The Incidence of Stroke in The Kuopio Area of East Finland

JUHANI Sivenius, M.D.,* OLLI P. Heinonen, M.D.,† KALEVI Pyörälä, M.D.,‡ JUKKA Salonen, M.D.,§ and PAAVO Riekkinen, M.D.*

IN THE COLLABORATIVE STUDY coordinated by WHO from 1971 to 1974 the incidence of stroke was investigated in 17 populations.¹ The annual age and sex adjusted incidence rates for first stroke varied from 0.24 to 2.87/1000 persons. The lowest rate was observed in Sri Lanka and the highest rate in Japan. Two Finnish populations were included in this study. The population of North Karelia county had an annual incidence rate of 1.71/1000 persons and it was fourth highest among the WHO study populations.

The purpose of the present study was to determine the incidence and prognosis of stroke in four communities of the Kuopio county in East Finland.

Study Population and Methods

The Department of Neurology at the University Hospital of Kuopio started a Stroke Register for the Kuopio area on October 1st, 1978. The register functioned for twenty months, up to May 31, 1980. The study area consisted of one town, Kuopio, and three rural communities (fig. 1). The study was based on the population of this area. The distribution of the total population was: Kuopio 73,733 and the rural communities 21,687. Thus the majority of the study population was: Kuopio 10, Finland.

The Kuopio Area of East Finland

From the Department of Neurology, University Hospital of Kuopio,* National Public Health Institute,† the Department of Medicine, University Hospital of Kuopio, and the Research Institute of Public Health, University of Kuopio, Finnland.§ Address correspondence to Juhani Sivenius, M.D., Department of Neurology, University Hospital of Kuopio, P.O.B. 138, SF-70101 Kuopio 10, Finland.

Received May 25, 1984; revision #1 accepted August 31, 1984.

References

INCIDENCE OF STROKE IN KUOPIO, FINLAND/Sivenius et al

Definition of Stroke and Diagnostic Categories

Stroke was defined as rapidly developing clinical signs of a focal or global disturbance in cerebral function, lasting longer than 24 hours or leading to death, with no apparent origin other than a vascular source. Included were: subarachnoid haemorrhage (SAH, ICD 430), intracerebral haemorrhage (ICH, ICD 431), ischaemic brain infarction — both embolic and nonembolic (INF, ICD 432–434) — and unspecified acute CVD (NOS, ICD 436). TIA (ICD 435) was excluded by the definition.

Diagnostic Criteria

SAH: Angiographic identification of an aneurysm or arteriovenous malformation as the source of haemorrhage or demonstration at autopsy of recent bleeding or demonstration at autopsy of a saccular aneurysm or arteriovenous malformation. When angiograms were not performed, the clinical diagnosis of SAH was made by applying the criteria of Pakarinen.2

ICH: The minimum criterion for an intracerebral haemorrhage was bloody cerebrospinal fluid (CSF), demonstration of an intracerebral hematoma by computed tomography or an avascular mass effect in cerebral angiography (without evidence of aneurysm or arteriovenous malformation), or autopsy evidence.

Occlusion of precerebral arteries: Of the non-haemorrhagic strokes, those with angiographic or autopsy evidence of neck artery occlusion.

Brain infarction: Of the cases without CSF examination or autopsy, those with a history of TIA and without disturbance of consciousness.

Embolic brain infarction: Accepted as embolic were those cases of infarction which featured an abrupt onset and had an identified source of embolism, such as a recurrent myocardial infarction or atrial fibrillation.

Unspecified stroke: Cases with insufficient data.

Embolic brain infarction and occlusion of the precerebral arteries are here combined under the diagnostic category of brain infarction.

Incidence Rates

The number of persons falling ill with stroke was expressed as an annual rate per 100,000 of the total population. Persons with previous stroke who experienced a new stroke during the study period were included in the incidence figures. However, if a registered patient had a recurrence during the first study year, this incidence was not included. Thus the incidence figures refer to the number of persons experiencing a stroke in a year. Age and sex adjustment of the rates was made by a direct method using the 1979 mean population of Finland as a standard.

Results

Incidence of Stroke

During the 20 month study period 373 people suffered a stroke, an annual incidence of 235/100,000 for the entire population. There was no difference in the incidence rates between men and women (table 1). The incidence of stroke as a whole increased steeply with age.
age in both sexes. The mean ages of female and male patients were 70.8 ± 13.0 and 63.3 ± 13.5 years, respectively.

Age and sex adjusted total incidence was 270/100,000/year (table 2). Adjusted incidence rates for different types of stroke were (per 100,000 persons per year): SAH 18, ICH 23, cerebral infarction 217 and NOS 9.

Concerning only the first stroke the crude annual incidence was 189/100,000 persons (table 3).

The diagnostic distribution (table 4) for the first and the recurrent strokes (total cases) was: 300 patients (80%) had cerebral infarction, of whom 199 (53%) had cerebral thrombosis, 87 (23%) embolism and 12 (3%) occlusion of precerebral arteries. Two patients had thrombosis in an intracranial artery aneurysm with secondary embolism. ICH was diagnosed in 33 (9%) and SAH in 28 (8%) of the patients.

Fifty one of the patients submitted to thorough examination (337) had a brain stem lesion.

Case-Fatality

One year after the stroke, the cumulative fatality rate was 37% (fig. 2); within the first three months it was 26%. Among the patients with SAH all deaths occurred during the first three weeks. In the infarct group, the fatality increased throughout the study period but remained the lowest. Recurrent stroke occurred in 23 out of 373 patients (6%) during the first year after the stroke.

Discussion

Validity of Methods

The study area was chosen to insure that its population would receive care in only one hospital, the Central University Hospital of Kuopio.

Hospitalized patients were detected with the highest probability in every case. Every morning the diagnoses of patients seen in the emergency room were checked. The neurologist on duty had a protocol for all stroke cases. The physicians of the departments of medicine were informed to make contact when a stroke case was found among patients treated for other reasons. In addition a computer list was generated for all patients discharged from the hospital with diagnoses of stroke or TIA.

For fatal cases the coverage of the registration must be close to 100%. Besides the careful screening of hospitalized patients all death certificates of the study population were regularly examined in the study period and three months thereafter.

It may be more difficult to prove that all mild cases were detected. The health care organisation in Finland is centralized so that nearly all of the open care is given by municipal physicians. They were personally contacted and asked to notify the register of all new strokes not referred as emergency patients to the hospital. In

The following table summarizes the distribution of patients according to diagnosis and sex:

<table>
<thead>
<tr>
<th>Type of stroke</th>
<th>Male</th>
<th>Female</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>All No (%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>INF</td>
<td>142</td>
<td>158</td>
<td>300</td>
</tr>
<tr>
<td>ICH</td>
<td>13</td>
<td>20</td>
<td>33</td>
</tr>
<tr>
<td>SAH</td>
<td>16</td>
<td>12</td>
<td>28</td>
</tr>
<tr>
<td>NOS</td>
<td>6</td>
<td>6</td>
<td>12</td>
</tr>
<tr>
<td>Total</td>
<td>177</td>
<td>196</td>
<td>373</td>
</tr>
</tbody>
</table>

The following table shows the annual incidence per 100,000 population of stroke in selected communities, age groups 35-74 years:

<table>
<thead>
<tr>
<th>Community</th>
<th>Study years</th>
<th>35-44</th>
<th>45-54</th>
<th>55-64</th>
<th>65-74</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rochester</td>
<td>1955-69</td>
<td>35</td>
<td>110</td>
<td>364</td>
<td>791</td>
</tr>
<tr>
<td>Frederiksberg</td>
<td>1971-73</td>
<td>—</td>
<td>300</td>
<td>640</td>
<td></td>
</tr>
<tr>
<td>Akita</td>
<td>1971-74</td>
<td>504</td>
<td>967</td>
<td>2693</td>
<td></td>
</tr>
<tr>
<td>Espoo-Kauniainen</td>
<td>1972-73</td>
<td>59</td>
<td>173</td>
<td>358</td>
<td>1061</td>
</tr>
<tr>
<td>National Survey U.S.</td>
<td>1975-76</td>
<td>31</td>
<td>106</td>
<td>262</td>
<td>582</td>
</tr>
<tr>
<td>Tilburg</td>
<td>1978-79</td>
<td>—</td>
<td>302</td>
<td>868</td>
<td></td>
</tr>
<tr>
<td>Melbourne</td>
<td>1978-79</td>
<td>24</td>
<td>123</td>
<td>342</td>
<td>1028</td>
</tr>
<tr>
<td>Kuopio Area</td>
<td>1978-80</td>
<td>60</td>
<td>238</td>
<td>494</td>
<td>1107</td>
</tr>
</tbody>
</table>

*Incidence rates are based on first attacks of stroke.
†Incidence rates include patients with previous stroke.
discussions with local physicians it was frequently stated that they very rarely treated stroke patients, even mild cases, at home. Most cases that were not admitted directly to the hospital were admitted for diagnostic purposes to the neurology outpatient unit.

At autopsy (which was performed in 58% of fatal cases) only one clinical diagnosis appeared to be erroneous (a cerebral astrocytoma in an elderly male). Diagnostic procedures were frequently utilized (examined by a neurologist in 91%, lumbar puncture in 69%, brain scan in 61%, EEG in 51%, angiography in 12% and CT in 8% of patients).

Incidence
The adjusted annual incidence of stroke was 270 per 100,000. This incidence is high compared to international studies,1 where the other two Finnish studies are represented. Comparison of the total incidence with findings in other studies is not possible, but where age grouping fits with the present series, the age-specific incidence rates are comparable with those in several recent series.1, 3-6 Our findings suggest a clearly lower incidence rate than in the Japanese study, but otherwise the figures are higher than in the other series (table 5). The difference is most marked in the youngest age groups.

Besides comparing of age-specific incidences previous Finnish stroke studies,5, 7 are suitable for comparison of the total incidence (table 6). The adjusted incidence of stroke was higher in the Kuopio area for both females and males than elsewhere in Finland; the difference was not statistically significant.

In Finland and many other countries the mortality from cerebrovascular diseases has been decreasing. From 1967 to 1976 it declined in all age groups in both sexes, but the change was most marked in the oldest groups.10 The same trend occurred in the county of Kuopio (Valkonen, unpublished results, 1983).

It has been reported also that the incidence of stroke, including non-fatal events, is decreasing in most developed countries.5, 11 The same phenomenon has occurred in Finland in North Karelia,12 where the incidence rates decreased by 34% among men and by 32% among women between 1972–73 and 1976–77.

In this study the incidence rates are higher. The best explanation may be the more intensive case-finding and perhaps the more precise diagnostic methods.

In the international surveys the incidence of SAH varies widely, the highest rates being in Finland. In three Finnish studies the rates were 15.7,2 23.99 and 19.4/100,000/year.13 In this study the incidence was of the same high level, 18/100,000/year.

Case Fatality
The cumulative fatality within one year after the stroke was 37% which is fairly low compared with many previous studies.14-16 Likewise in the National Survey of Stroke4 and in the cooperative WHO coordinated study1 48% of patients died during one year. In the Finnish study of Espoo-Kauniainen6 the three month’s mortality of 45.8% was higher than in the population of the present study during the whole year. In the extension of this study to the years 1978–80 the case fatality was decreased to 40% per year.14

The relatively small case fatality in the present study has several explanations. It is possible that a larger proportion of mild cases was detected than in the other studies. The proportion of cerebral infarcts among all incident cases, 80%, was higher than in the other studies in Finland. Since the prognosis is better after a cerebral infarct than after cerebral haemorrhage, this might be an explanation of the better prognosis for patients in our study.

Improved control of hypertension might be one factor contributing to better survival of stroke patients. It is possible that the favorable one year survival in our study population partly reflects improving medical care.

<table>
<thead>
<tr>
<th>TABLE 6</th>
<th>Total Crude Sex-specific and Age-adjusted Annual Incidence (per 100,000 persons) in the Three Populations in Finland</th>
</tr>
</thead>
<tbody>
<tr>
<td>Incidence Rate</td>
<td>Kuopio Male</td>
</tr>
<tr>
<td>Crude</td>
<td>236</td>
</tr>
<tr>
<td>Age-adjusted*</td>
<td>274</td>
</tr>
</tbody>
</table>

*Age-adjusted by direct method to the 1979 population of Finland.

References
Fibrinogen, Blood Viscosity, and Cerebral Ischemia

J. Grotta, M.D.,* P. Ostrow, M.D., Ph.D.,† E. Fraifeld, B.S.,* D. Hartman, B.S.,† and H. Gary Ph.D.*

SUMMARY This study examines the effect of fibrinogen and consequent blood viscosity reduction on cerebral blood flow and cellular injury following severe cerebral ischemia for 30 minutes in 78 Wistar rats. In half of these rats 10 to 15 cc's of blood was removed and replaced with a mixture of 5% albumin and autologous red blood cells maintaining a constant hematocrit but resulting in a 30% decrease in fibrinogen and corresponding reduction in viscosity. Fibrinogen reduction resulted in a slight increase in baseline CBF and the elimination of post-ischemic hyperemia at 24 hours. Both study and control animals showed a similar decrease in CBF at 30 minutes and 2 hours. There was no significant difference in the severity of ischemic cellular change between the fibrinogen reduction group and controls, although there was a significant inverse relationship between the amount of viscosity change and severity of cellular injury within the treatment group. Fibrinogen reduction alone cannot significantly ameliorate ischemic injury in this model. Viscosity reduction therapy should include reduction of hematocrit and alteration of red cell deformability.

MANY POSSIBLE THERAPIES for acute cerebral ischemia alter blood viscosity, by hemodilution or phlebotomy,1-3 alteration of red blood cell deformability4-5 or reduction of fibrinogen concentration.6-7 The present study focuses on the effect of fibrinogen reduction alone on cerebral blood flow and cellular injury in an animal model of severe cerebral ischemia.

The viscosity of blood involves a complex relationship of red blood cells, fibrinogen, and the shearing forces within the vessel lumen6-10 (fig. 1). Several recent reviews have summarized how these rheologic factors may play a role in cerebral ischemia.11,12 Since a combination of blood viscosity, vascular tone, and intracranial pressure determine cerebral vascular resistance, lowering viscosity should increase CBF since

\[
\text{CBF} = \frac{ \text{perfusion pressure} }{ \text{cerebral resistance} } 
\]

Increased CBF and clinical improvement have been documented following viscosity reduction by phlebotomy1 and hemodilution1-2,4,5 but these maneuvers effect viscosity primarily by decreasing hematocrit. Less attention has been focused on the effect of reducing fibrinogen, even though the concentration of this molecule is increased in cerebral ischemia.13

Methods

Seventy-eight 300 gram male Wistar rats were fed ad lib. After anesthesia with 0.3 cc IM of a premixed cocktail of ketamine hydrochloride (one hundred milligrams per milliliter), Xylazine (20 milligrams per milliliter), and Acepromazine maleate (10 milligrams per milliliter) in a 3:3:1 ratio, all rats underwent bilateral vertebral artery cautery, and cannulation of the jugular vein and tail artery according to the method of Pulselli.14 After separating each common carotid artery from its sympathetic nerve trunk, loose ligatures were looped around the artery and the ligatures were delivered to the subcutaneous region. The skin was closed with metallic clips. Blood pressure was monitored throughout the procedure by connecting the tail artery to a Hewlett Packard transducer with strip chart recorder. (Hewlett Packard Co., Palo Alto California). Twenty-four hours after the initial surgery, the animals were exposed to ether for a few seconds and the skin clips removed. The common carotid arteries were
The incidence of stroke in the Kuopio area of East Finland.
J Sivenius, O P Heinonen, K Pyörälä, J Salonen and P Riekkinen

Stroke. 1985;16:188-192
doi: 10.1161/01.STR.16.2.188

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://stroke.ahajournals.org/content/16/2/188