EVALUATION OF CEREBROSPINAL FLUID GLUTAMIC-OXALOACETIC TRANSAMINASE ACTIVITY IN PATIENTS OF HEAD INJURY

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The G.O.T. activity in C.S.F. has been determined in 35 cases of acute head injury. The clinical manifestation of neuronal damage in most cases is correlated with the alternations in the CSF-GOT levels. The enzyme activity reaches a peak level on 3rd or 4th day after the injury and then declines gradually towards the normal level in the following days.

Apart from well recognised routine clinical and laboratory methods, the estimation of Glutamic-Oxaloacetic Transaminase (G.O.T.) in the cerebrospinal fluid (CSF) is considered to be a new technique for the detection of the presence and degree of neuronal damage suffered by a patient of cranio-cerebral trauma. Wakim and Fleisher (6) in their experimental studies observed an increase in CSF-GOT levels, which roughly paralleled the degree of neuronal damage artificially inflicted.

In the present study, the CSF-GOT has been serially estimated in patients of head injury admitted to Rajendra Hospital, Patiala. The significance of the findings is discussed.

MATERIALS AND METHODS

The methods of Yatzidis (7), as employed for serum, was utilised for GOT estimation on CSF, using the units of Cabaud, Leeper and Wroblewski (1).

The material comprised of 35 cases of head injury (28 males and 7 females).

RESULTS

The upper limit of CSF-GOT activity in our normal controls was 40 units per m. Any figure above this level was considered as abnormal. Depending upon the results of CSF-GOT estimation, the patients under study were divided into three groups.

GROUP 'A' consisted of 17 patients (Nos. 1 to 17), in whom the highest level (peak) of the CSF-GOT was more than 40 units/ml.

GROUP 'B' consisted of 16 cases (Nos. 18 to 33), in whom the peak CSF-GOT level was less than 40 units/ml.

GROUP 'C' included two patients (Nos. 34 and 35), admitted for treatment of the sequelae of their old head injuries, and who had their enzyme levels below 40 units/ml.

GROUP 'A'

The results of CSF-GOT estimations in these patients, except two cases, are shown in Fig I.
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The following observations were drawn from the analysis of these cases:

In 7 out of 10 cases who reported on the first or second day of their injury, the rise to
maximum in their enzyme activity was detected on the third or fourth day of injury. This
sustained gradual rise to maximum after injury was seen only in those patients in whom the initial
enzyme level was sufficiently high.

The patient No. 13 showed a secondary rise on the second post-operative day. This case
was admitted on the 5th day of the injury and had an open wound in the skull from which brain
matter was protruding out. The enzyme level on the day of his admission was 85 units/ml.
The patient was operated upon and necrotic brain tissue and debris were removed. Next day,
the enzyme level rose to 91 units/ml.

After the “peak” level, in all the cases enzyme activity, as assessed by serial estimations,
tended to gradually decrease. Generally, in cases with less neuronal damage, it usually reached
the normal limit in about a week’s time, but in patients with severe neuronal damage it took
relatively longer time to do so.

Three patients had their “peak” CSF-GOT levels above 90 units/ml. Two of these (Nos
3 and No. 15), whose enzyme activities registered “peak” levels above 100 units/ml, died. The
third (No. 13) with “peak” level of 91 units/ml, who had an open wound in the skull, survived,
but was left with right sided hemiplegia and speech defect.

Of the four cases, who had “peak” levels between 70-90 units/ml, three were semicomatose
and the fourth was severely confused as assessed by the criteria of Rowbotham (4). One
discharge, one of them had signs of residual hemiplegia and aphasia. The remaining three showed residual mental deficits, prolonged post-traumatic amnesia and headache.

Out of the six patients with their CSF-GOT “peak” levels between 50-70 units/ml, one left the hospital with a permanent speech defect and another with mental changes. In the remaining four patients no neurological defect was detected.

Two patients (Nos. 14 and 16), admitted on 14th day of head injury, had the GOT “peak” levels between 55-60 units/ml. One, out of them, was comatose and the other severely confused. They did not improve much during their hospital stay. Follow up studies showed signs of hemiplegia and mental deficit in one while the other could not be contacted.

In cases Nos. 5 and 7 (not included in Fig. 1), the “peak” activities were only slightly higher (i.e. 44 and 45 units/ml) than normal level and both of them recovered completely within a week’s time.

**GROUP ‘B’**

In all the sixteen patients, there was a definite history of unconsciousness following head injury. Their enzyme levels were within normal limits and none of them showed any sign of neurological injury, although four patients revealed linear skull fractures. In most of them there were associated injuries like scalp wound, haematoma and fracture of the limbs or jaws.

**GROUP ‘C’**

In both patients of this group the preoperative and post-operative enzyme levels were below 40 units/ml. These cases had paralytic sequelae due to old head injuries. In both of them X-ray showed depressed skull-fracture. In each case the fracture was elevated under general anaesthesia, the operative interference being limited to outside the dura mater only.

**DISCUSSION**

The experimental studies of cerebral infarction on animals by Wakin and Fleisher, (6) and Smith, et al (5) indicated that the destruction of the brain cells resulted in an elevation of CSFGOT level and the rise in enzyme was proportional to the degree of damage inflicted. Our observations in cases of head injury in human beings are generally in agreement with the findings of the above mentioned workers.

Patient No. 3 of group ‘A’ sustained head injury by a headlong fall, and immediately became unconscious and remained so, till he died on the 15th day of his injury. Postmortem examination revealed a big area of laceration of the left temporal lobe and generalised congestion of the brain tissue. The “peak” CSF-GOT activity registered was 107 units/ml, the highest recorded in our series.

Similar serious clinical picture and fatal end result was observed in patient No. 15, who registered a “peak” activity of 105 units/ml. The postmortem examination could not be carried out.

Evidently in both these cases there was a close relationship between the CSF-GOT activity and neuronal damage. The observations on case No. 13 were especially revealing. This patient sustained brain injury by a sword and the brain matter was protruding out through the skull wound. The CSF-GOT level on the fifth day of injury was 85 units/ml. On the 7th day neces-
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primary operative intervention was carried out, the debris and necrotic brain tissue removed. The operative procedure resulted in some further unavoidable trauma to the brain tissue and this was reflected correspondingly in the elevation of the CSF-GOT level to 91 units/ml. This secondary rise in CSF-GOT level would not have been there but for the operative interference. Liberman et al (3) also reported similar findings in respect of their two patients.

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<tr>
<th>Relationship between C.S.F-G.O.T. level and the Clinical Picture of Patients</th>
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<tr>
<td>C.S.F.-G.O.T. “Peak” Units</td>
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<td>----------------------------</td>
</tr>
<tr>
<td>90 or above</td>
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<td>70—90</td>
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<td>50—70</td>
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<td>55—60*</td>
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<td>40—50</td>
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<td>Below 40***</td>
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*Patients who reported on the 14th day of their head injury.
**One of the two patients who returned for follow up.
***Patients of group ‘C’ who have been excluded from the category of chronic cases.

An overall survey of Group A and B cases, as shown in Table I, indicates that the higher the CSF-GOT level the worse the prognosis. Smith et al.,(5), from their clinical study, also concluded that there is a close relationship between the amount of neuronal damage and the peak of CSF-GOT activity.

In patients of Group B, the SCF-GOT estimations indicated absence of any neuronal damage, as enzyme activity in all of them was within normal limits. This inference was fully supported by their clinical picture and follow up. This conclusion of ours is in line with the observations of Dioguardi et al (2), that trauma to the skull and shock after head injury as such do not alter the levels of CSF-GOT activity.

In patients of Group C, both preoperative and postoperative levels of CSF-GOT were within normal limits. The normal preoperative level was explained by the long interval between injury and reporting of patients. The postoperative level did not rise, because operation was strictly extradural.

Finally, it may be concluded that GOT liberated as a result of neuronal injury, rises to a "peak" level on 3-4th day and then progressively declines to pretraumatic level. Therefore, GOT estimation on 3-4th day after head injury can be of value in early detection of neuronal damage and to some extent for prognosis.

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REFERENCES


