Subarachnoid Hemorrhage: Epidemiology, Diagnosis, Management, and Outcome

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SUMMARY A population-based study of primary subarachnoid hemorrhage in Auckland (population 829,454), New Zealand, identified 180 cases in a two-year period. This represented an age adjusted incidence rate of 10.5 and 18.3 per 100,000 for men and women respectively. Sixty-eight percent of all cases had a proven intracranial aneurysm or arteriovenous malformation, 15% had negative angiographic findings and in the remaining 17%, the presence or absence of a localized lesion was unknown since neither angiography nor autopsy were performed. Twenty-six patients (15%) died before hospitalization and a further 36 patients (20%) died within 48 hours of onset. Only 94 patients (53% of all patients registered) were fit enough to undergo angiography. A surgical operation was carried out on 60 of the 68 patients in whom an aneurysm was confirmed at angiography. The overall case fatality rate was 36% within the first 48 hours, 43% in the first week and 57% at both six months and one year. The high early case fatality rates are similar to those found in previous population-based studies, suggesting that despite the major advances to individual patients from technological advances, the potential contribution of hospital management to the reduction of subarachnoid haemorrhage mortality rates is likely to be limited.

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AN ANALYSIS OF NATIONAL SUBARACHNOID HEMORRHAGE DEATH RATES in New Zealand revealed a decline during the last decade although no such trend has been noted in other studies. The decline in mortality could be due to a decrease in incidence or to an improvement in case fatality due to better management. Subarachnoid hemorrhage is a relatively rare form of cerebrovascular disease, comprising only 6.8% of all stroke events in New Zealand. In contrast with other stroke subcategories, however, subarachnoid hemorrhage affects younger people, women more than men, and has a higher early case fatality rate. Many patients die before admission to hospital, and studies based only on survivors give a false account of incidence and of the effect of treatment on the true mortality. This paper provides information from a large population-based study on the natural history of subarachnoid hemorrhage, the pattern and use of diagnostic procedures and hospital resources, and the role of surgery in the management of subarachnoid hemorrhage patients in the Auckland region.

Methods

All cases of primary subarachnoid hemorrhage in residents of the Auckland region during the year beginning 1 March 1981 were identified as part of a larger study of myocardial infarction, sudden death and cerebrovascular disease. In addition, cases occurring in the year beginning 1 January 1983 were identified. The study population, 829,454 persons (1981 census), represents one-quarter of New Zealand’s total population. Full details of the diagnostic criteria and methods used are reported elsewhere. Briefly, subarachnoid hemorrhage was defined as a spontaneous rupture of a blood vessel, most often a cerebral aneurysm or arteriovenous malformation (AVM) leading to bleeding into the subarachnoid space. Diagnosis was based on the abrupt onset of a severe headache and/or impaired consciousness or focal neurological symptoms associated with at least one of the following clinical signs: lumbar puncture (LP) findings of uniform blood staining and xanthochromia of the cerebrospinal fluid (CSF); computerised axial tomography (CT scan) evidence of blood in the subarachnoid space; cerebral angiographic identification of an aneurysm or AVM at surgery or autopsy. The definition excluded primary intracerebral hemorrhage with secondary rupture into the subarachnoid space, and bleeding due to trauma, neoplasma or infection.

The main sources of case-finding for subarachnoid hemorrhage patients included daily searches of hospital admission lists and visits to the relevant wards of the three main hospitals in Auckland, together with a systematic search of hospital discharges, post mortem reports, and death certificates. Comprehensive checks were made during the study to ensure that all cases were found. These checks included referrals from a 50% representative sample of Auckland general practitioners of all patients in their practices who had experienced a subarachnoid hemorrhage, and a request to the medical records officers of major hospitals in New Zealand to identify any Auckland residents who had suffered a subarachnoid hemorrhage while out of the area. These checks did not reveal any cases which had not already been identified.

Information about the current episode, past medical history, as well as socio-demographic variables were obtained by a trained nurse interviewer (S.T.) using a standard questionnaire. The interview was conducted with the patient when possible, otherwise with a close family member. Details of the patterns of management and diagnostic investigations were obtained from medical records. For those patients identified during the 1983 study year, grading was assigned according to the Botterell classification at 12 hours after onset.
A follow-up interview was conducted six months following the onset for all those identified in the 1981–2 study year and a telephone follow-up at one year established dead or alive status for all patients.

### Statistical Methods

The denominators for calculating the age specific rates were the mean annual populations for the Auckland statistical region in the years 1981 and 1983 for the appropriate age and sex groups. The age adjusted rates were calculated by the direct method using the total 1981 (census year) Auckland population. The Mantel-Haenszel test was used to compare rates.8

### Results

#### Incidence

One hundred and eighty new episodes (in 65 men and 115 women) of primary subarachnoid hemorrhage were identified during the 2-year study period. Four patients had a previous history of subarachnoid hemorrhage and have been omitted from the calculation of incidence rates (table 1).

The age specific incidence rates rose from 2.3 per 100,000 at the youngest age group (15–24 years) and peaked at 30 per 100,000 among the 65–74 age group. With the exception of the youngest age groups (15–34 years), rates for women were markedly higher than those for men. The age adjusted rates for women (18.3 per 100,000) were 43% higher than for men (10.5 per 100,000 population). This difference was statistically significant (MH \( \chi^2 \) 9.4, OR 1.7, 95% confidence interval 1.2, 2.2, \( p < .005 \)). The median age for women, 56 years, was 13 years later than that for men despite the fact that 52% of men were under the age of 45 years compared to 27% of women in this age range (\( p < .05 \)).

#### Management

Twenty-six people died before medical attention or hospitalisation could be arranged. Of the 154 patients who were hospitalised, 136 (88%) were admitted without delay to hospital. Of the remaining 18, the first diagnostic investigation was carried out on 94 patients (53% of those hospitalised) and with 13 patients there was an initial misdiagnosis: 5 delayed seeking medical help, and with 13 patients there was an initial misdiagnosis: 10 were treated for headache and/or influenza, and 3 who were sent to hospital for diagnosis following the sudden onset of a severe headache were not admitted. Further bleeds in these 3 patients occurred five to six weeks later, and after the nature of the headache was recognised, admission followed.

Table 2 summarizes selected investigations — LP, CT scan, and angiography — for the 154 hospitalised patients, and indicates the proportion of each of these investigations which confirmed the diagnosis. Using CT scan as a reference, LP correctly identified 83% of true positives in this study. LP was more often used as the first diagnostic investigation; of 55 patients who had both a CT scan and LP, 35 (64%) had LP first; information on the sequence of procedure was not available for 3 cases. No LP was performed before admission to hospital.

Four-fifths of all patients admitted to hospital underwent at least two of the investigations and one-third all three. Of the 38 patients who died within the first 48 hours, 16 had a LP, 7 had a CT scan and 2 had surgery preceded by angiography. Autopsy was performed on 78% of deaths which occurred within 48 hours; the total autopsy rate was 47%.

The outcome of various subsets of patients within the first year, including the proportion of patients in whom a ruptured aneurysm was identified as the source of bleeding, is described in figure 1. One hundred and eighteen patients with intracranial aneurysms and 5 with AVM’s were identified (68% of all cases). The three most common sites were the anterior communicating artery (34%), the middle cerebral artery (28%), and the internal carotid and posterior communicating artery (22%). There were negative angiographic findings for 27 patients (15%), and in the remaining 30 patients (17%), neither angiography nor autopsy were undertaken.

Angiography, which is used to identify a surgically treatable lesion, was carried out on 94 patients (53% of all cases). Of the 68 patients who were identified as having an aneurysm, surgical treatment was undertaken in 60 patients (22 men and 38 women). The median length of time from hospitalisation until surgery was 8 days. Ten of the 60 patients who received surgery died within two weeks and in a further 10 patients, there were complications following the surgical procedure.

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**Table 1** Average Annual Incidence Rates for Subarachnoid Hemorrhage, Auckland, New Zealand, by Sex

<table>
<thead>
<tr>
<th>Age group</th>
<th>Men</th>
<th>Women</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>15–24</td>
<td>3.9 (6)</td>
<td>0.7 (1)</td>
<td>2.3 (7)</td>
</tr>
<tr>
<td>25–34</td>
<td>8.8 (11)</td>
<td>5.3 (7)</td>
<td>7.0 (18)</td>
</tr>
<tr>
<td>35–44</td>
<td>16.1 (16)</td>
<td>21.5 (22)</td>
<td>18.9 (38)</td>
</tr>
<tr>
<td>45–54</td>
<td>19.7 (16)</td>
<td>28.1 (22)</td>
<td>23.8 (38)</td>
</tr>
<tr>
<td>55–64</td>
<td>13.0 (9)</td>
<td>35.0 (26)</td>
<td>24.4 (35)</td>
</tr>
<tr>
<td>65–74</td>
<td>8.6 (4)</td>
<td>46.3 (27)</td>
<td>29.6 (31)</td>
</tr>
<tr>
<td>75+</td>
<td>4.7 (1)</td>
<td>41.2 (8)</td>
<td>14.9 (9)</td>
</tr>
</tbody>
</table>

**Crude rate** 10.6 (63) 17.8 (113) 14.3 (176)

**Age adjusted rate** 10.5 18.3

*Per 100,000 population.
†Number of cases in parentheses.
‡Age adjusted to total 1981 census population.

**Table 2** Selected Investigations and Surgical Procedure for 154 Hospitalised Patients (number and percent)

<table>
<thead>
<tr>
<th>Investigation/procedure received</th>
<th>n (%), yield of positive diagnoses as % of total number of tests (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lumbar puncture</td>
<td>96 (62), 72</td>
</tr>
<tr>
<td>CT scan</td>
<td>94 (61), 83</td>
</tr>
<tr>
<td>Angiography</td>
<td>94 (61), 74</td>
</tr>
<tr>
<td>Surgery</td>
<td>60 (39), —</td>
</tr>
</tbody>
</table>
Details of the Botterell Classification recorded for the patients in 1983 who underwent surgery, indicate that of the 4 post surgical deaths in that year, 3 were grade 5 patients and the fourth was grade 3. The eight patients who did not receive surgery either died before surgery could be undertaken, or had inaccessible aneurysms. None survived two weeks. The subgroup with negative angiographic findings had a good prognosis since all but one patient survived at least one year.

Case Fatality
The overall case fatality rate was 43% at one week, 54% at one month and 57% at six months; the hospital case fatality rate was 47%. Six month case fatality for those with Botterell grades 1–2 was 27%, in comparison to 83% for those with grade 4, and 93% for those with a grade 5 classification. The case fatality rate of those with an aneurysm or AVM was 58% compared to only 19% for those in whom no aneurysm could be detected. In the group of patients for whom no investigation of the cause was undertaken, the case fatality was 87% (table 3).

There were no known additional deaths between six months and one year although two patients were lost to follow-up at one year—one had gone overseas and the other had changed address within Auckland but could not be traced.

Discussion
This large population-based study has investigated the management of all patients who had a spontaneous subarachnoid hemorrhage in the Auckland region in a two year period, irrespective of whether they were hospitalised. It thus provides an epidemiological overview of the clinical course and pattern of management of subarachnoid hemorrhage patients. The incidence rates in table 1 are more stable than those presented earlier which were based on only one year of data collection.

The proportion of patients who died before receiving medical attention (15%) is higher than that reported in recent studies but similar to a large population-based study carried out in the 1950’s. A further 21% died within 48 hours of hospitalisation suggesting that current hospital-based management strategies are unlikely to be successful for more than one third of all patients. Studies based on hospital cases alone underestimate the impact of this disease from the total community perspective.

The importance of recognising the signs of subarachnoid hemorrhage have been stressed in other studies. Cases can range from catastrophic headache and coma to trivial or misleading symptoms such as general headache, face, eye, neck and back ache and even fever and diarrhoea. Delay in seeking treatment by five patients, and the initial misdiagnosis in a further 13 patients in this study, indicates that there may be potential for improvement in terms of accurate early clinical diagnosis.

The number of diagnostic investigations received by patients admitted to hospital is lower than that reported in other series. In a recent Hobart study,14 because of the mortality and morbidity associated with LP in 9% of cases, it was concluded that LP should be omitted if it is clinically obvious that subarachnoid hemorrhage has occurred and CT scanning has demonstrated a hemorrhage. In the Auckland study there were 12 instances where a CT scan did not reveal subarachnoid bleeding although LP demonstrated that subarachnoid hemorrhage had occurred. Lumbar puncture was said to be positive for subarachnoid hemorrhage if the red blood cell count was > 2000 cu mm and the CSF was xanthochromic, therefore it is unlikely that false positive LP results were a problem in this study. These results suggest that LP still has an important role in the clinical sequence for diagnosis of subarachnoid hemorrhage.

Only 61% of all hospitalised patients were considered fit enough to undergo angiography, the necessary prelude to surgery; surgical management was performed in less than two-thirds (64%) of these patients. The likelihood of surviving subarachnoid hemorrhage with angiographic evidence of an aneurysm as the source of bleeding was only 32%; as far as could be determined, no patient with an aneurysm survived except for those who had been treated surgically.

The question of rebleeding and the timing of surgery remain unresolved although an important recent finding from the Cooperative Aneurysm study15 confirms that the peak rebleeding occurs the same day as the initial hemorrhage. This raises a dilemma, however,

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Survived (n)</th>
<th>Died (n)</th>
<th>Six months case fatality (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aneurysm</td>
<td>50</td>
<td>68</td>
<td>(58)</td>
</tr>
<tr>
<td>AVM</td>
<td>2</td>
<td>3</td>
<td>(60)</td>
</tr>
<tr>
<td>No aneurysm</td>
<td>22</td>
<td>5</td>
<td>(19)</td>
</tr>
<tr>
<td>Unknown source of bleeding</td>
<td>4</td>
<td>26</td>
<td>(87)</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>78</strong></td>
<td><strong>102</strong></td>
<td><strong>(57)</strong></td>
</tr>
</tbody>
</table>
because in most series, mortality associated with early operation, especially when the Botterell score is high, has been excessive. The surgical mortality rate in this study appeared to be high in comparison with other studies, but the proportion receiving surgical treatment was also greater than that reported elsewhere. Because so many deaths occur before surgery can be contemplated, surgical management, while clearly of great benefit to selected patients, is unlikely to have a major impact on overall case fatality rates. It is also unlikely that surgery is responsible for the observed decline in the New Zealand national death rate through prevention of later recurrent fatal hemorrhage in patients who survive the first bleed. In this series only 4 patients had a past history of subarachnoid hemorrhage; 2 of these had previously had surgery for a ruptured aneurysm.

The high early case fatality rate and low late mortality of subarachnoid hemorrhage has been noted elsewhere. Those who survived the first month, especially those without an aneurysm demonstrated, have a good prognosis for survival. Overall, however, only half of those who survived six months (23% of all cases registered) were free of disability and had returned to their previous lifestyle. This rather poor outcome is similar to other comparable studies. A falsely optimistic picture of the outcome following a subarachnoid hemorrhage can be gathered from studies which follow only those patients who receive surgery. The proliferation of increasingly sophisticated technology and pharmacological management in the neurological and neurosurgical arenas has allowed more precise diagnosis and treatment. Despite this, however, it has not been possible to demonstrate clear improvements in overall case fatality rates as a result. The one year case fatality rate in this study of the 1980’s is higher than that found in the large population-based study of primary subarachnoid hemorrhage in Helsinki in the 1950s.

The finding that the majority of deaths occur in the first 48 hours after the onset of symptoms, suggests that new management strategies for minimising rebleeding within the first few hours of the initial hemorrhage are required in order to alter effectively the clinical course of this disease. In particular, these results highlight the need for the identification of modifiable risk factors in the hope of preventing the occurrence of subarachnoid hemorrhage.

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