THIAMINE DEFICIENCY IN HEAD INJURY: A MISSED INSULT?

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Abstract — Practice regarding the use of thiamine in head-injured patients at risk of Wernicke–Korsakoff syndrome in Scottish neurosurgical units was surveyed by questionnaire and revealed no clear policy. A 2 year retrospective study of 218 admissions to one of these units of patients who had taken alcohol shortly before sustaining head injury is also described. The minority (20.6%) of the total had been given thiamine, with just over half (56.1%) of those categorized as alcoholic receiving this treatment. Additional carbohydrate loads, in the form of i.v. dextrose or parenteral nutrition, had been given to 44.5% of patients and only 28.9% of this group had also been given thiamine. The dose and duration of thiamine given was inadequate in most cases. It is suggested that failure to ensure that head injury patients at risk of Wernicke–Korsakoff syndrome receive appropriate thiamine prophylaxis represents a missed and treatable additional insult to the damaged brain.

INTRODUCTION

Alcohol abuse is an important contributing factor in traumatic brain injury (TBI), with studies showing that approximately one in two of the population presenting to hospital with a head injury is an alcohol abuser, compared with only one in ten in the general population (Dikmen et al., 1995; Corrigan et al., 1995; Kelly, 1995). It has also been shown that alcohol abusers suffer greater mortality and morbidity from TBI than non-drinkers and have a poorer eventual outcome (Ruff et al., 1990; Rönty et al., 1993; Corrigan et al., 1995). In particular, increased cognitive deficits are associated with alcoholism, both at the time of discharge from hospital and in the longer term (Sparadeo and Gill, 1989; Brooks et al., 1989). Several possible reasons for these findings have been postulated, but little attention has been paid to the possible role of thiamine (vitamin B1) deficiency in this population. Alcohol abuse is the commonest cause of thiamine deficiency in Western countries, and thiamine deficiency is considered a likely factor in neuropsychiatric syndromes associated with alcoholism such as the Wernicke–Korsakoff syndrome (WKS) (Victor et al., 1989; Lishman, 1990; Joyce, 1994).

The acute form of WKS, Wernicke’s encephalopathy, is notoriously difficult to diagnose clinically, with as few as one third of cases being diagnosed during life (Harper, 1979; Harper et al., 1995). Its diagnosis in the head-injured patient is made even more problematic as the primary symptoms and signs of confusion, ataxia, ophthalmoplegia and nystagmus, are also commonly seen as direct consequences of the head injury itself (Brismar et al., 1983). Memory disturbances, seen as part of both Wernicke’s encephalopathy and the more chronic presentation, Korsakoff’s psychosis, are also very common sequelae of head injury (Victor et al., 1989). With increasing acceptance that even sub-clinical thiamine deficiency has a pathogenic role in Korsakoff’s psychosis (Lishman, 1990), the possibility of an association between thiamine deficiency and the longer term cognitive problems after TBI exists.

As part of acute management of TBI, it is common for patients with a depressed level of consciousness, who may also be under the influence of alcohol, to be rehydrated with dextrose infusions or given total parenteral nutrition (TPN) which has a high glucose content. These carbohydrate loads, added to that of any

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Table 1. Definitions used to categorize patients according to alcohol history

<table>
<thead>
<tr>
<th>Units of alcohol/week</th>
<th>Alcoholic</th>
<th>Heavy drinker</th>
<th>Moderate drinker</th>
<th>Light drinker</th>
<th>Teetotal</th>
<th>Unknown</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male &gt; 50</td>
<td></td>
<td>Male 21-50</td>
<td>Male &lt; 21</td>
<td></td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Female &gt; 35</td>
<td>Alcohol, known alcoholic</td>
<td>Female 14-35</td>
<td>Female &lt; 14</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Descriptive terms</td>
<td>Alcohol, heavy, excessive</td>
<td>Moderate</td>
<td>Social, occasional</td>
<td>Teetotal, none, abstains</td>
<td>Unknown, blank (not recorded)</td>
<td></td>
</tr>
</tbody>
</table>

alcohol ingested prior to injury, could be responsible for further depletion of already low thiamine stores, and may precipitate Wernicke's encephalopathy (Vortemeyer et al., 1992; Chataway and Hardman, 1995).

Despite significant progress in recent years in identification, prevention and treatment of secondary insults in head injury, cognitive deficits remain an important factor preventing head-injured patients from participating in, and benefiting fully from, the rehabilitation process. Cognitive rather than physical deficits can be the primary factor determining whether a patient successfully re-integrates into the community following head injury (Newcombe, 1982; Long and Webb, 1983; Brooks, 1991). In our clinical experience, in a brain injury rehabilitation centre, a number of patients recovering from TBI who were known to have alcohol problems had not received thiamine supplementation during their initial care in the neurosurgical units which had referred them.

The present study aimed to establish the current practice regarding the use of thiamine, as treatment or prophylaxis, in the four specialist neurosurgical units in Scotland and to document the actual pattern of thiamine use in one regional neurosurgical unit, establishing whether risk factors for thiamine deficiency identified by history, clinical examination or laboratory tests were leading to the administration of thiamine to patients.

METHODS

To determine practice with regard to thiamine prophylaxis, a questionnaire was administered to consultants in the four neurosurgical centres in Scotland: Aberdeen, Dundee, Edinburgh and Glasgow. This addressed whether a written policy was used and/or junior medical staff were routinely instructed regarding thiamine prophylaxis after head injury. They were also asked the circumstances in which thiamine would be given by simple yes or no options, with the opportunity to specify additional examples to the options of: a known history of alcoholism; evidence of self neglect and clinical stigmata of alcoholic liver disease; smelling of alcohol; or all cases of coma where history was unavailable. Enquiry was not made as to dosage or route of administration of thiamine. The questionnaire is not reproduced here, but copies are available from the first author on request.

The Department of Clinical Neurosciences at the Western General Hospital, Edinburgh maintains a data base concerning all TBI cases admitted to the unit, which serves South East Scotland, population ~1.2 million. It is compiled by clinical and research staff, who kindly granted us access to data for the years 1994 and 1995. Included in the routinely collected details is whether a patient has 'had alcohol on board' at the time of injury, based on: a history of drinking shortly before injury; alcohol detected on the breath clinically or by alcometer; or a positive blood alcohol result. The case records of all patients so identified were scrutinized. Information collected included the cause of injury and its severity according to the Glasgow Coma Scale (GCS) (Teasdale and Jennett, 1974); clinical findings; results of laboratory investigations and treatment given. Particular attention was given to details of alcohol consumption and any previous alcohol related medical history. Patients were categorized, wherever possible, according to levels of alcohol consumed as described by the Medical Council on Alcoholism (1995) as shown in Table 1. Treatment details included the dose and duration of thiamine treatment if administered.
and the amounts and duration of i.v. dextrose or artificial feeding given.

Results were entered into a data base, observing the requirements of the Data Protection Act, and were analysed using Microsoft Excel (version 4.0a) and SPSS for Windows (Release 6.0).

RESULTS

Neurosurgeon questionnaire

All four Scottish neurosurgical units returned the same answers to the questionnaire. None of the units had a written protocol or specifically instructed their juniors with regard to vitamin B1 treatment or prophylaxis. All said that they would treat patients with thiamine prophylactically if there was a known history of alcoholism or stigmata of chronic alcoholic liver disease, but none would treat patients on the basis of their smelling of alcohol or where their history was unknown or under any other specific circumstances.

The study population

There were 894 TBI admissions recorded in the database for the years 1994 and 1995, and 241 (27%) were recorded as ‘having alcohol on board’ at the time of injury. Case records were available for 222 of this group. In four instances, scrutiny of the notes failed to confirm any evidence of alcohol consumption before injury either from history or examination including breath or blood alcohol tests. The remaining 218 admissions formed the study population. Three patients had two separate admissions for TBI in the period.

According to alcohol history, 41 (18.8%) were alcoholics, 42 (19.3%) heavy drinkers, 42 (19.3%) moderate drinkers, 31 (14.2%) light drinkers, three (1.4%) teetotallers and in 59 patients (27%) the alcohol history was unknown or not recorded. For the purposes of analysis, the teetotaller group is included with the light group.

The age distribution of the population is shown in Fig. 1. The median age for patients was 38.2 years (mean 42.2; range 14.8–88.6 years).

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The alcoholics had the highest median age at 49.3 years, and median (and mean) age in each group decreased with decreasing alcohol intake to the light/teetotaller group at 26.8 years. The differences in the means for each category were statistically significant (analysis of variance between groups: sum of squares = 10510.8856, d.f. = 3, mean square = 3503.6285, $F = 14.5398$, $P < 0.0001$). Males comprised 202 (97.7%) of the population.

Cause and nature of injury

A cause of injury was recorded in the notes of
197 patients (90.4%). The commonest was a fall (47.6%), then assault (38.6%), followed by pedestrian road traffic accident (RTA) (7.6%), car RTA (3.2%), motorcycle and bicycle RTA and sport (0.5% each) and 'other' (1.5%). Figure 2 summarizes the cause of injury for each alcohol consumption category. The alcoholic group had a higher incidence of falls at 66.7%, with a corresponding decrease in the number of assaults, but this fell short of statistical significance at the 95% probability level (Kruskal–Wallis one-way ANOVA: \( \chi^2 = 6.4094, \text{d.f.} = 4, P = 0.1706 \)). The number of pedestrian RTAs and injuries of unknown mechanism were approximately the same across the groups, apart from the occasional/teetotaller group, which had no injuries of unknown mechanisms recorded. There were no car, motorcycle or bicycle RTAs or sporting injuries in the alcoholic group.

The nature of the injury was classified simply into focal or diffuse, where focal indicates localized brain damage including haematoma on CT scan or depressed fracture, and diffuse refers to cases with no local lesions on scan but includes

<table>
<thead>
<tr>
<th>Clinical</th>
<th>Total (n = 218)</th>
<th>Alcoholic (n = 41)</th>
<th>Heavy (n = 42)</th>
<th>Moderate (n = 42)</th>
<th>Light/teetotal (n = 34)</th>
<th>Unknown (n = 59)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ophthalmoplegia</td>
<td>7 (3.2%)</td>
<td>2 (4.9%)</td>
<td>0 (0%)</td>
<td>3 (7.1%)</td>
<td>0 (0%)</td>
<td>2 (3.4%)</td>
</tr>
<tr>
<td>Nystagmus</td>
<td>13 (6.0%)</td>
<td>4 (9.8%)</td>
<td>3 (7.1%)</td>
<td>2 (4.8%)</td>
<td>0 (0%)</td>
<td>4 (6.8%)</td>
</tr>
<tr>
<td>Ataxia</td>
<td>4 (1.8%)</td>
<td>2 (4.9%)</td>
<td>0 (0%)</td>
<td>2 (4.8%)</td>
<td>0 (0%)</td>
<td>0 (0%)</td>
</tr>
<tr>
<td>Confusion</td>
<td>67 (30.7%)</td>
<td>13 (31.7%)</td>
<td>17 (40.5%)</td>
<td>11 (26.2%)</td>
<td>7 (20.6%)</td>
<td>19 (32.2%)</td>
</tr>
<tr>
<td>Confabulation</td>
<td>0 (0%)</td>
<td>0 (0%)</td>
<td>0 (0%)</td>
<td>0 (0%)</td>
<td>0 (0%)</td>
<td>0 (0%)</td>
</tr>
<tr>
<td>One symptom/sign</td>
<td>79 (36.2%)</td>
<td>17 (41.5%)</td>
<td>18 (42.8%)</td>
<td>16 (38.1%)</td>
<td>7 (20.6%)</td>
<td>21 (35.6%)</td>
</tr>
<tr>
<td>More than one</td>
<td>6 (2.8%)</td>
<td>2 (4.9%)</td>
<td>1 (2.4%)</td>
<td>1 (2.4%)</td>
<td>0 (0%)</td>
<td>2 (3.4%)</td>
</tr>
<tr>
<td>symptom/sign</td>
<td></td>
<td></td>
<td></td>
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</tr>
</tbody>
</table>
simple fractures and reports of diffuse axonal injury. Focal damage was present in 46.1% of the total but accounted for 58.5% of the injuries in the alcoholic group. These differences were not statistically significant.

Severity was assessed on the basis of the Glasgow Coma Scale (GCS), on admission. It is customary to classify injuries on the GCS of ≤8 as severe; 38 (17.8%) of the 218 injuries were graded as severe by this method. For each category of alcohol consumption the percentage of cases with GCS scores of ≤8 were: alcoholic 26.8%; heavy drinkers 9.5%; moderate drinkers 19.0%; light drinkers/teetotallers 5.9%; and unclassified 22.0%. These findings were not statistically significant (Kruskal–Wallis one-way ANOVA: \( \chi^2 = 6.9030, \) d.f. = 4, \( P = 0.1411 \)).

Clinical and laboratory findings

Several clinical signs are common to both WKS and TBI. The frequency of these signs being noted in the case records is summarized in Table 2 for each category of alcohol consumption. By the nature of the study, only positive reports of findings could be included and in many instances no comment was made as to the presence of normal or abnormal signs. There was no correlation between the frequency of any of the signs and the degree of alcohol consumption.

Blood glucose levels were recorded in 81 (37.2%) of the sample, liver function tests in 67 (30.7%) and haematology results in 107 (49.1%). The only parameter that showed any correlation with alcohol consumption category was mean cell volume (MCV). Five of the 30 subjects in the alcoholic group had an MCV > 98 fl and this represented a significant difference from the other groups (Kruskal–Wallis one-way ANOVA: \( \chi^2 = 8.0293, \) d.f. = 3, \( P = 0.0454 \)).

Treatment

A total of 97 patients (44.5%) received either 5% dextrose or 0.18% (w/v) normal saline/4% (w/v) dextrose as i.v. infusions during their admission. The amount infused during their hospital stay ranged from a single bag (500 ml), to 46 (23 1) with a median of five bags (2.5 l). Twenty-two (53.7%) of the alcoholic and 16 (38.1%) of the heavy drinker groups were given dextrose by infusion.

Total parenteral nutrition (TPN) was given to five patients, two alcoholic and three others. Only one patient had the full prescription detailing additives recorded, and this showed that Solivito N had been used as vitamin additive. Solivito N is a mixture of water soluble vitamins used to supplement i.v. nutrition with one vial recommended as meeting the daily requirements of an adult. One vial contains 3.1 mg of thiamine mononitrate (British National Formulary, 1996). In the other cases, there was no record of constituents of the TPN. Thirty patients received feeding via nasogastric or gastrostomy tubes. The alcoholic group had the highest number at 11 (26.2%), and the moderate the next at eight (19.0%). The majority of patients were fed using Osmolyte, a standard tube feed which contains added thiamine.

Thiamine was given to a total of 45 patients (20.6%). The breakdown of thiamine treatment according to alcohol group and route of administration is presented in Table 3. No patients

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**Table 3. Thiamine administration according to history of alcohol consumption**

<table>
<thead>
<tr>
<th></th>
<th>Total (n = 218)</th>
<th>Alcoholic (n = 41)</th>
<th>Heavy (n = 42)</th>
<th>Moderate (n = 42)</th>
<th>Light/teetotal (n = 34)</th>
<th>Unknown (n = 59)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Given thiamine</td>
<td>45(20.6%)</td>
<td>23(56.1%)</td>
<td>11(26.2%)</td>
<td>3(7.1%)</td>
<td>1(2.9%)</td>
<td>7(11.9%)</td>
</tr>
<tr>
<td>I.v. route only</td>
<td>4(1.8%)</td>
<td>3(7.3%)</td>
<td>0(0%)</td>
<td>0(0%)</td>
<td>1(1.7%)</td>
<td>0(0%)</td>
</tr>
<tr>
<td>Both i.v. and oral</td>
<td>7(3.2%)</td>
<td>4(9.8%)</td>
<td>1(2.3%)</td>
<td>0(0%)</td>
<td>1(2.9%)</td>
<td>1(1.7%)</td>
</tr>
<tr>
<td>Oral only</td>
<td>34(15.6%)</td>
<td>16(39.0%)</td>
<td>10(23.8%)</td>
<td>3(7.1%)</td>
<td>0(0%)</td>
<td>5(8.5%)</td>
</tr>
<tr>
<td>Discharge prescription</td>
<td>16(7.3%)</td>
<td>8(19.5%)</td>
<td>3(7.1%)</td>
<td>1(2.4%)</td>
<td>0(0%)</td>
<td>4(6.8%)</td>
</tr>
</tbody>
</table>
Table 4. Duration and dosage of thiamine given

<table>
<thead>
<tr>
<th>Dose and duration</th>
<th>All patients (n = 45)</th>
<th>I.v. (n = 11)</th>
<th>Oral (n = 34)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Duration of treatment (days)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Range</td>
<td>1–37</td>
<td>1–14</td>
<td>0–37</td>
</tr>
<tr>
<td>Mean</td>
<td>9.2</td>
<td>3.6</td>
<td>8.4</td>
</tr>
<tr>
<td>Median</td>
<td>5</td>
<td>2</td>
<td>5.5</td>
</tr>
<tr>
<td><strong>Daily dose (mg)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Range</td>
<td>25–750</td>
<td>250–750</td>
<td>25–300</td>
</tr>
<tr>
<td>Mean</td>
<td>205.1</td>
<td>295.5</td>
<td>190.6</td>
</tr>
<tr>
<td>Median</td>
<td>200</td>
<td>250</td>
<td>200</td>
</tr>
</tbody>
</table>

received thiamine by the i.m. route. The proportion of patients in each alcohol category who were treated with thiamine decreased with the decreasing alcohol consumption history, the differences between the categories being statistically significant (Kruskal-Wallis one-way ANOVA: $\chi^2 = 37.470$, d.f. = 3, $P < 0.0001$). However, no other factor in any of the historical, clinical or laboratory findings, dextrose infusion or parenteral or tube feeding showed statistically significant correlation with thiamine treatment.

Taking both i.v. and oral routes into account, the median duration of treatment (Table 4) was 5 days (mean 9.2; range 1–37 days). Table 4 also shows a summary of treatment and dosage for the different routes. Thiamine was continued after discharge in 16 patients, or 35.6% of those who had been treated as inpatients. There was no correlation between the doses or duration of treatment and alcohol history, amount of i.v. dextrose given, GCS level or any other clinical or laboratory parameter recorded (data not shown).

**DISCUSSION**

The impression given by the results of the questionnaire, that clinicians would treat most high-risk patients with thiamine, is not borne out by this retrospective study, with only 41% of those with a documented history of alcohol consumption >50 units per week receiving thiamine. The consultant neurosurgeons stated that they would give thiamine to TBI patients with a known history of alcoholism or clinical stigmata of chronic alcohol use. However, it is often the junior medical staff who are responsible for the initial management of these patients and yet no written protocol or routine instructions regarding thiamine prophylaxis are given to them. While the literature on coma of medical or unknown cause recommends the administration of thiamine (Bates, 1993), no reference is made to the subject in recent reviews of acute head injury management (Miller, 1993; Lang, 1994; Teasdale, 1995). Commonly no history of alcohol use is obtainable in the early hours or days after head injury and the clinical features of liver disease are often absent in thiamine-deficient alcohol abusers. The Committee on Safety of Medicines (1989) issued a warning of possible anaphylactic reaction to Parentrovite, which was subsequently withdrawn. Similar warnings apply to thiamine with the recommendation that parenteral treatment be restricted to those in whom it is considered essential (British National Formulary, 1996). This may have resulted in a change of practice in neurosurgical units where Parentrovite was previously given routinely in suspected alcohol abusers.

The present study population was identified on the basis of suspected recent alcohol consumption in patients admitted to hospital with TBI. The lack of routine alcometer or blood alcohol estimations makes it possible that some patients may have had recent alcohol intake missed. This, and the fact that the total numbers of TBI cases includes children, might explain the fact that the group studied represented 27% of the total admitted during the study period, which is lower than the proportion of people reported as having taken alcohol shortly before injury in other studies (Dikmen et al., 1995; Kelly, 1995). TBI is recognized as occurring about three times more commonly in men than in women and having peak occurrences in young adults, with a secondary
lesser peak in the elderly (Pentland et al., 1986). The great majority of the study population was male and a bimodal age distribution was present suggesting that it is fairly representative of the general head injury population, although the median age of 38.2 years is higher than in some series (Sparadeo and Gill, 1989; Dikmen et al., 1995).

The confounding factors of head injury, alcohol intoxication, a low level of consciousness, unreliable history and difficult examination make interpretation of signs and symptoms common to both WKS and head injury problematic. There was no correlation between alcohol history and these signs and symptoms, but this may have been affected by the large number of patients with unknown or unrecorded alcohol history. This group had the highest percentage of confused patients overall. The difficulties inherent in any retrospective study are particularly relevant here. The clinical emphasis in the early life-threatening stages following head injury is the detection of focal signs of intracranial complications and monitoring of vital signs relevant to secondary insults such as hypoxia, shock and metabolic disturbances. Routine thorough neurological examination for features such as ophthalmoplegia, nystagmus and ataxia is often difficult in these circumstances, particularly when conscious level is impaired or the patient is confused and uncooperative. A prospective study with careful examination for these features would be necessary to provide accurate incidence figures.

It must be noted that, if the premise is correct that thiamine deficiency could be the likely mechanism or a major contributing factor to most neurological syndromes associated with alcohol abuse (Lishman, 1990; Joyce, 1994), then WKS may only be the most manifest of disorders. TBI is more common in alcoholics, and indeed may go unrecognized in them, leading to significant neuropsychological deficits (Hillbom and Holm, 1986). Thiamine deficiency may well be playing a role in the cognitive impairments found in those injured who had a high alcohol intake previously.

Finally, even in those patients who did receive thiamine, treatment was not always adequate. Owing to minimal absorption of oral thiamine in alcohol abusers, most authorities conclude that oral treatment in patients suspected of being thiamine-deficient is not appropriate in the acute situation (Chataway and Hardman, 1995; Cook and Thomson, 1997). Where parenteral thiamine was given, the dose and duration were less than that generally recommended for effective therapy, i.e. 500 mg to 1500 mg for 3 to 5 days (Cook and Thomson, 1997). In the present study, only one patient received a thrice daily dose of 250 mg thiamine i.v., all the others receiving single daily doses of 250 mg. In about one third of cases, parenteral therapy was not followed with oral thiamine and almost two-thirds of those who were given thiamine in hospital did not receive discharge prescriptions, despite the recommendations that supplements should be continued for at least 3 months (Chataway and Hardman, 1995). Some patients were given Vitamin B Compound or Vitamin B Compound Strong, but as these preparations contain only 1 mg and 5 mg of thiamine respectively, such treatment falls far short of the recommended supplementation dose of at least 200 mg daily (Chataway and Hardman, 1995). If one also notes the additional glucose load given to just under half of the patients, the need for adequate thiamine dosage is clearer still.

Both parts of our study have shown that thiamine administration to TBI patients with a history of alcohol abuse appears somewhat arbitrary, and, even when given, it is probably given in inadequate amounts in the majority of cases. As head injury is one of the commonest causes of coma in alcoholics and conditions such as the WKS are likely to be more difficult to detect in the presence of traumatic brain damage, there is a need for more research of a prospective nature. It would appear useful to determine whether or not thiamine deficiency is common in the head-injured population and, if it is, whether this correlates with neuropsychiatric syndromes seen in alcoholics. It may also be worthwhile to study the incidence of preceding head injury in populations of patients with WKS. If such further work supports the hypothesis that thiamine deficiency plays a role in the brain damage in this group, it would constitute a potentially treatable additional insult.

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REFERENCES


