Cardiac Arrhythmia Associated With Reversible Damage to Insula in a Patient With Subarachnoid Hemorrhage

Viktor Svigelj, MD, MSc; Anton Grad, MD, PhD; Igor Tekavčič, MD, MSc; Tomaž Kiauta, MD, PhD

Background The insular cortex has been shown experimentally to contain an arrhythmogenic center that may play an important role in the genesis of cardiac arrhythmias and electrocardiographic changes in patients with intracranial (eg, cerebrovascular) lesions. The description of our case is intended to substantiate this claim with a clinical observation.

Case Description A 37-year-old woman with subarachnoid hemorrhage suffered a severe reversible cardiac arrhythmia after neurosurgical clipping of an arterial aneurysm and removal of an intracerebral hematoma from the region of the left insula.

Conclusions The observed association of a neurosurgical intervention in the region of the left insular cortex with a cardiac arrhythmia supports but does not prove the suggested role of the insula in the causation of heart rhythm disturbances after stroke. (Stroke. 1994;5:1053-1055.)

Key Words • cerebral cortex • electrocardiograph • arrhythmia • subarachnoid hemorrhage

The mechanism of electrocardiographic (ECG) abnormalities after subarachnoid hemorrhage (SAH) has not yet been satisfactorily explained. Recently the insula was proposed to be an arrhythmogenic center important for the development of cardiac arrhythmias. The insula represents a cortical area with profuse interconnections with the limbic system, hypothalamus, and other areas involved in autonomic control. It also has an autonomic afferent input. To illustrate the possible importance of the insula in provoking cardiac arrhythmias, we present an SAH patient in whom evacuation of a hematoma and clipping of an aneurysm in the region of the left insula seemed to be associated with a severe reversible cardiac arrhythmia.

Case Report

A 37-year-old woman was admitted to the Department of Neurology of Medical Centre Ljubljana after an abrupt onset of severe headache, nausea, and vomiting. She gave a history of childhood tuberculosis but none of heart disease, angina, stroke, or any other disease. No ECG recordings taken before the admission were available. She smoked 20 cigarettes per day.

On admission the patient’s blood pressure was 16/10.6 kPa (120/80 mm Hg), her heart rate was 68 beats per minute, and her body temperature was normal. She was somnolent and had moderate nuchal rigidity. No lateralizing signs were found on neurological examination. Lumbar puncture revealed an opening pressure of 450 mm H 2O and a grossly bloody cerebrospinal fluid with xanthochromia. Computed tomographic scan showed blood in subarachnoid spaces, particularly the left-sided spaces, and an intracerebral hematoma in the left temporal region (Fig 1). A three-vessel femoral arteriogram revealed a large medial cerebral artery aneurysm (Fig 2) and a mild spasm of the left-sided cerebral arteries.

All blood tests performed during hospitalization (except for a postoperative normocyte normochromic anemia) were normal. Calcium and potassium blood levels on admission were 2.3 mmol/L and 3.9 mmol/L, respectively. On days 2 through 7 after admission, potassium levels were 3.8, 4.3, 4.0, 3.6, 4.6, and 3.5 mmol/L, respectively. (Levels between 3.5 and 5.2 mmol/L are considered normal in our laboratory.) Magnesium concentration was not determined. The results of analysis of daily 12-channel body surface ECG recordings during the first 7 days after SAH are shown in the Table and a sample of computer-recorded ECG in Fig 3.

The patient was transferred to the Department of Neurosurgery and after presurgical preparation underwent surgery on the morning of the fourth day after SAH. An intracerebral hematoma was evacuated from the region of the left insula. The aneurysm of the left middle cerebral artery, facing down toward the insula and in the anterior direction, was partly thrombosed and two to three times larger than it appeared on angiograms. A Yasargil clip was applied to the neck of the aneurysm. The wall of the aneurysm was coagulated and the wound closed. At the beginning of anesthesia administration, 3 mg of propranolol was given intravenously. No other cardioactive drugs were given before, during, or within the first day after the operation. No
intraoperative complications were noted, and no intraoperative heart arrhythmias were described in the anesthesia protocol.

In the two ECG recordings taken 2 and 6 hours after surgery, frequent premature ventricular complexes were present (Fig 4). The corrected QT period ($QT_c$) was from 0.42 to 0.44 second.

During the first week after SAH, the patient had no significant complications. Plasma norepinephrine concentrations, determined on days 1, 3, and 7 after SAH, were normal. The patient was discharged 16 days after SAH.

An ECG taken 6 months after SAH showed sinus rhythm, 61 beats per minute, an ST segment elevation in lead $V_3$, and a $QT_c$ of 0.38 second.

Discussion

Central autonomic regulation of heart rhythm is complex and not yet sufficiently explained. Recent studies suggest that the insula might be an arrhythmogenic center. In animal studies Oppenheimer et al. demonstrated an insular chronotropic organization. It is possible that the completely reversible cardiac arrhythmia in our patient, which developed postoperatively in an electrically somewhat unstable myocardium (as shown by the prolonged $QT_c$) and both appearing and disappearing on the day of the operation, was the result of manipulation of the insular region during the neurosurgical procedure or, in view of the absence of a description of heart arrhythmias in

![Fig. 2. Three-vessel angiogram shows a large medial cerebral artery aneurysm (black arrow) and a mild spasm of the left-sided cerebral arteries.](image1)

![Fig. 2. Three-vessel angiogram shows a large medial cerebral artery aneurysm (black arrow) and a mild spasm of the left-sided cerebral arteries.](image2)

![Fig. 3. Sample of a computer-recorded electrocardiographic tracing taken 2 hours after the operation. Note numerous premature ventricular complexes.](image3)
Electrocardiographic Findings During the First Week After Subarachnoid Hemorrhage

<table>
<thead>
<tr>
<th>Day</th>
<th>Heart Rate, bpm</th>
<th>ST Segment</th>
<th>T Wave</th>
<th>U Wave</th>
<th>QTc, s</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>85-94</td>
<td>Normal</td>
<td>Normal</td>
<td>Lead V2</td>
<td>0.43</td>
</tr>
<tr>
<td>2</td>
<td>89-92</td>
<td>Elevated in V2</td>
<td>Normal</td>
<td>Absent</td>
<td>0.44</td>
</tr>
<tr>
<td>3</td>
<td>64-70</td>
<td>Elevated in V2</td>
<td>Normal</td>
<td>Absent</td>
<td>0.40</td>
</tr>
<tr>
<td>4*</td>
<td>50-56</td>
<td>Elevated in V2</td>
<td>Negative in lead III</td>
<td>Lead V2</td>
<td>0.38</td>
</tr>
<tr>
<td>4†</td>
<td>64-70</td>
<td>Normal</td>
<td>Normal</td>
<td>Lead V2</td>
<td>0.42</td>
</tr>
<tr>
<td>4‡</td>
<td>70-75</td>
<td>Normal</td>
<td>Normal</td>
<td>Lead V2</td>
<td>0.44</td>
</tr>
<tr>
<td>5</td>
<td>64-80</td>
<td>Normal</td>
<td>Normal</td>
<td>Absent</td>
<td>0.38</td>
</tr>
<tr>
<td>6</td>
<td>57</td>
<td>Elevated in V3</td>
<td>Negative in lead III</td>
<td>Lead V2</td>
<td>0.40</td>
</tr>
<tr>
<td>7</td>
<td>56-60</td>
<td>Normal</td>
<td>Negative in lead III</td>
<td>Lead V2</td>
<td>0.41</td>
</tr>
</tbody>
</table>

bpm indicates beats per minute; QTc, corrected QT interval.
*Electrocardiogram (ECG) recorded on fourth day after subarachnoid hemorrhage (SAH), before operation.
†ECG recorded on fourth day after SAH, 2 hours after operation.
‡ECG recorded on fourth day after SAH, 6 hours after operation.

We conclude that the observed temporal coincidence of a surgical intervention in the region of the left insula with a transient, reversible cardiac arrhythmia appears to support the suggested role of the insula in the causation of ECG changes after SAH. The data are insufficient to be taken as proof of a causal relation, however.

References

Cardiac arrhythmia associated with reversible damage to insula in a patients with subarachnoid hemorrhage.
V Svigelj, A Grad, I Tekavcic and T Kiauta

Stroke. 1994;25:1053-1055
doi: 10.1161/01.STR.25.5.1053

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/25/5/1053