Recovery after subarachnoid haemorrhage

P McKenna, J R Willison, D Lowe, G Neil-Dwyer

Abstract

Objective—To determine the implications of subarachnoid haemorrhage for quality of life and aftercare.

Design—Prospective follow up study of patients surviving subarachnoid haemorrhage over one year (at discharge, three months, and one year) by examination of cognitive functions (a test battery) and changes in everyday life (semi-structured interview).

Setting—Regional neurosurgical unit at a tertiary referral centre.

Patients—100 Patients with subarachnoid haemorrhage; 17 were lost during the study because of ineligibility (further surgery, previous head injury, relevant psychiatric history, and cultural differences), loss of contact, and non-compliance; a further 13 patients who developed a neurological deficit were considered separately.

Main outcome measure—Performance on cognitive test battery and reported changes in quality of life.

Results—At discharge patients with and without neurological deficit scored below established norms with most tests, but by three months the difference had resolved in patients without deficit. Reduced quality of life attributable to subarachnoid haemorrhage at one year mainly included less energy (seven patients), adverse emotional changes (five), early retirement, affected social life, and domestic tension (three each). None reported reduced capacity for work.

Conclusions—Patients surviving subarachnoid haemorrhage without neurological symptoms have a good prognosis and should be encouraged to return to a normal lifestyle within about three months.

Introduction

Subarachnoid haemorrhage is relatively uncommon, with a mortality of one in three. Health care workers in primary care often have little experience of patients who have had a haemorrhage. Many papers have been published on subarachnoid haemorrhage, but virtually none is of use to those who are concerned in aftercare. Moreover, it may take many years for a representative sample of patients to pass through a general practice. The need for better information became clear to us while conducting a follow up study of 100 patients recovering from a subarachnoid haemorrhage. We found that when patients were discharged back to the care of their general practitioners many of the doctors were unable to comment on the minor problems their patients described and often referred patients to the neurosurgeon—a lengthy route for a few words of reassurance, which was sometimes the only treatment required.

Trying to learn about subarachnoid haemorrhage from current publications would only confuse the non-specialist. Though earlier studies tended to report excellent recovery rates, more recent researchers claim that the closer they looked the more they found in terms of impaired intellect and impoverished quality of life. In these investigations, based on retrospective group studies, the patient was often assessed many years after the event: thus the results have a limited application in treating patients in the first few months of convalescence.

We describe the implications for the aftercare of patients who have had a subarachnoid haemorrhage in a one year prospective follow up study of a representative sample of patients at the South East Thames regional neurosurgical unit, Brook Hospital, London.

Patients and methods

One hundred patients with a diagnosis of subarachnoid haemorrhage constituted the experimental group. Each patient was assessed three times: at initial discharge from hospital, at three months, and at one year. The assessment consisted of a two hour examination of cognitive functions using a test battery derived from the psychology department of the National Hospital for Nervous Diseases, London. A semi-structured interview with a close friend or relative of the patient (often a spouse) was carried out at each assessment, and questionnaires on mood and behaviour were also used. The most valuable source of information was found to be the semi-structured interview, which could be tailored to suit the idiosyncrasies of each patient. The contribution of the illness to the overall quality of life (work and social and domestic life) was judged from these data.

Results

Of the 100 patients with subarachnoid haemorrhage who entered the study, 17 were lost during their first year through ineligibility (because of the need for further surgery, a previous head injury, relevant...
distributions examination. The psychiatric history, and cultural differences), loss of contact, and lack of cooperation. Of the remaining 83, 13 developed neurological deficits at some early stage of the illness, which were observable on standard clinical examination. Table I shows the age and sex distributions and the clinical details of the patients. At

<table>
<thead>
<tr>
<th>Age (years):</th>
<th>Patients without neurological deficit</th>
<th>Patients with neurological deficit</th>
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<tbody>
<tr>
<td>21-30</td>
<td>10</td>
<td>P</td>
</tr>
<tr>
<td>31-40</td>
<td>30</td>
<td>31</td>
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<td>41-50</td>
<td>54</td>
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<td>51-60</td>
<td>15</td>
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<tr>
<td>61-66</td>
<td>12</td>
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Initial discharge both groups scored significantly poorer than established norms on most tests. For the 70 patients with no neurological deficit this difference had mostly disappeared by three months, and by one year their scores were virtually indistinguishable from the established norms. Unlike the patients with no deficit, the group with a neurological deficit obtained worse scores throughout and remained below their expected optimum. The detailed results are available from the authors.

Table II shows the reduced quality of life for the group with no neurological deficit and the degree to which the illness could be considered to have contributed to a reduced quality of life after other relevant variables had been taken into account. This shows the relatively small effect of the haemorrhage in reducing the quality of life of these patients.

<table>
<thead>
<tr>
<th>Clinical features of</th>
<th>Patients without neurological deficit</th>
<th>Patients with neurological deficit</th>
</tr>
</thead>
<tbody>
<tr>
<td>Subarachnoid haemorrhage:</td>
<td>4 1</td>
<td>4 1</td>
</tr>
<tr>
<td>Unusual</td>
<td>12</td>
<td>12</td>
</tr>
<tr>
<td>Anocerebral malfunction</td>
<td>7</td>
<td>7</td>
</tr>
<tr>
<td>Anterior communicating</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>Left middle cerebral</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Left internal carotid</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Right middle cerebral</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Right internal carotid</td>
<td>12</td>
<td>2</td>
</tr>
<tr>
<td>Vertebrobasilar</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Others</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Operations</td>
<td>60</td>
<td>12</td>
</tr>
</tbody>
</table>

Discussion

We do not intend to discuss details of the 13 patients who survived a subarachnoid haemorrhage but developed a neurological deficit. They formed a heterogeneous group with diverse cognitive deficits of varying degrees of severity and were similar to any group of patients suffering a cerebral infarction. These patients are best thought of as exceptions to the general rule of recovery after a subarachnoid haemorrhage. The 70 patients with no neurological deficit formed a homogeneous group whose pattern of recovery was distinct.

At the initial assessment of these 70 patients, within a few weeks of the onset of illness, the results of cognitive function testing were appreciably different from the norms. The degree of underfunctioning was mild, but most patients gave the clinical impression of being confused to some extent even if this was apparent only on harder tests. Many patients could not withstand the one and a half to two hours of testing in one session and were seen over one or two days. By three months nearly all patients were functioning at or near their premorbid level as judged by standard psychometric criteria. A few patients showed minimal signs of underfunctioning, though this was rarely of any consequence to their overall cognitive state. Most patients, however, felt that they were back to normal, apart from tiring more easily than usual.

By one year no evidence of cognitive deficit could be found on mental testing for the 70 patients with no neurological deficit. Many reported that they had problems related to their quality of life, but these were rarely attributable to the central effects of their illness.

PRACTICAL CONSIDERATIONS FOR EVERYDAY LIFE

At discharge there was usually some residual confusion even if this was not immediately apparent to onlookers. Because of this patients thought that they had never been told what had happened or they had a distorted picture of events during their stay in hospital. Recall of past events was faulty because these were often not properly registered in the first place.

Relatives were often unable to understand fully the underlying mechanism of the illness or the surgical procedure as they were still in shock when they were given this information. This information cannot be repeated often enough to both relatives and patients in the early stages of recovery.

During the first few weeks at home patients often complained that they could not concentrate well. They could not enjoy conversation, reading, or watching television. Fatigue was debilitating, and the need for sleep and rest continued for many weeks or months for some.

The emotional effects of experiencing a subarachnoid haemorrhage usually occurred after the patients were at home when they began to realise the seriousness and implications of their illness. The biggest fear was of a recurrence, and many were afraid initially of being left alone and later of going out alone. Though reassurance helped, much of the fear seemed to stem from an awareness of their own mortality and vulnerability. One patient expressed this succinctly: “Never mind, it shouldn’t happen again; it shouldn’t have happened in the first place…” The threshold of perceived risk was lowered. With reassurance and encouragement this fear receded in time, and normal activities, including sex and sports, were then resumed.

One of the most alarming but common symptoms reported was headache. Strange and severe headache patterns and sensations were common in our patients and caused alarm. Again, telling patients that this is common and usually unimportant sequel of a subarachnoid haemorrhage was often sufficient to reassure them. Most employed people had returned to work three months after their illness, initially part time, with most working full time within weeks. Many of our patients took the option of early retirement, and a few decided to leave an occupation in which they had long been unhappy or over stretched. Clearly, for most people returning to work at the earliest possible moment was one of the most therapeutic steps in their recovery. There was little benefit in prolonging their convalescence beyond three months.

At the end of one year the only typical residual symptom was tiredness, though this did not affect the patients’ lifestyle. By this time most patients considered that the experience of the subarachnoid haemorrhage was behind them. There were a few dramatic exceptions to this generally favourable pattern of recovery in
the patients with no neurological deficit. One patient who showed a personality change was found to have a small left thalamic infarct. Another who became severely depressed was considered to have lived with severe domestic stress for many years. A third patient became debilitated with various illnesses and died two years later following a myocardial infarction. At necropsy widespread atheroma was found. Other patients deviated from the general pattern of emotional well being in proportion to the degree of stress they were experiencing in other aspects of their lives. Such factors included divorce, bankruptcy, and death in the family.

Conclusion

Our experience of the survivors of subarachnoid haemorrhage suggests that a positive approach to prognosis is warranted if a neurological deficit has not occurred. One year after the haemorrhage no cognitive deficits were evident in our patients and residual symptoms seemed to be minor. The typical pattern in a straightforward case was a gentle convalescence and then the patient returning to a normal lifestyle by three months. In treating and managing survivors of subarachnoid haemorrhage normality should be encouraged.

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Energy expenditure in children with type I diabetes: evidence for increased thermogenesis

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Abstract

The aim of the study was to assess whether increased energy expenditure causes the negative energy balance (tissue catabolism) commonly seen in children with insulin dependent (type I) diabetes. Resting metabolic rate and thermogenesis induced by adrenaline were measured in five healthy children and 14 children with type I diabetes who were all free of clinical signs of late complications of diabetes mellitus but differed in their degree of glycaemic control (in eight-glycated haemoglobin concentration <10% and in the six others >10%). When compared with the control subjects children with type I diabetes had normal resting metabolic rates but their urinary nitrogen excretion was significantly raised (11.5 (SD 5.4) mg/min in those with glycated haemoglobin concentration <10%, 11.6 (5.2) mg/min in those with concentration >10% v 5.4 (3.0) mg/min in control subjects). During the infusion of adrenaline the diabetic children showed a threefold and sustained increase in thermogenesis and disproportionate increases in the work done by the heart, in lipid oxidation rate, and in plasma concentrations of glucose, free fatty acids, and ketone bodies. The increased thermogenic effect of adrenaline did not correlate with the degree of glycaemic control.

Increased thermogenesis may explain the tissue wasting commonly seen in children with type I diabetes during intermittent stress.

Introduction

Alterations in energy expenditure contribute to energy balance and thus to changes in body weight. Patients with type II diabetes that is resistant to insulin are commonly overweight and show a blunted thermogenic response to a meal and reduced stimulation of energy expenditure by the sympathetic nervous system. Whether defective thermogenesis is primary or secondary in type II diabetes is not known, but limited energy expenditure predisposes to a positive energy balance, weight gain, and thus obesity. When compared with patients with type II diabetes mellitus many patients with insulin dependent (type I) diabetes are also resistant to insulin and have a normal or commonly reduced body weight. Little is known, however, about their daily energy expenditure. Resting metabolic rate was reported to be increased in type I diabetic patients during severe hyperglycaemia and ketoacidosis and to return to normal values within three hours after insulin replacement. This finding would partly explain tissue wasting in patients with poorly controlled type I diabetes and also their weight gain after adequate insulin replacement. In addition, in patients with poorly controlled type I diabetes the thermogenic effect of a meal was abnormal and the response to infused noradrenaline reduced. This defect was not improved by continuous infusion of insulin subcutaneously despite optimal glycaemic control. The thermogenic effect of noradrenaline was, however, normal in three patients given biguanides with insulin, which suggests that insulin resistance causes defective thermogenesis in both type I and type II diabetes. By contrast, the responses of several energy consuming processes—for example, the work of the heart and hepatic gluconeogenesis—to β adrenocorter agonists were reported to be enhanced in adult patients with type I diabetes independently of their glycaemic control.

The fact that increased β adrenergic sensitivity predisposes to a negative energy balance partly explains the differences in body weight between type I and type II diabetic patients. To investigate this hypothesis we selected a group of 14 children of normal weight with type I diabetes who differed in the degree of their glycaemic control (as reflected in their glycated haemoglobin concentrations) but had no experience of

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