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REVIEW

K. N. Fountas · E. Z. Kapsalaki · T. Machinis · I. Karampelas · H. F. Smisson · J. S. Robinson

Review of the literature regarding the relationship of rebleeding and external ventricular drainage in patients with subarachnoid hemorrhage of aneurysmal origin

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Abstract Acute hydrocephalus is a well-documented complication of subarachnoid hemorrhage. The insertion of external ventricular drainage (EVD) has been the standard of care in the management of this complication, aiming primarily at immediate improvement of the clinical condition of these patients, making them more suitable candidates for surgical or endovascular intervention. In our current communication, we review the pertinent literature regarding the relationship of rebleeding and EVD. Several studies have implicated a significantly increased risk of rebleeding in patients with EVD, compared with patients without it. Abrupt lowering of the intracranial pressure could lead to rebleeding due to decreased transmural pressure or removal of the clot sealing the previously ruptured aneurysm. However, a variety of parameters that could affect the rebleeding rate, such as the timing of surgery, the timing and duration of drainage, the size of the aneurysm, as well as the severity of the initial hemorrhage, do not seem to have been adequately explored in the majority of these studies. In addition, a number of clinical trials have failed to provide evidence for the negative role of EVD in the development of rebleeding. Conclusively, further long-term multi-center studies are required in order to establish the exact nature of the relationship between EVD and rebleeding after aneurysmal subarachnoid hemorrhage.

Keywords Aneurysm · External ventricular drainage · Intracranial pressure · Rebleeding · Subarachnoid hemorrhage

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K. N. Fountas · T. Machinis · I. Karampelas · H. F. Smisson · J. S. Robinson
Department of Neurosurgery,
The Medical Center of Central Georgia,
Mercer University, School of Medicine,
Macon, GA 31201, USA

E. Z. Kapsalaki
Department of Neuroradiology,
The Medical Center of Central Georgia,
Mercer University, School of Medicine,
Macon, GA 31201, USA

K. N. Fountas (✉)
840 Pine St. Suite 880,
Macon, GA 31201, USA
e-mail: knfountasmd@excite.com
Tel.: +1-478-7437092
Fax: +1-478-7430523

Introduction

The development of acute hydrocephalus after subarachnoid hemorrhage (SAH) was firstly described by Bagley in 1928 [2]. Since then, several clinical series have well established the relationship between SAH and hydrocephalus, as well as the existence of two types of the latter: an acute one, observed immediately after the ictal event and a chronic one observed in a second phase, approximately 7–10 days post-ictally [1, 4, 8, 10, 11, 15, 18, 21, 22, 25, 29, 31, 35, 42, 43]. The incidence of post-SAH hydrocephalus varies significantly among different clinical series; Spallone reported 9% incidence [36], van Gijn et al. [39] and Kusske et al. [22] reported 20%, Vassilouthis and Richardson 12.4% [40], while Black reported 67% [3]. This inconsistency can be explained by the significant variations of the defining criteria of hydrocephalus, the diagnostic methodology utilized for applying these variables and also some systematic biases due to the fact that the examined surgical series could include only good surgical candidates of better clinical grade.

The insertion of an external ventricular catheter as a diagnostic and therapeutic tool for acutely increased intracranial pressure (ICP), due to the development of hydrocephalus, has been considered the standard of care in the management of these patients [20]. The rationale of using external ventricular drainage (EVD) has been outlined by Kusske et al. [22]: (1) patients with acute hydrocephalus are generally poor surgical candidates and the insertion of EVD (via dealing effectively with increased ICP-related factual and/or potential complications) could enhance their

overall fitness to undergo surgery, and (2) the necessity of a permanent cerebrospinal fluid (CSF) shunt can be accurately assessed by monitoring ICP. In their study, they concluded that external ventriculostomy was beneficial not only in improving short-term but also long-term outcome in patients with post-SAH hydrocephalus [22]. However, the use of external ventriculostomy has in itself been associated with various complications, among which the most serious is the reported increased occurrence of acute rebleeding, due to abruptly decreased transmural aneurysmal pressure [5, 24, 26, 28, 30, 38, 39]. A relatively small number of clinical series of limited size has been published previously, examining the incidence of rebleeding in patients with aneurysmal SAH in relation to the placement of an external ventriculostomy [5, 23, 26, 28, 32, 33, 36–38, 41].

The purpose of our current communication is to review all the related published series, in an attempt to enlighten this very controversial topic and identify those factors that might affect the incidence of rebleeding in patients with aneurysmal SAH harboring acute onset hydrocephalus.

Background data

It is well known that intra-aneurysmal pressure is equal to the systemic blood pressure and the transmural pressure can be defined as the difference between the blood pressure (BP) and the ICP [9]. It is apparent that any sudden increase of BP or decrease of ICP could lead to a rebleeding episode in a patient with a SAH due to a ruptured aneurysm [34]. Nornes has reported that increased ICP can produce a "brain tamponade"; the concomitant absence of cerebral blood flow, though, represents a major problem in this ultimate "hemorrhagic-compressive" pressure state [26]. In his well-designed prospective study of 29 patients with ruptured saccular-aneurysms unfit for immediate surgery, ICP was monitored by using miniature epidural pressure transducer [26]. He found an increased risk of rebleeding when ICP was within normal limits after SAH (280 mmH_20) [26]. Similarly, Voldby and Enevoldsen in their clinical study examined the relationship of ICP (measured by an intraventricular catheter) and rebleeding rate, which was 17% (9/52) in their series [41]. They concluded that ventricular drainage of CSF to a level of 25 mmHg did not increase the rate of rebleeding [41]. On the other hand, they observed that the usage of ventricular drainage while the repeat rupture was taking place seemed to exert a deleterious effect on the natural mechanisms that lead to arrest of hemorrhage [41]. They suggested that drainage of CSF should be avoided during recurrent hemorrhage and should not be resumed until a steady-state ICP level has been reached [41].

Several mechanisms have been implicated in the pathogenesis of a rebleeding associated with the insertion of an external ventricular drain and subsequent lowering of the ICP [5, 12, 28, 34]. This sudden change of ICP can cause ventricular shift with subsequent displacement of the formatted aneurysmal clot, tamponading the previously ruptured aneurysm [12]. It has been shown previously in a histopathological study that re-rupture of an aneurysm occurs again through the original tear, an area very sensitive to any pressure changes or biomechanical stress [7]. Additionally, the insertion of a ventricular catheter may induce intracranial fibrinolytic activity, which can activate a cascade, resulting in lysis of the aneurysmal clot [12]. Similarly, Rosenorn et al. [34] have shown that lowering of ICP by administering mannitol could be responsible for rebleeding of a previously ruptured aneurysm.

Troupp, in a limited series of ten patients with the admitting diagnosis of SAH, (seven patients with one or more aneurysms and three patients with SAH of non-aneurysmal etiology) employed EVD with concomitant ICP monitoring [38]. In two patients (28%), rebleeding occurred after pressure-reducing measures had been taken [38]. Moreover, Hasan et al. [13], in their large (473 consecutive patients) prospective series, examined the association of early ventricular drainage with rebleeding in patients with post-SAH hydrocephalus. They found that the rebleeding rate in patients with ventricular drainage was significantly higher than the one in patients without acute hydrocephalus (43% vs 20%; P=0.019) and also in patients with acute hydrocephalus but without ventricular drainage (43% vs 15%; P=0.025). Similarly, van Gijn et al. [39] reported in a multi-institutional prospective study (174 consecutive patients with aneurysmal SAH) a rebleeding rate of 17.6% (6/34) among patients with hydrocephalus and EVD, while the rebleeding rate among patients without ventricular drainage was only 9% [39]. Although the rebleeding rates in this study are unusually low, the rebleeding rate among patients with ventricular drainage is almost twice the one observed in patients without drain [39]. Pare et al. [28], in their cohort study of 128 patients with confirmed aneurysmal SAH, found increased rebleeding rate among patients with EVD (30%) compared with the ones without (8.3%). This difference reached the level of statistical significance (P<0.006), although the validity of the statistical analysis was questionable due to multiple testing [28]. At the same wavelength, Bogdahn et al. [5], in their series consisting of 45 patients with SAH of aneurysmal (17 patients) and of unknown (28 patients) etiology, reported a rebleeding rate of 42% (19/45) among the patients with an external ventriculostomy, compared with 27% of a historical control group of patients with SAH without EVD. Furthermore, Kawai et al. [17], in their retrospective study of 93 patients with grade V (WFNS classification) subarachnoid hemorrhage, observed a three-fold higher rate of rebleeding within 72 h after admission among patients with ventricular drainage compared with those without (32.1% vs 10.3%; P=0.03).

Contrarily, McIver et al. [23], in a recent well-designed retrospective clinical study of 45 consecutive patients, harboring an external ventriculostomy for acute post-SAH hydrocephalus of aneurysmal origin, reported a rebleeding rate of only 4.4% (2/45). Their results are in agreement with those of Rajshekhar and Harbaugh [32], who reported a rebleeding rate of 14% in patients with external ventriculostomy after post-SAH acute hydrocephalus. Sundbarg

and Ponten [37] reported a rebleeding rate of 16.6% in their large series (127 patients) with continuous CSF ventricular drainage when ICP>15 mmHg and concluded that ventricular drainage did not provoke rerupture of the underlying aneurysm. Similarly, Roitberg et al. [33] reported no rebleeding associated with EVD in patients with acute hydrocephalus after aneurysmal SAH. The common characteristic of these clinical series, besides their low rebleeding rates, is the employment of early surgical intervention.

The observed variance of rebleeding rate in these patients among different clinical series seems to be affected by four factors: (1) the heterogeneous composition of these series, (2) the timing of insertion of an external ventriculostomy and the length of the draining period, (3) the timing of surgical clipping or endovascular coiling of the underlying aneurysm(s), and (4) the clinical grade of the patients upon their admission.

Meticulous review of existent literature points exactly to the significant variability and heterogeneity of the reported series [5, 13, 23, 26, 28, 32, 33, 37–39, 41]. Unfortunately, although some investigators have included exclusively patients with acute hydrocephalus secondary to an aneurysmal SAH in their series [23, 26, 28, 32, 33, 41], others have reported patients with SAH of aneurysmal but also of unknown etiology [5, 13, 37, 38]. Interestingly, some investigators have control groups in their studies [13, 23, 28, 37], while others have used historical control groups without making clear if these historical control groups were referring to patients with SAH without hydrocephalus or patients with post-SAH hydrocephalus but without ventricular drainage [5, 32, 33, 38, 41]. This variability makes any comparison very difficult and the extraction of any conclusions, regarding the etiologic relationship of rebleeding and ventricular drainage, very risky.

Another parameter which could affect the rebleeding rate, is the timing of insertion of an EVD, as well as the length of the draining period. McIver et al. [23] reported that all their re-hemorrhages occurred within 8 h after the insertion of the EVD, which had been inserted within 24 h from the ictal event [23]. Hasan et al. [13] did not provide any information regarding the timing of the ventriculostomy, while they vaguely reported that their rebleeding rates determined in a 12–day post-ictal period. Similarly, Pare et al. [28] and Troupp [38] reported no data regarding these parameters. Bogdahn et al. [5] reported that most rebleedings occurred during the first week of drainage, confirming the observations of Nornes [26] regarding the occurrence of all of his rebleeding cases within the first two post-ictal weeks (average 7.7 days). Based on these data, the chance of rebleeding in patients with an external ventriculostomy increases with time during the first two post-ictal weeks and follows the well-described time pattern of rebleeding in cases of a ruptured aneurysm without external CSF drainage.

It is obvious that early clipping of the ruptured aneurysm minimizes the risk of rebleeding; McIver et al. [23], Roitberg et al. [33] and Rajshekhar et al. [32] found rebleeding rates significantly lower than ones reported by other series with prolonged draining without clipping of the aneurysm. Early surgical or endovascular intervention not only minimizes the risk of rebleeding associated with EVD placement, but also allows the more aggressive management of ICP, which could subsequently minimize the chance of an ischemic complication. The insertion of an EVD seems to adequately serve the purpose of quickly improving the clinical neurological grade of poor surgical candidates and facilitating early prompt surgical or endovascular intervention, which would eliminate the risk of rebleeding [22, 23]. In those cases that prolonged CSF drainage is required without early surgical intervention, a spinal lumbar drainage might represent a valid option for managing hydrocephalus and avoiding the deleterious effects of ischemia and vasospasm along with a decreased chance of rebleeding [6, 12, 19].

Finally, another parameter that could confound the etiologic relationship of rebleeding due to the insertion of an EVD is the poor neurological grade upon the patient's admission. It is widely accepted that patients requiring an EVD insertion upon their admission have a worse grade; it has also been postulated that patients of poor clinical grade might be harboring aneurysms of larger size (large aneurysm size has been identified as a predisposing aneurysmal rerupturing factor) and more dense SAH, parameters that could be independent factors for rebleeding or co-factors acting synergistically with transmural pressure changes due to EVD [14–16, 27, 28]. Unfortunately, in the vast majority of the existent studies examining the relationship of rebleeding and EVD, have the issues of the

Table 1 Summary of the most significant clinical studies examining the role of EVD in rebleeding among patients with subarachnoid hemorrhage of aneurysmal origin

Author (year of publication)	Number of patients	Type of study	Findings
Sundbarg and Ponten (1976)	127	Retrospective	No difference in rebleeding rate in patients with EVD
Voldby and Enevoldsen (1982)	52	Prospective	No increase in rate of rebleeding after EVD when ventricular drainage maintained up to a level of 25 mmHg
van Gijn et al. (1985)	174	Prospective	Increased rebleeding rate in patients with EVD
Hasan et al.(1989)	473	Prospective	Increased rebleeding rate in patients with EVD
Bogdahn et al. (1992)	45	Retrospective	Increased rebleeding rate in patients with EVD
Pare et al. (1992)	128	Prospective	Increased rebleeding rate in patients with EVD
Kawai et al. (1997)	93	Retrospective	Increased rebleeding rate in patients with EVD
McIver et al. (2002)	45	Retrospective	No difference in rebleeding rate in patients with EVD

size of the ruptured aneurysm or the extent and degree of the underlying SAH been addressed. However, Kawai et al. [17] (in the only study specifying the grade of the clinical condition of the involved patients) reported that the rebleeding rate among patients with grade V subarachnoid hemorrhage was still three times higher in patients who underwent EVD compared with those without ventriculostomy.

In summary, the comparative findings of the major studies to date are listed in Table 1.

Conclusion

The insertion of an EVD in patients with acute hydrocephalus secondary to aneurysmal SAH, particularly in patients of poor clinical grade is a necessity; this maneuver will immediately improve the clinical grade of these patients, make them better surgical candidates and allow definite surgical or endovascular intervention. The maintenance of ICP above 15–20 mmHg and timely prompt intervention will minimize the risk of rebleeding. A risk which is definitely increased in patients with prolonged EVD compared with ones without it. Multi-institutional, prospective, large clinical series are required for defining the role of the aneurysmal size, as well as the admitting clinical grade in the relationship of rebleeding and EVD.

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