Hydrocephalus in the Adult Secondary to the Rupture of Intracranial Arterial Aneurysms

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The interest paid during the last years to the hydrocephalic syndrome in the adult is reflected in the increasing number of publications dealing with this subject. The nomenclature is somewhat confusing, for terms such as "occult hydrocephalus," "non-pressure hydrocephalus," "normal-pressure hydrocephalus," "low-pressure hydrocephalus," "idiopathic hydrocephalus," and "nonobstructive adult hydrocephalus" have been used to refer, within some limitations, to the same clinical entity. Some new etiological aspects have been reported and more active therapy is being carried out in most neurological centers.

This report concerns the hydrocephalic picture that may develop in the adult secondary to subarachnoid hemorrhage from the rupture of arterial intracranial aneurysms. A classical study of the reactions caused by blood when injected in the subarachnoid space and the relation of these reactions to the development of hydrocephalus was reported in 1928 by Bagley. Although since then both clinical and pathological reports on short series have been published, there are still no statistics on the incidence of this complication in a large series of adults and the frequency of hydrocephalus due to basal arachnoiditis following subarachnoid hemorrhage is not known. Statements like these, and the fact that during the treatment of our cases some interesting clinical, radiological, and etiological factors were found, have prompted this study.

Material and Methods

The serial carotid angiograms of 100 consecutive patients with proven subarachnoid hemorrhage following the rupture of arterial intracranial aneurysms have been analyzed according to the following principles. The size and shape of the lateral ventricles have been studied from the appearance of the central veins outlining these cavities, namely, the tributaries of the internal cerebral, including the thalamostriate. The frontal projection was used to obtain a numerical expression of the width of the lateral ventricles. For this purpose, measurements were made from a sagittal plane tangential to the medial border of the internal cerebral vein to the most lateral extension of the thalamostriate tributaries. These figures serve as an expression of the width of the lateral ventricle at the level of the sella media.

To minimize the factor of error, the measurements were made in a standard projection and the numerical values expressed in terms of "ventriculocranial index," thus eliminating errors of magnification. This index was obtained from the quotient:

\[
\text{ventriculocranial index} = \frac{\text{lateral ventricle width in mm}}{\text{half of the skull width in mm}}
\]

The index represents the width of the ipsilateral half of the skull measured from the sagittal plane to the inner table at the level of its maximal breadth. The changes in shape of the thalamostriate vein in the anteroposterior projection (Fig. 1) were used to check our measurements in case important index changes between two angiograms were registered. The venograms on the lateral projection were used in the same way. The accuracy of this method has been reported previously.

In this study, we will not refer to the pneumoencephalograms done in some of the cases, because for statistical purposes we have considered the medical indications for this study as a factor of selection per se. Furthermore, it is a well-known fact that for measurement purposes pneumoencephalograms have some disadvantages due mainly to unequal distribution of air between the
lateral ventricles and a certain degree of dilatation when the air is absorbed.

Changes of index greater than 0.025 (approximately 2 mm on the film) between two or more consecutive ipsilateral angiograms were considered as true changes in size of the lateral ventricles and expressed in terms of dilatation or shrinkage. When the size of the ventricle increased to reach figures greater than an index of 0.33, that case was considered as having a “hydrocephalic index.” The cerebral circulation time was determined only on the serial angiograms performed after the third week following the last subarachnoid bleeding, consequently eliminating the influence that vasospasm, (which is usually gone by that time, 7,22) may have upon the values obtained.

The cases having enlargement of the lateral ventricles according to this evaluation have been studied separately on a clinical basis and the findings related to the radiological features. The clinical condition of our patients was analyzed according to the usual criteria accepted by different authors dealing with hydrocephalus in the adult. 1,2,11,16,17,25,31

The combination of these signs and symptoms has resulted in the classification of our case material (patients with enlargement of the lateral ventricles) into three categories (Table 1):

Group 1. Asymptomatic patients

Group 2. Patients with mild signs and symptoms (initial picture)

Group 3. Patients with severe signs and symptoms (advanced picture).

Although all patients included in a specific group did not necessarily have the whole complex of symptoms, each had a combination of at least three of them.

Results

Ventricular Dilatation. Of the 100 patients who had subarachnoid bleeding as a consequence of the rupture of intracranial aneurysms, 34 developed definite enlargement of the lateral ventricles (Fig. 2). Of these 34 cases, 17 had a “hydrocephalic index” (above 0.33), and 27 had index values below this level (Fig. 3). In 11 cases it was impossible for various reasons to estimate the size of the lateral ventricles. In 27 patients, only one angiographic study could be analyzed; three of these angiograms showed a hydrocephalic ventriculocranial index. Whether the enlargement of the ventricles in these cases was related to the actual disease could not be stated because of a lack of previous angiograms for comparison.

Ventricular Shrinkage. In none of the cases could a significant shrinking of the lateral ventricles be observed except for those patients in whom a ventriculoatrial shunt had been performed (three cases). One patient who presented a constantly increasing dilatation of the lateral ventricles during 3 years of observation (angiographic studies repeated four times at 3- to 12-month intervals), and who showed in the last study a spontaneous regression, still had a hydrocephalic ventriculocranial index.
TABLE 1

Signs and symptoms in 34 adults with post-hemorrhagic dilatation of the lateral ventricles

<table>
<thead>
<tr>
<th>Category</th>
<th>Hydrocephalic Status</th>
<th>Specific Signs and Symptoms</th>
<th>No. of Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group 1 (15 patients)</td>
<td>asymptomatic; index rate = 0.311</td>
<td>unrelated signs and symptoms</td>
<td>15</td>
</tr>
<tr>
<td>Group 2 (10 patients)</td>
<td>mild signs and symptoms; index rate = 0.318</td>
<td>disturbances in thinking and behavior, essential hypertension, mild gait disturbances of “apraxic ataxic” type, mild paraparesis, seizure-like episodes, chronic headache</td>
<td>9</td>
</tr>
<tr>
<td>Group 3 (9 patients)</td>
<td>severe signs and symptoms; index rate = 0.343</td>
<td>presenile dementia, essential hypertension, severe impairment of gait, spastic paraparesis, epilepsy (grand mal), chronic headache, incontinence, akinetic mutism</td>
<td>9</td>
</tr>
</tbody>
</table>

**Significant Clinical Findings.** The following findings observed in the 34 patients with post-hemorrhagic enlargement of the lateral ventricles were considered significant:

1. **Cerebral circulation time.** There was a significant correlation between the size of the lateral ventricles and the cerebral circulation time (Fig. 4).

2. **Arterial spasm.** Angiography showed arterial spasm after bleeding in 68% of the cases that later developed ventricular dilatation, in 78% of the cases with a ventriculocranial index of more than 0.33, and in 41% of the cases that preserved normal ventricular size.

3. **Intracerebral hematoma.** Of the 34 pa-

![Graph](image-url)  

**Fig. 2.** Graph plotting the ventriculocranial index of patients in whom a dilatation of the ventricular system was disclosed. Index changes more than 0.025 = 2 mm. The broken lines connect the index figures obtained in different ipsilateral angiograms on the same patient. Values above the full line (index 0.33) are considered as radiological evidence of hydrocephalus.
4. Location of the aneurysm. The aneurysm was located on the internal carotid artery in 11 cases, the anterior communicating artery in 10, the pericallosal artery in 1, and the middle cerebral artery in 9. Multiple aneurysms were present in three cases. No cases with aneurysm on the vertebro-basilar system were found.

5. Number of hemorrhages. The incidence of multiple hemorrhages was 70.5% (24 of 34 patients), and 82% in those cases with a hydrocephalic index above 0.33.

6. Intracranial operation. Previous intracranial surgery had been performed on 29% of the cases that developed hydrocephalus and 24% of those without ventricular dilatation. Accordingly, in our series, this feature appeared to lack importance as a hydrocephalus-inducing factor.

7. Sex and age. No important sex difference was found but age seemed a factor in that younger patients showed a greater tendency to develop hydrocephalus than older patients. Of the 34 patients, three were under 20 years of age, and two of these developed severe hydrocephalus.

8. Time from subarachnoid hemorrhage to hydrocephalus. In our series, hydrocephalus was manifested as early as 3 weeks after subarachnoid hemorrhage, although in most instances it followed a progressive course during months or years according to different factors in the particular case. Almost invariably the process of dilatation that led to the hydrocephalus started (except in two patients) during the month following the hemorrhage (Fig. 2).

Clinical Picture. The hydrocephalic picture of the 34 patients with enlargement of the lateral ventricles is described below according to their group classification.

Group 1. These 15 patients (44.1%) were found to be asymptomatic or had signs and symptoms that could not be related to the hydrocephalic picture. The ventriculocranial index rate in this group was 0.311.

Group 2. These 10 patients (29.4%) had mild symptoms indicating early post-hemorrhagic hydrocephalus. The ventriculocranial index rate in this group was 0.318. Our Group 2 patients can be compared to those in Stages 1 and 2 of Ekbom, et al. The symptoms and signs have been well described elsewhere, although in our Group 2 cases some clinical features should be emphasized. The mental disturbances and seizure-like episodes (Table 1), namely, "abrupt loss of senses lasting a few minutes during which the patient lies motionless and flaccid," have in many instances been labeled previously as "psychogenic" and not as having any organic basis; our review shows, however, that hydrocephalus was present...
Fig. 4. Graph demonstrating the correlation between the size of the ventricles (expressed in terms of the ventriculocranial index) and the cerebral circulation time (expressed in seconds). The dotted lines indicate upper normal values. (Normal circulation time = 3.5 ± SD 0.5.)

and may have been contributory to these episodes.

Essential hypertension, a relatively new aspect of this clinical complex,11,15 was found to be present in eight out of these 10 previously normotensive patients, according to the hypertensive criteria of the Swedish Life Insurance Companies (i.e., pressures above 150/90 under the age of 40, 160/95 in the age group 40-60, and 180/100 in older age groups).11,14,16 This was in contrast to the fact that none of the patients without ventricular enlargement became hypertensive.

Group 3. These nine patients (26.4%) presented severe symptoms indicating an advanced stage of adult hydrocephalus. Presenile dementia, as in Stage 3 of Ekbom, et al.,11 was the most striking feature, accompanied in some instances by gait disturbances, incontinence, and spastic paraparesis. Five patients suffered grand mal epileptic attacks that appeared for the first time between 6 to 18 months after the last subarachnoid hemorrhage. The syndrome of akinetic mutism was present in four cases. Three patients developed hypertension.

Discussion

The development of communicating hydrocephalus following an inflammatory reaction caused by blood in the basal cisterns is a fact that has been extensively discussed in the literature, both from the etiological5,6,10,12,18,20,21,26,30,32 and clinical5,6,12,18,26,30,31 points of view. Its occurrence in our series as the result of rupture of intracranial aneurysms seems to be more common than previously accepted.

The fact emerges from our study that approximately 33% of the patients affected by this kind of cerebral insult may be expected to develop ventricular dilatation, and that 19% will suffer from neurological deficits. Thus, an unexpected clinical impairment can occur in one out of five patients with ruptured intracranial aneurysms, related to post-hemorrhagic communicating hydrocephalus. Due to the nature of the process, similar figures could be applied to all kinds of patients with subarachnoid hemorrhage of different origins.

We have confirmed that both the enlargement of the ventricles and the development of the clinical picture are progressive and that the degree varies from case to case. In some instances, a typical clinic-radiological picture of adult hydrocephalus can appear as early as 3 weeks following the intracranial bleeding, but in most cases a steady dilatation of the ventricles will be observed during a certain period up to 3 years (Fig. 2) and then remain stable. The process of fibrosis of the leptomeninges requires at least 2 weeks to take place.20 Contrary to what might be expected, the clinical picture presented by these cases is not always proportional to the degree of dilatation; this fact depends on the capacity of each particular case to compensate for the hydrocephalus. However, in our series a slight correlation has been found between the intensity of the symptoms and the ventriculocranial index rate in each clinical group (Table 1).

In many instances the initial complaints of
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the patients were reported as mental disturbances and behavioral problems, which because of their nonspecific nature may not have been thought related to an organic process such as hydrocephalus. Such complaints in our series included family conflicts, incapacity to perform a previous job, and impairment of memory and concentration; the patients were erroneously treated as neurotic and their hydrocephalic factor disregarded. In the same way, patients having seizure-like fits were labeled as "cases of hysteria" because of the non-specificity of the attacks and their close relationship to behavioral disturbances.

Essential hypertension has been found to occur in patients with hydrocephalus due to ectasia of the basilar artery and in patients with aqueductal stenosis. It is therefore worth mentioning that, in our series, 11 of 34 patients with hydrocephalus became hypertensive. Hence, the occurrence of high blood pressure in a previously normotensive patient may suggest that this patient is developing hydrocephalus.

In the same way, it is advisable to suspect hydrocephalus when a patient presents grand mal seizures 6 to 18 months after a subarachnoid hemorrhage. In such cases, investigations should be carried out to exclude hydrocephalus, for it is possible, after shunting, to control the epileptic fits (as in one of our three surgical cases and in similar cases of Ekbom, et al.). Here, modern echoencephalographic techniques have a definite application.

We believe that the signs and symptoms discussed above are closely related to diffuse cerebral dysfunction resulting from disturbances in the normal physiological pattern of intracranial pressure and especially to alterations in cerebral hemodynamics. It has been demonstrated that cerebral blood flow is reduced in these cases, and as shown in our present study, there is often an alteration of the cerebral circulation time (Fig. 4). The elongation and stretching of the intracranial vessels due to the enlargement of the ventricles have been thought to explain the narrowing of the arteries and the reduction of flow. According to Yakovlev spastic paraparesis can also be explained as a result of a similar mechanism, namely, stretching of the long paracentral fibers to the lower extremities. Periventricular demyelinization as reported by Penfield and Elvidge in cases of hydrocephalus can result as a consequence of mechanical as well as circulatory disturbances. It is likely that the apraxia of gait or Brun's frontal lobe ataxia has its basis in alterations related to similar pathophysiological mechanisms.

Not only may the dilatation of the ventricular system be due to obstruction of the cerebrospinal fluid pathways following basal pachymeningitis, but to other factors as well. Vasospasm (68% of the cases) following intracranial bleeding is a phenomenon that must be considered directly related to the etiology of this complication. Here it probably acts through anoxic ischemia as observed by Cate; this results in cellular degenerative changes and a diffuse wasting of brain tissue. These alterations are more marked at the level of the periventricular structures. Furthermore, in the same areas, the occurrence of hemorrhage in previous hypoxic regions has been reported in cases of infantile hydrocephalus.

Age is a factor that may play a relative role, as observed experimentally on dogs by Bagley. Our younger patients appear to have had a greater tendency to develop hydrocephalus than older patients.

The location of the aneurysm is obviously a significant factor, since aneurysms in direct communication with the subarachnoid space are most likely to bleed freely into the cisterns and consequently produce a greater reaction of the leptomeninges to blood; in our series, however, unlike others, a relatively large number of cases with middle cerebral artery aneurysms had this complication. This can partly be explained because, in our particular group, arterial spasm was almost always the rule and a greater number of intracerebral hematomas were present. It has been reported that in cases with intracerebral hematoma the intensity and duration of angiospasm is increased. As expected, multiple bleedings are in direct relation to a higher incidence of hydrocephalus.

Summary

Post-hemorrhagic hydrocephalus secondary to subarachnoid hemorrhage caused by the rupture of arterial intracranial aneurysms has been investigated in a series of 100 con-
secutive patients on the basis of angiographic and clinical studies. Of the 100 cases, 34% were found to show enlargement of the lateral ventricles and 19% to have signs and symptoms of adult hydrocephalus.

The value of serial carotid angiography in the study of ventricular size and hemodynamic changes in hydrocephalus has been analyzed and emphasized. The role played by different factors in the pathophysiology of this complication has been discussed, with special reference to cerebral angiospasm.

The clinical aspects characterizing the patients in this series have been pointed out. Of special interest was the fact that a significant number of the hydrocephalic patients developed essential hypertension.

References


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