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Blood pressure changes after aneurysmal subarachnoid hemorrhage and their relationship to cerebral vasospasm and clinical outcome

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ABSTRACT

Objective: Cerebral vasospasm (VS) and resulting delayed ischemic brain injury constitute the most severe secondary complication after subarachnoid hemorrhage (SAH). Identification of early clinical predictors of developing vasospasm and poor outcome has remained a major challenge in neurointensive care medicine. Aim of the present study was analyze the relevance of spontaneous changes in blood pressures and their predictive value for predicting vasospasm as well as adverse clinical outcome.

Methods: 98 aneurysmal SAH patients were analyzed retrospectively. Patients were divided into two study groups: (1) VS+ (developing VS) and (2) VS− (not developing VS). Repeat-angiography was routinely performed on day 8 after SAH or earlier if clinical signs were suggestive for overt vasospasm. Systolic, diastolic and mean blood pressures were averaged hourly and plotted over time. Secondly, blood pressure (BP)-progression was analyzed with respect to clinical outcomes as assessed by the Glasgow outcome scale.

Results: Mean, systolic, and diastolic blood pressure values progressed in both VS− and VS+ cohorts over time. However, as early as 4 days after SAH a significant dissociation of RR curves was observed between the groups with patients in the VS+ group displaying a significantly higher slope coefficient of blood pressure elevation. An increase of mean arterial pressure >20% within the first 4 days was predictive of developing vasospasm. Elevation of mean arterial blood pressure in the VS+ group was mainly attributable to changes in diastolic pressure. Elevation of mean arterial blood pressure >25% within the first week after SAH was associated with unfavorable outcome.

Conclusions: SAH leads to spontaneous and progressive elevations in mean arterial blood pressure. Vasospasm might be anticipated by identifying early elevations of mean arterial blood pressure. Finally, spontaneous elevations of mean arterial blood pressure correlate with poorer outcomes.

1. Introduction

Little is known about the natural course of blood pressure following subarachnoid hemorrhage (SAH) [1–3]. Early reports have suggested a correlation between spontaneous increases in blood pressure and the development of delayed cerebral ischemia and poor outcome [4,5]. However, to date, insight into this correlation and the underlying pathophysiology remains scarce.

Since arterial blood pressure monitoring is routinely performed in SAH patients during their intensive care unit stay, detection of mean arterial blood pressure changes might represent an easily accessible and cost effective strategy to assist in identifying patients at risk for developing delayed cerebral ischemia (DCI). Still, an early detection of these patients at risk has remained one of the major challenges in neurointensive care [6,7]. Especially, in time detection of developing cerebral vasospasm may allow for an earlier initiation of hemodynamic or interventional treatment strategies with the aim to prevent DCI and cerebral infarctions.

Based on this, the first objective of this study was to document spontaneous blood pressure changes in SAH patients and to determine whether changes in blood pressure after SAH may be used to predict the development of VS and DCI. The second objective was to study the predictive value of blood pressure changes for clinical outcome.

2. Methods

Over a time period of 32 months, 141 patients with aneurysmal SAH were included into this retrospective study. Patients who

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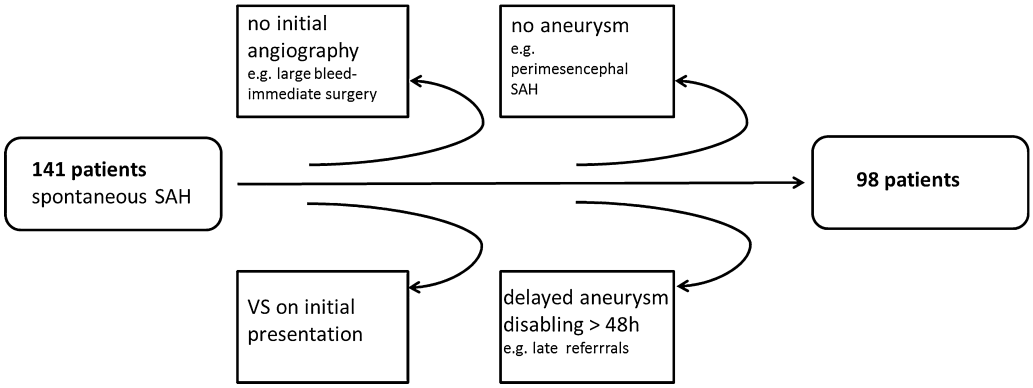

Fig. 1. Reduction of cohort number after excluding patients without detection of an aneurysm, without initial or late digital subtraction angiogram, with delayed aneurysm disabling and with initial presentation of VS.

did not receive a conventional angiogram 7–9 days after admission to confirm or exclude vasospasm, as well as patients with initial presentation of VS were excluded from the study, likewise patients in whom no aneurysm as source of subarachnoid bleeding could be determined and also patients that received delayed aneurysmal disabling (after day 2 post SAH), e.g. late or secondary referral (see flow chart, Fig. 1). Patient characteristics were stratified by gender, age, clinical grade, method of aneurysm repair and preexisting cardiovascular illness, including previous antihypertensive medication.

Patients were treated following a standardized institutional protocol. Upon admission, each patient received a cranial computed tomographic (CT) scan including CT angiogram and was then admitted to the intensive care unit. Catheter angiography was performed within 12 h of admission and the ruptured aneurysm was excluded from the circulation within 24 h of initial presentation by surgical clipping or endovascular coiling. Postoperative CT scans were conducted within 24 h after aneurysm repair.

Routine blood pressure management was permissive (between MAP 70 and 130) and fluid management aimed at normovolemia. Important for our analysis, none of the patients in this study received prophylactic vasopressor therapy before diagnosis of vasospasm. In patients with a history of hypertension who were on antihypertensive drugs, the medication was continued. All patients received oral nimodipine (60 mg every 4 h) starting on the day of admission to the ICU and continued for 14 days, without exception. In case of hypotensive side effects nimodipine was discontinued prematurely. This allowed us to follow spontaneous blood pressure changes in the acute and subacute phase after SAH.

Transcranial Doppler ultrasound was performed daily. Catheter angiography was routinely repeated on day 7–9 after SAH in asymptomatic patients in order to control for angiographic cerebral vasospasm. Patients exhibiting clinical or TCD signs suggestive of vasospasm, received an angiogram on the same day of exhibiting those signs. Clinical signs suggestive of VS were defined as follows: (1) Delayed neurological deterioration which was not attributable to another source (e.g. hydrocephalus). (2) TCD flow elevation (mean MCA velocity > 120 cm/s, ΔMCA velocity 24 h>50 cm/s, or Lindegaard index > 3). Patients were allocated to the vasospasm group (VS+) only if their angiogram demonstrated a >33% narrowing of cerebral vessels.

Upon angiographic verification of cerebral vasospasm hypertensive therapy was induced with vasopressors. Any active induction of hypertension marked the end of the data acquisition for the individual patient. Thus, for VS+ patients, their data was only plotted until the day of diagnosis and induction of hypertensive therapy, in order to avoid interference with vasopressor-induced active changes in blood pressure.

In all SAH patients, clinical charts were reviewed as follows: systolic, diastolic and mean blood pressures were averaged hourly and daily and plotted over time starting from the admission to the hospital with respect to the moment of clinical and angiographically verified vasospasm. Patients were divided into two study groups: (1) VS+ (developing VS) and (2) VS− (not developing VS). First, the course of spontaneous blood pressure after admission was plotted chronologically. Sequential unpaired *t*-tests were performed between groups and parameters to evaluate the slope behaviors. In addition, patients were stratified for clinical grades (WFNS grades I–V). Second, blood pressure changes were correlated with clinical outcome, as assessed by the Glasgow clinical outcome scale (GOS), independent of the prevalence of vasospasm. The different GOS cohorts were then again compared using Student's *t*-tests. All graphs were plotted with Graphpad Prism 4 software and statistical analysis was done using Graphpad Instat software.

3. Results

Of the 141 patients analyzed, 98 met the inclusion criteria. Patient characteristics are summarized in Table 1. Notably, there was no statistical difference regarding past history of hypertension between the two groups. As expected, clinical grades were statistically different between the VS+ and VS− groups (Mann–Whitney *U*-test, $p < 0.01$).

Table 1
Clinical characteristics of 98 patients included.

Patient characteristics	VS+ group $n = 50$ (n, %)	VS− group $n = 48$ (n, %)
Sex		
Female	31 (62%)	34 (71%)
Male	19 (38%)	14 (29%)
Age		
(Years, mean ± SD)	52 ± 12	51 ± 11
Clinical grade (WFNS)		
I	8	28
II	14	12
III	8	1
IV	11	4
V	9	3
Treatment		
Early surgery (0–2 days after SAH)	34 (68%)	37 (77%)
Endovascular	16 (32%)	11 (23%)
Hypertension (past history)		
Presence	13 (26%)	11 (23%)
Absence	37 (74%)	37 (77%)

Values represent numbers of patients with percentages given in parentheses. VS, vasospasm; SAH, subarachnoid hemorrhage.