhibited by molecules such as dimethyl amino ethanol, hemicholinium and tetra ethyl ammonium chloride. Since choline can not be synthesized by the brain, it has been proposed that the blood-brain barrier transport may regulate the formation of acetylcholine in the CNS[20].

The source of acetylcholinesterase (AChE) in CSF may be the small portion of "naturally soluble" enzyme found in mammalian brain. Inflammation would lead to an increase of enzyme secretion from the intracellular pool of soluble AChE[4].

The origin of cerebrospinal fluid-acetyl cholinesterase (CSF-AChE) is unknown. It has been shown that AChE is secreted from nervous tissue into the CSF. The rise in AChE in CSF of patients with meningitis could be due to an increased release of this enzyme because the disease might influence the secretion mechanism[5].

Many authors reported that enzyme activity in CSF in meningitis may be raised due to the following reasons:

1- An increased out flow from serum is due to injury to the blood brain barrier (BBB).

2- Presence of cells or bacteria in CSF.

3- Injury to the brain tissue itself and increased release of the enzyme in CSF.

4- Decreased rate of removal of the enzyme from CSF.

5- A combination of some or all of the above reasons [2,21].

The mean value of CSF glutamine in patients with B.M. was significantly increased by 82.7%, P≤0.001 as compared to normal controls (Table 2) (Fig. 5). These results are in good agreement with the following authors.

Some authors [22-24] reported an increase in the concentration of CSF glutamine in patients with

Table (3): Mean levels ± SD of amino acids (glutamine, serine, glutamic acid and glycine) µmol/L in serum of controls and patients with bacterial meningitis.

Enzyme	Control	Patients
Glutamine (Mean ± SD) Change	630 ± 50.79	653.5±39.28 3.73%
Serine (Mean ± SD) Change	140.5±23.18	134.8±22.88 4.10%
Glutamic acid (Mean ± SD) Change	64.5±10.11	66±10.07 2.32%
Glycine (Mean ± SD) Change	253±29.68	245±31.97 2.96%

Data are presented as means ± S.D., P <0.05 was calculated using student "t" test. The values of significance represent the difference between mean values of the meningitis patients and corresponding control.

Table (4): Mean levels ± SD of CSF (sodium Na^+, potassium K^+, calcium Ca^{2+} and magnesium Mg^{2+}) mEq/L in controls and patients with bacterial meningitis.

Enzyme	Control	Patients
Sodium (Mean ± SD) Change	134.6±7.46	135.8±9.9 0.89%
Potassium (Mean ± SD) Change	1.84±0.611	2.98±1.03* 62%
Calcium (Mean ± SD) Change	2.3±0.29	2.44±0.54 6.1%
Magnesium (Mean ± SD) Change	2.47±0.21	2.41±0.386 2.43%

Data are presented as means ± S.D. P <0.05 was calculated using student "t" test. *The values of significance represent the difference between mean values of the meningitis patients and corresponding control.

purulent meningitis and elevation in the concentration of CSF glutamine in patients with aseptic meningitis.

The mean value of CSF aspartic acid in patients with B.M. was significantly increased by 57.6%, P≤0.001 (Table 2). (Fig. 6). These results are in good agreement with those obtained before [7,8,24,25].

The mean value of CSF glutamic acid in patients with B.M. was highly significantly increased by 45.6%, P≤0.001 (Table 2), (Fig. 5). These results showed good agreement with those obtianed before[7,8] who found significant elevation of the excitatory amino acids, aspartate and glutamate in the CSF of animals with meningitis.

The mean value of CSF GABA in patients with B.M. was insignificantly increased by 6.4%, P≤0.1 as compared to controls (Table 2), (Fig. 7). These results are in accordance with Kuroda[26] who reported that insignificant changes of CSF were observed in Patient with meningitis.

The mean value of CSF glycine in patients with B.M. was increased by 487%, P≤0.001 (Table 3), (Fig. 6). These results are in accordance with those results obtained recently [7,24,25,27].

The mean value of CSF tyrosine in patients with B.M. was increased by 701.6%, P≤0.001 as compared with control (Table 2), (Fig. 8). These results are in good agreement with those of San Joaquin et al.[25] and Halawa et al.[24].

The mean value of CSF phenylalanine in patients with B.M was significantly increased by 472%, (P≤0.001) as compared with controls (Table 3), (Fig. 8). These results are in accordance with the results of Corston et al. [22] and San Joaquin et al. [25].

The mean value of CSF histidine in patients with B.M. was increased by 174%, P≤0.001 as compared with controls (Table 2), (Fig. 8). These results are in good agreement with those of Corston et al.[22] and San Joaquin et al.[25] who found increased concentrations of phenylalanine and histidine in CSF of patients with meningitis. The mean value of CSF tryptophan in patients with B.M. was increased by 372%, P≤0.001 (Table 2) (Fig. 7).

Also, Briem et al.[28] reported increased total concentrations of amino acid in CSF of patients with purulent meningitis. San Joaquin et al.[25] found increased concentrations of tryptophan in the CSF of patients with meningitis. Schott and Meier [29] revealed that almost all CSF amino acids were highly elevated in meningitis.

In general, Ohtsuka [30] reported that the levels of free amino acids in CSF increased in the acute phase of bacterial meningitis.

As to amino acid in serum in this study glutamine, serine, glutamic acid and glycine were measured in patients and normal groups. The mean values of serum (glutamine, serine, glutamic acid and glycine) were insignificantly changed p≤0.1 (Table 2), (Fig. 9, 10). The aforementioned results are in good agreement with the following authors.

Corston et al.[22] reported that plasma amino acid concentration did not differ significantly from normal P>0.05. San Joaquin et al.[25], demonstrated that serum levels do not account for the altered CSF pattern. Among the plasma amino acid levels, there was no significant change between healthy controls and aseptic meningitis patients [24].

The changes in CSF and serum levels of different amino acids may be due to damage to nervous tissue rather than to alteration in brain metabolism or alteration in the removal of amino acids by active transport.

Heiblim et al.[27] suspected that the reason for the elevations of most amino acids in CSF during the acute phase of bacterial meningitis may be secondary to alterations in brain metabolism, changes in the kinetics of formation of CSF at choroidal and extrachoroidal sites, and alterations in the removal of amino acids (Some to a greater extent than others) by active transport mechanisms, or a combination of factors.

Corston et al.[27] reported the possibility that the increase in CSF amino acid concentrations might result, at least in part, from amino acid release from damaged nervous tissue. This has some support from the significant increase noted for CSF glutamine concentrations, since glutamine is one of the major amino acid components of cerebral tissue.

In a recent study, Halawa et al.[24] reported that:

(i) in the CSF samples, the increase in most of the essential amino acids such as phenylalanine, in the patients with aseptic meningitis, may be caused by the breakdown of the brain-blood barrier. It is generally known that carrier-mediated transport plays an important role for some amino acids not only at the blood brain barrier but also at the blood CSF barrier.

(ii) among the non-essential amino acids, the level of glutamine is increased in patients with aseptic meningitis. This was as a result of glutamine that being released from the damaged nervous tissue. Glutamine is released into the CSF because it is a major component of cerebral tissue. It is also found to be increased in the CSF during various infectious diseases.

(iii) among the neurotransmitter amino acids, the

Fig. (1): The mean levels of cerebrospinal fluid (GGT) U/L in controls and patients with bacterial meningitis

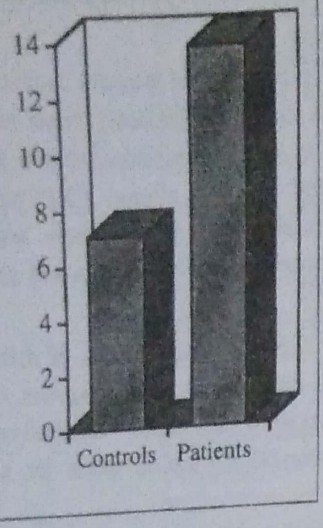

Fig. (2): The mean levels of cerebrospinal fluid true cholinesterase U/L in controls and patients with bacterial meningitis

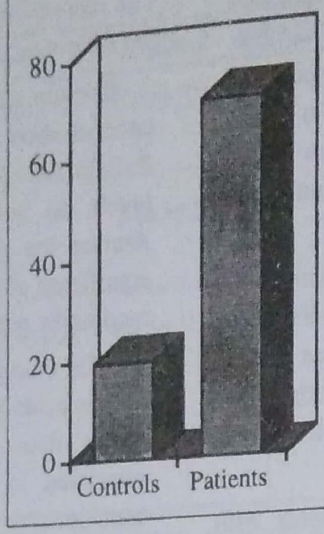

Fig. (3) : The mean levels of serum (GGT) (U/L in controls and patients with bacterial meningitis

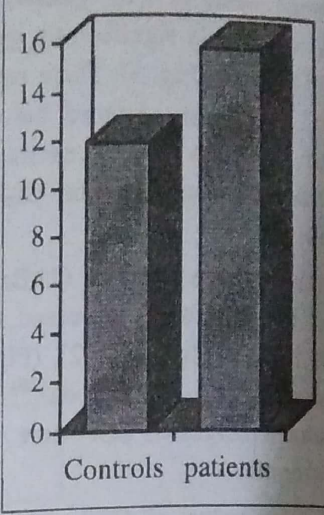

Fig. (4): The mean levels of serum true cholinesterase (U/L) in controls and patients with bacterial meningitis

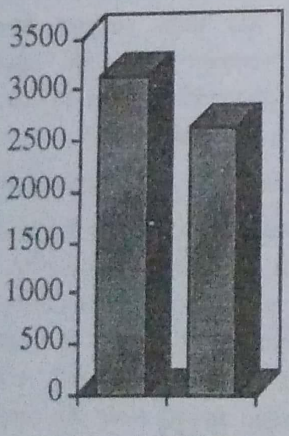

Fig. (5): The mean levels of cerebrospinal fluid amino acids (glutamine and glutamic acid) µmol/L in controls and patients with bacterial meningitis

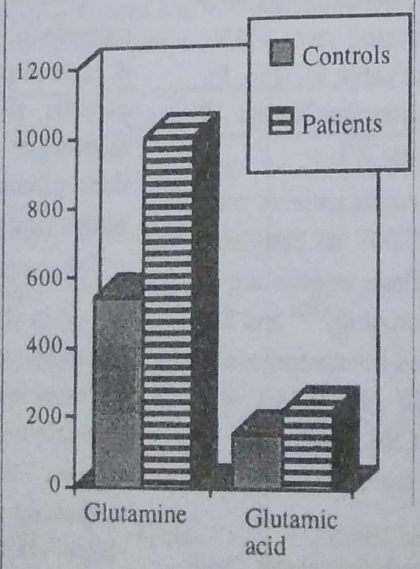

Fig. (6): The mean levels of cerebrospinal fluid amino acids (aspartic acid and glycine) µmol/L in controls and patients with bacterial meningitis

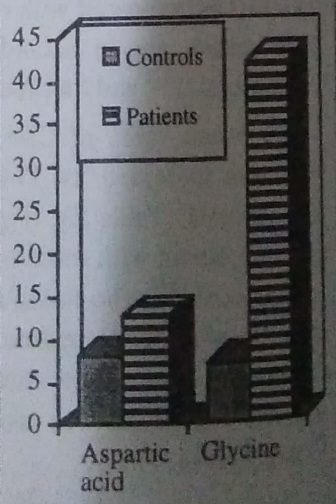

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Fig. (7): The mean levels of cerebrospinal fluid amino acids (GABA) and tryptophan µmol/L in controls and patients with bacterial meningitis.

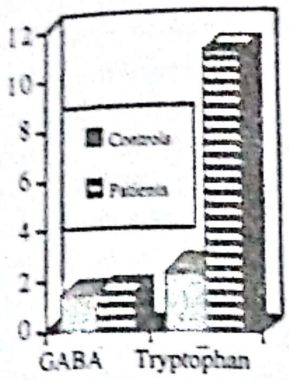


Fig. (8): The mean levels of cerebrospinal fluid amino acids (tyrosine, phenylalanine and histidine) µmol/L in controls and patients with bacterial meningitis.

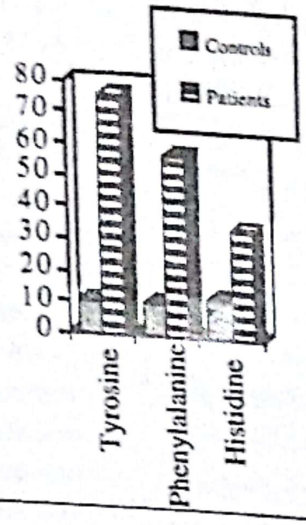


Fig. (9): The mean levels of serum amino acids (glutamine and glycine) µmol/ L in controls and patients with bacterial meningitis.

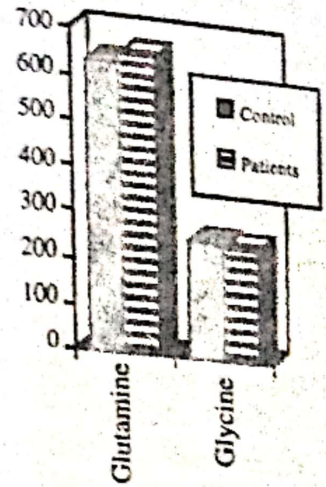


Fig (10) The mean levels of serum amino acids (serine and glutamic acid) µmol/L in controls and patients with bacterial meningitis.

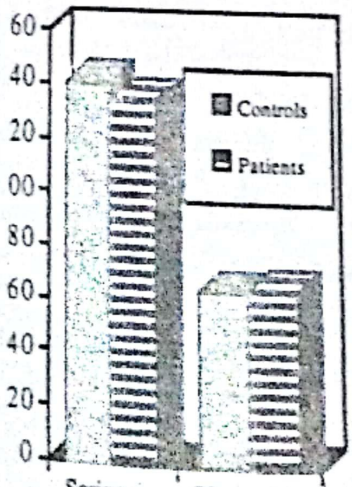


Fig. (11) The mean levels of cerebrospinal fluid (sodium) mEq/L in controls and patients with bacterial meningitis.

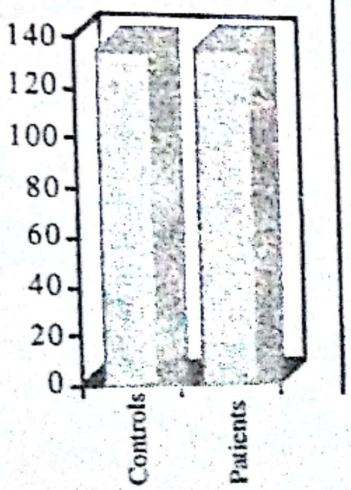
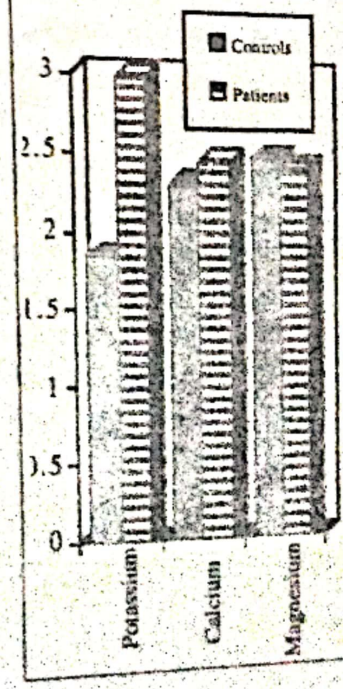


Fig. (12): The mean levels of cerebrospinal fluid (potassium, calcium and magnesium) mEq/L in controls and patients with bacterial meningitis.



levels of aspartic acid, glutamic acid and glycine are increased. Among these, glutamic acid is the most abundant in the central nervous system and has several clinically important pathways. Glutamic acid is a neurotransmitter which may produce toxic damage in the central nervous system because the increased levels are shown to cause excitotoxic cell damage and may lead to certain neurodegenerative diseases.

Same author reported that, the increase of glycine is suggestive of increased release from spinal cord and brain or decreased out flow from CSF as plasma amino acids in patients with aseptic meningitis remain unchanged as compared to the control group. These differences can not be adequately explained at this time. Possible causes include the different techniques used in the amino acid analysis as well as the diversity of study populations in terms of age, etiology of the meningitis, severity of these disease and duration of the illness prior to the measurement of the CSF amino acids [25].

In our opinion, we noticed that there is no relation between the levels of the studied amino acids in the sera and CSF of patients with B.M compared to controls. Also we can conclude that excitatory amino acids are increasingly implicated in the pathogenesis of B.M.

As to the electrolytes (sodium, potassium, calcium and magnesium) were measured in CSF of B.M. patients and control group. The mean value of CSF Na^+ of patients with B.M. was insignificantly increased by 0.89%, $P \leq 0.1$ (Fig 11).The mean value of CSF K^+ of patients with B.M. was significantly increased by 62%, $P \leq 0.001$ as compared with controls (Table 4), (Fig. 12). These results are in good agreement with those of Awadein[9] who reported a highly significantly increase in the CSF potassium levels in patients with B.M. but observed no change in the sodium levels in the same patients.

While the mean value of CSF Ca^{2+} of patients with B.M. was insignificantly increased by 6.1%, $P \leq 0.1$ and magnesium showed insignificant decrease by 0.56%, $P \leq 0.1$ (Table 4), (Fig. 12). These resutls are in accordance with those of Venkataraman [10] who found that the concentrations of Ca^{+2} and Mg^{+2} were not altered in patients with meningitis.

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بعض التغيرات البيوكيميائية فى مرضى الإلتهاب السحائى البكتيرى

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تم فى هذا البحث دراسة ٣٠ حالة مرضية بالإلتهاب السحائى البكتيرى وأعمارهم تتراوح بين ٧ إلى ٢٠ سنة تقريبا وذلك بأخذ عينة من السائل النخاعى الشوكى وعينة من الدم لكل مريض وقياس مستوى بعض الانزيمات، بعض الأحماض الأمينية والالكتروليتات فى عينة السائل النخاعى الشوكى ومصل الدم لكل مريض بالإلتهاب السحائى البكتيرى بالمقارنة بالمجموعة الضابطة.

أظهرت هذه الدراسة ما يأتى:

مستوى انزيم الناقل للجلوتاميل قد زاد زيادة ذات قيمة احصائية عاليه فى عينات السائل النخاعى الشوكى ومصل الدم فى حالة مرضى الالتهاب السحائى البكتيرى عند مقارنتهم بالمجموعة الضابطة كما لوحظ أن الزيادة فى مستوى الانزيم فى عينات السائل النخاعى الشوكى أكثر من الزيادة فى عينات مصل الدم وذلك لنفس المرضى.

أما بالنسبة لانزيم الكولين استريز الحقيقى فقد لوحظ زيادة عالية لها قيمة احصائية فى عينات السائل النخاعى بينما يقل فى مصل الدم لمرضى الالتهاب السحائى البكتيرى عند مقارنتها بالمجموعة الضابطة. ومعنى ذلك أنه لا يوجد علاقة بين تركيز الإنزيمات فى عينات السائل النخاعى وتركيزها فى مصل الدم فى مرضى الالتهاب السحائى البكتيرى.

أما فى حالة الأحماض الأمينية نجد أن مستوى كل من الجلوتامين، حمض الأسبارتيك، حمض الجلوتاميك، التيروسين، الفينيل ألانين، الهستدين والتربتوفان، قد زادوا زيادة عاليه لها قيمة احصائية فى عينات السائل النخاعى الشوكى لمرضى الالتهاب السحائى البكتيرى عند مقارنتها بالمجموعة الضابطة.

أما بالنسبة لحمض الجاما أمينو بيوتيرك لم يحدث له تغير ملحوظ فى عينات السائل النخاعى لمرضى الالتهاب السحائى البكتيرى عند مقارنتها بالمجموعة الضابطة وفى حالة الأحماض الأمينية (الجلوتامين، السيرين، حمض الجلوتاميك، والجليسين نجد أنه لم يحدث لهم تغير ملحوظ له قيمة احصائية فى عينات مصل الدم لمرضى الالتهاب السحائى البكتيرى عند مقارنتهم بالمجموعة الضابطة أى أنه لا يوجد علاقة بين تركيز الأحماض الأمينية فى عينات السائل النخاعى الشوكى وعينات مصل الدم فى مرضى الالتهاب السحائى البكتيرى. وفى حالة الإلكتروليتات وجد أنه لم يحدث تغير ملحوظ له قيمة احصائية فى تركيز أيون الصوديوم، أيون الكالسيوم وأيون الماغنسيوم فى حين أن تركيز أيون البوتاسيوم قد زاد زيادة واضحة لها قيمة إحصائية فى عينات السائل النخاعى الشوكى لمرضى الالتهاب السحائى البكتيرى عند مقارنتهم بالمجموعة الضابطة.

من كل هذه النتائج السابقة يمكننا أن نستنتج أن هذه العناصر المعنية محل الدراسة يمكن استخدامها فى التشخيص المبكر والمتابعة لمرضى الالتهاب السحائى البكتيرى.