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# Neurocognitive Sequelae and Rehabilitation after Subarachnoid Hemorrhage: Optimizing Outcomes

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**Abstract:** Subarachnoid hemorrhage (SAH) is a medical emergency that requires immediate intervention. The etiology varies between cases; however, rupture of an intracranial aneurysm accounts for 80% of medical emergencies. Early intervention and treatment are essential to prevent long-term complications. Over the years, treatment of SAH has drastically improved, which is responsible for the rapid rise in SAH survivors. Post-SAH, a significant number of patients exhibit impairments in memory and executive function and report high rates of depression and anxiety that ultimately affect daily living, return to work, and quality of life. Given the rise in SAH survivors, rehabilitation post-SAH to optimize patient outcomes becomes crucial. The review addresses the current rehabilitative strategies to combat the neurocognitive and behavioral issues that may arise following SAH.

**Keywords:** subarachnoid hemorrhage (SAH); rehabilitation; neuropsychiatric; neurocognitive



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## 1. Introduction

Subarachnoid hemorrhage (SAH) is a devastating condition with high mortality and morbidity rates and is often accompanied by significant physical, behavioral, and neurocognitive comorbidities [1]. These comorbidities present acutely (within three months) and persist long-term (up to two years) post-SAH [2,3]. The behavioral and neurocognitive sequelae associated with SAH include psychological distress affecting mood and anxiety, post-traumatic stress disorder (PTSD), social dependence, fatigue, and alterations in sexual function [3,4].

Regardless of SAH severity, a significant number of SAH patients develop neurocognitive and behavioral psychosocial issues due to several mechanisms that may include: organic brain injury secondary to vascular disruption; the unmasking of underlying neuropsychiatric conditions; development of PTSD following hospitalization; and associated life stressors (e.g., unemployment, etc.) [5,6]. The present review discusses the neuropsychiatric and cognitive sequelae post-SAH. Furthermore, we highlight current evidence-based rehabilitative strategies to combat the neurocognitive and behavioral issues that may arise following SAH in the acute and chronic stages.

## 2. Neuropsychiatric and Neurocognitive Sequelae Following SAH

### *2.1. Depression*

Depression has been reported after SAH and appears to persist [7]. The frequency of depression following SAH is variable and depends on the timing of assessment following the SAH ictus. A recent meta-analysis by Tang et al. showed that the overall pooled frequency of depression was 26.3% post-SAH. Furthermore, the progression of depression from months to years in the SAH ictus also depended on the assessment tools utilized [8].

In another study, depressed mood occurred in 47% of patients during the first year of recovery post-SAH; notably, in the same study, non-Caucasian ethnicity was a risk factor for developing depression post-SAH [9]. Other premorbid conditions, including a prior history of mood disorders, tobacco use, alcohol use disorder, illicit drug use, chronic obstructive pulmonary disease, and non-English fluency, have been shown to increase the risk of depression following SAH [7,8]. Additionally, patients with posterior circulation aneurysm rupture have been shown to have significantly more problems with depression [10].

Depression significantly affects the patient's quality of life, employment, and functional outcomes following SAH [9,11]. The mechanism for depression post-SAH remains unclear; some studies have proposed a link between low basal cortisol levels and depression post-SAH [12,13]. Given this association, future studies are warranted to investigate the contributions of the hypothalamic-pituitary-adrenal (HPA) axis in post-SAH-associated depression.

Despite the prevalence of depression post-SAH, there is a paucity of data on the treatment of post-SAH depression. In the general population, selective serotonin receptor inhibitors (SSRIs) are the first-line antidepressant drugs used to manage depression [14]. Given the risk of intracerebral hemorrhage (ICH) with SSRIs within the first month of use, the optimal initiation of antidepressants while exploring other pharmacological and multimodal treatment strategies needs to be studied in SAH survivors [15]. Such options for multimodal therapy may include light and music therapy, motivational interviewing, transcranial magnetic stimulation, ecosystem-focused therapy, etc.

### *2.2. Anxiety*

Given the strong association between anxiety and depression, assessing anxiety in patients post-SAH is paramount. Similar to depression, the prevalence of anxiety post-SAH is dependent on the study tools utilized and the timing of the assessment. For instance, Barlet et al. showed a pooled anxiety prevalence of 32.2%, 19.2%, 40.5%, and 47.6% prevalence at 3, 6, 12, and 24 months, respectively, post-SAH. Using the State Trial Anxiety Inventory method, the overall increased anxiety burden post-SAH showed statistically increased anxiety symptoms of 39%, 41%, and 54% at 3 months, 1 year, and >2 years follow-up, respectively, post-SAH [16]. Passive coping strategies, unemployment at 6 months, and a prior history of a psychiatric disorder were associated with an increased risk of an anxiety disorder post-SAH [11]. Additionally, patients with posterior circulation aneurysm rupture have been shown to have significantly more problems with anxiety [10]. Further research is needed to delineate the brain circuits and neurochemical factors that perpetuate anxiety post-SAH.

### *2.3. PTSD*

PTSD post-SAH is prevalent and associated with poor quality of life despite relatively good clinical outcomes [17–19]. The prevalence of PTSD following SAH ranges from 18–37% [17,18]. The variability in prevalence arises from differences in the assessment tools utilized and the timing of assessment. Importantly, significant others/caregivers (e.g., spouses, etc.) of patients who survived SAH have been shown to have increased symptomatology of PTSD. This finding is important given that PTSD in this subgroup could interfere with effectively administering care to SAH patients [20,21].

While the underlying mechanism for PTSD following SAH remains unclear, some studies have suggested that PTSD post-SAH may result from the patient's adjustment to the experience of having had a SAH and the fear of recurrence [19,22,23]. Risk factors for PTSD post-SAH are similar to those for depression and anxiety. Notably, patients with a history of psychiatric disorders are more at risk of developing PTSD following SAH [17].

### *2.4. Sexual Dysfunction*

Brain injury can affect the way patients express their sexuality. Many patients following brain injury have reported reduced sexual drive, reduced arousal, impotence, or

increased compulsive sexual behaviors [24]. Sexual dysfunction as it relates to SAH is understudied; several studies have shown that both men and women report sexual dysfunction and dissatisfaction following stroke [25–27]. These findings become relevant when caring for patients post-SAH, since SAH can cause stroke secondary to hemorrhagic ischemia [28]. In another study, 9 of 19 women (47%) reported having sexual dysfunction according to the female sexual function index (FSFI) following an aneurysmal SAH (aSAH). Interestingly, all 19 of the women reported having hypoactive sexual desire disorder. The authors also noted that 7 of 14 men had erectile dysfunction using the International Index of Erectile Function (IIEF) [4]. While the exact mechanisms responsible for sexual dysfunction post-SAH remain unclear, some studies have suggested that hypothalamo-pituitary dysfunction may contribute to impaired sexual function post-SAH [29,30].

### *2.5. Cognitive Dysfunction*

Cognitive dysfunction is a common issue following SAH due to the diffuse brain injury that occurs during the initial insult. Various cognitive domains may be affected, including memory, language, spatial processing, and executive function, even if patients appear relatively clinically normal at discharge [31]. Furthermore, many of these complications are long-term and continue to affect patients in the months following discharge, appearing to have a significant impact on quality of life and return to work [32–34]. In a retrospective study, 94.6% of patients evaluated 3 months post-SAH had at least one cognitive deficit, the most common being memory [35]. Despite the high probability of cognitive deficits in the SAH population, these complications go underreported [36].

## 3. Rehabilitation after Subarachnoid Hemorrhage in the Acute Setting

The diagnosis of SAH from a ruptured aneurysm or traumatic cause is typically established in the emergency setting. Once this is established, care is transitioned to a neurosurgical team, if necessary and not contraindicated, and then to a neurocritical care unit (neuro ICU). Patients may present with various neurological symptoms, but the most common is a severe headache refractory to home treatment. While only 1% of headaches presenting in the emergency department (ED) are attributed to subarachnoid hemorrhage, the mortality rate of SAH may be as high as 65%. Early diagnosis in the ED and prompt patient stabilization are vital to reducing mortality [37]. This section briefly discusses the management of SAH in the acute setting and rehabilitative strategies that improve patient outcomes.

### *3.1. Fluid and Electrolyte Management*

An initial primary goal in the neuro ICU is proper fluid management to prevent secondary cerebral injury due to poor cerebral perfusion and inadequate cerebral oxygenation. Volumetric status is typically aimed at euvolemia with constant hemodynamic monitoring, such as central venous pressure, pulmonary wedge pressure, and fluid balance [38]. Fluid status is generally achieved via a mean intake of 3–4 L per day of crystalloids [39]; however, the choice of crystalloids is currently being evaluated in a current clinical trial (NCT04043598). Current and past clinical trials have also evaluated more aggressive fluid strategies such as hypervolemia, steroid-based therapies as opposed to crystalloids, and others; however, there has been no evidence to suggest any benefit over the current fluid management strategies [39]. Some studies suggest that hypervolemic goals increase the risk of complications and result in poorer outcomes [40].

Fluid status is also important in preventing other post-SAH metabolic-related complications such as hyponatremia, anemia, and hypo- or hyperglycemia [41–43]. Hyponatremia is an especially common complication and can lead to further adverse events such as delayed cerebral ischemia and symptomatic vasospasm [40]. The current guidelines for treating hyponatremia recommend using hypertonic saline and/or steroid-based therapies to correct the electrolyte imbalance and avoid free water and fluid restriction [40]. However, care must be taken to prevent rapid correction of the sodium imbalance so as to prevent

central pontine myelinolysis [44]. Clinical trials have evaluated the use of ADH receptor antagonists, such as conivaptan, but have been unsuccessful due to adverse effects from rapid imbalance correction [45]. The guidelines for treating anemia and hypoglycemia are well established and involve the use of packed red blood cells and glucose, respectively [40].

### *3.2. Nutritional Rehabilitation*

Nutritional goals have also proved vital in the acute setting. The international guidelines for nutrition in the critically ill are typically followed, as there are no defined guidelines for SAH at the time of this review [46,47]. These current guidelines suggest that an oral diet should be maintained if the patient is able to eat and cover at least 70% of their nutritional needs [46]. If the patient is unable to eat, enteral nutrition (EN) or parenteral nutrition (PN) should be started promptly. EN is typically preferred in SAH patients, but many studies suggest this route does not meet the nutritional requirements and may increase the risk of adverse events [48,49].

Further studies have recommended the initiation of supplemental PN if EN cannot meet the caloric requirements needed to reduce mortality in critically ill patients [50]. Higher protein intake has also been shown to decrease mortality and lead to favorable outcomes [51]. Based on the current data, nutrition should be addressed early through the use of both EN and PN to achieve adequate caloric and protein intake to reduce complications and mortality.

### *3.3. Early Mobilization and Cognitive Rehabilitation*

Early mobilization has been addressed in the acute setting post-SAH. A prospective, interventional study compared SAH patients who received standard mobilization therapy with those who received early mobilization therapy. Karic et al. found that early mobilization did not increase complications, and other adverse events, such as thromboembolic events or even death, were similar between the groups. Interestingly, the risk of severe vasospasm decreased by 30% in the early rehabilitation group [52]. A retrospective study compared early and late mobilization groups and found that patients in the early mobilization group had earlier discharges to home with better functional status at discharge [53]. Lastly, a randomized clinical trial (RCT) compared early mobilization within 24 h to the current standard of care for acute ischemic or hemorrhagic stroke and found fewer patients in the early mobilization group had favorable outcomes at 3 months [54]. Based on the current data, early mobilization and rehabilitation in SAH patients may be feasible, but more studies primarily focusing on SAH patients are needed to determine the effect on outcomes precisely.

Early mobilization may also influence the improvement of cognitive function following SAH. Considering the impact cognitive deficits have on the quality of life following SAH, very little information exists on the benefit of focused cognitive rehab in the acute setting aside from the cognitive benefits shown as a result of early mobilization [36]. Furthermore, the timing of when to initiate early cognitive rehabilitation should also be considered and studied.

Neuropsychological tests may be used to determine the specific deficits, and the recommendation for which tests are ideal varies between studies [31,55]. Neuropsychological tests routinely used in the acute setting following SAH include the modified Rankin Scale, the Montreal Cognitive Assessment, and the MMSE; however, these tests poorly predict minor cognitive deficits. Specific neuropsychological tests, such as the CVLT-II, the Ray Osterich Complex Figure Tests, etc., for memory, may provide more sensitivity to better understand the extent of cognitive deficits and the cognitive domains affected, such as memory, learning, language, spatial awareness, etc. [31].

Once specific cognitive deficits in SAH patients in the acute setting are determined via neurocognitive testing, decisions on cognitive rehabilitation should be promptly initiated. Initiating cognitive rehabilitation too early or delaying it may hinder success and lead to suboptimal outcomes [56]. Rehabilitation should also be tailored to the specific deficit

identified. Technology may be utilized as an option to initiate cognitive rehabilitation quickly. An RCT by Chen et al. recently studied the early initiation of cognitive rehabilitation using tablets and found the feasibility of using technology to help address cognitive issues in the acute setting. However, a limitation of this study was the small sample size of participants [57].

The current data utilizing cognitive rehabilitation in the acute setting is limited despite well-established cognitive deficits during this time frame. With the current trend of cognitive rehabilitation being initiated more in the sub-acute or chronic period, further studies into the initiation of cognitive rehabilitation in the acute setting and its effect on long-term quality of life are warranted.

### *3.4. Multidisciplinary Care*

Considering the intensive care required and the wide range of complications that may arise in the acute setting of SAH, a multidisciplinary approach to care should be utilized. The primary teams involved in the initial diagnosis and treatment of SAH may include the ED teams, radiology/interventional radiology teams, neurosurgical teams, anesthesia teams, and neurocritical care teams. Treatment should be managed in a large facility with specialized neurological intensive care units to adequately provide the specialties needed for the immediate treatment of SAH. Following the early stages of care, several clinical specialties may also be consulted for optimal care [58]. Consulting specialties involved in fluid management and metabolic concerns in the acute setting may include cardiopulmonary specialists, endocrinologists, and nephrologists to address electrolyte and fluid abnormalities, as well as hematologists to address anemia concerns [59–62]. Dieticians and nutritional specialists may be involved in the nutritional aspects of care provided in the neuro ICU [63]. Several specialties may also be utilized for the mobilization and cognitive issues seen in SAH patients, including physical therapists, physiatrists, neuropsychologists and therapists, and psychiatrists [64–66]. While not all specialists listed may be directly involved, SAH patients benefit from a multidisciplinary approach and are more likely to be discharged home with fewer long-term complications [67–69].

## 4. Long-Term Rehabilitation Following Subarachnoid Hemorrhage

Advances in surgical techniques and medical management have led to a steady decrease in the mortality rate following SAH. Between 1980 and 2005, there was a 50% decrease in mortality rate and an absolute annual reduction of 0.9% in 30-day mortality post-SAH [70]. The improvement in SAH mortality has led to more survivors needing long-term treatment strategies to reintegrate into society. It is reported that one-third of these survivors have a reduced quality of life one year post-SAH ictus [71]. These data indicate a clear need for a multifaceted treatment plan following SAH to optimize long-term patient outcomes.

### *4.1. Long-Term Outcomes Following SAH*

Following SAH, only 55% of patients regain functional independence, while 19% remain dependent, and 26% ultimately die [72]. Post-SAH, a high proportion of patients reported impairments in memory, executive function, and language that led to the deterioration of activities of daily living, return to work, quality of life, and instrumental activities of daily living [5]. One study showed that more than half of patients deemed to have had good neurological outcomes one year post-ictus experienced mild to moderate difficulties with reintegration 20 years post-SAH. With this, their general quality of life was not affected [73]. It was reported that 94.6% of patients with moderate to good physical recovery living at home post-SAH had at least one cognitive or emotional complaint that impeded day-to-day life [35]. Interviews with patients about discharge in a retrospective study showed that patients discharged directly from neuro ICUs felt abandoned, found it challenging to find the appropriate support, and were confused about rehabilitation. On the other hand, patients referred to a rehabilitation clinic stated that they felt supported and

informed and described the process as organized. In general, these patients emphasized the need for multidisciplinary follow-ups, which were largely missing from their long-term treatment plans [74].

### *4.2. Determinants of Health Post-SAH*

Managing the long-term ailments associated with SAH requires a functional understanding of the determinants associated with these ailments. Several studies utilize the health-related quality of life (HRQoL) to determine outcomes post-SAH [75]. Some factors that determine poor HRQoL in patients post-SAH include the severity of SAH, fatigue, mood problems, physical disability, cognitive complaints, passive coping, female gender, and neuroticism [16,75,76]. Additionally, it has been found that passive coping, living alone, unemployment, restrictions in leisure, and poor HRQoL are determinants of anxiety and depression in the long-term following SAH [16,77]. These determinants should be considered when designing long-term rehabilitation plans for SAH patients, as they mitigate many cognitive and behavioral issues post-SAH.

Returning to work is an essential milestone in long-term SAH recovery and is associated with good neurological outcomes [78]. Only one-third of all patients post-SAH return to work at full capacity [34,79]. A failure to return to work is associated with lower quality of life, depression, insomnia, panic, fatigue, and anxiety [79–82], whereas successfully returning to work is associated with higher satisfaction levels, self-care ability, and financial stability [79]. The factors that affect the success of returning to work are depression, state anxiety, and trait anxiety [83]. More recently, it has been argued that returning to driving should be used as a long-term milestone in SAH recovery. Lai et al. argue that return to work is not a reliable predictor of a patient's status because of diverse determinants such as age, socioeconomic status, type of occupation, and complex psychosocial factors associated with returning to work. Additionally, return to driving is a more measurable outcome and is strongly associated with return to work, a positive neurological outcome, mobility, and return to daily living activities [78]. Notably, an equal amount of attention must be placed on early screenings and interventions in vocational rehabilitation as well as driving rehabilitation in patients post-SAH.

Significant others or primary caregivers of patients who suffer from SAH are often overlooked in long-term treatment plans. Significant others have a substantial drop in quality of life post-SAH and disruption in psychosocial domains such as social interactions, work, recreation, and emotional behavior [84]. Additionally, they have increased rates of anxiety and depression compared to a control population [21]. Covey et al. analyzed the fear of recurrence of SAH in patients and their significant others and found that the fear of recurrence in the significant others is higher than that of the patients. This fear of recurrence in the significant other was found to impede recovery as it hindered the patient's work/daily activities, social activities, and health ratings, leading to a decreased HRQoL within the patient [85]. Significant others should not be left out of the picture while developing a model for long-term care. Support measures should be given to ensure that family members of patients are adjusting to life post-SAH.

Support groups may also be beneficial to patients post-SAH. Support groups can act as a mechanism to detect psychological distress early so that patients can be referred to a proper healthcare professional. To our knowledge, only one study has addressed the use of support groups post-SAH. Noble et al. found that psychological distress went mostly undetected within their sample population prior to initiating support groups. Additionally, the authors found that their established support groups drew in distressed patients 2.5 times more than the average post-SAH population. This support group also had a population that had higher rates of anxiety, depression, and PTSD than the average post-SAH population [86]. There is a paucity of data analyzing the outcomes of support groups or the support groups of significant others post–SAH; thus, further studies are needed to elucidate these outcomes.

### 4.3. Novel Rehabilitative Strategies and Future Directions

New rehabilitative strategies have been explored as novel treatment modalities for individuals recovering from SAH. Recent studies indicate that hyperbaric oxygen therapy, most commonly used for decompression sickness, may inhibit cerebral vasospasm by mitigating inflammation. Through this mechanism, hyperbaric oxygen is thought to reduce post-SAH edema, increase cerebral blood flow, and minimize infarct size. Limitations to using hyperbaric oxygen include access to hyperbaric oxygen chambers and time for treatment. Studies indicate that delayed treatment may worsen cerebral ischemic injury. Future studies are warranted to determine the external factors that may influence the effectiveness of hyperbaric oxygen therapy, such as advanced age, gender, and underlying hypertension [87].

Neuromodulation is a relatively new therapy currently being considered for use in the recovery period following SAH. Neuromodulation is thought to target inflammatory markers released during the ischemic insult associated with SAH. Recent data has suggested that transcranial direct stimulation (tDCS), a specific type of neuromodulatory therapy, may be therapeutically beneficial post-SAH by inducing alterations in cortical excitability and perfusion. In a rodent study, tDCS therapy implemented 3 and 4 days post-SAH reduced the frequency and severity of vasospasm. This encouraging finding suggests that stimulatory therapy may provide further therapeutic benefit to patients recovering from SAH and could be recommended in addition to pharmacological therapy to prevent complications such as vasospasm post-SAH [88].

Like tDCS, trigeminal nerve stimulation (TNS) has been considered to improve post-SAH circulation and prevent delayed cerebral ischemia due to vasospasm and circulatory dysregulation. Shah et al. showed that TNS increases blood vessel lumen diameter, specifically in the internal carotid artery, middle carotid artery, and anterior cerebral artery in rodents post-SAH. As arterial vasospasm is one of the most common complications post-SAH, this increase in vessel diameter may be protective against this complication [89]. While most of the beneficial effects of neuromodulation are appreciated in preclinical studies, two smaller post-SAH clinical studies with cervical transcutaneous electrical neurostimulation (TENS) and tDCS have shown promising results in improving gait imbalance and cerebral vasospasm [90,91].

Despite a non-severe SAH severity, patients may still experience impairments in several domains, which stresses the need for continued long-term support [73]. Information sessions for patients and families about the rehabilitative process and continued follow-up in outpatient clinics should be encouraged prior to discharge [74,92]. The many problems patients face post-SAH emphasize the need for long-term multidisciplinary care. Multidisciplinary care teams may include a primary physician (neurologist or neurosurgeon), a dedicated stroke nurse, a rehabilitation physician, a mental health specialist, an occupational therapist, and a physical therapist [92,93]. Within the clinic, patients should receive physical, cognitive, emotional, return to work, and return to driving screenings regularly to assess milestones (see Figure 1 for the proposed rehabilitation paradigm post-SAH) [35,75]. There is little to no data on mobilization and nutrition strategies $\geq$ 3 months post-SAH. This area of research should be explored further to improve patient outcomes.

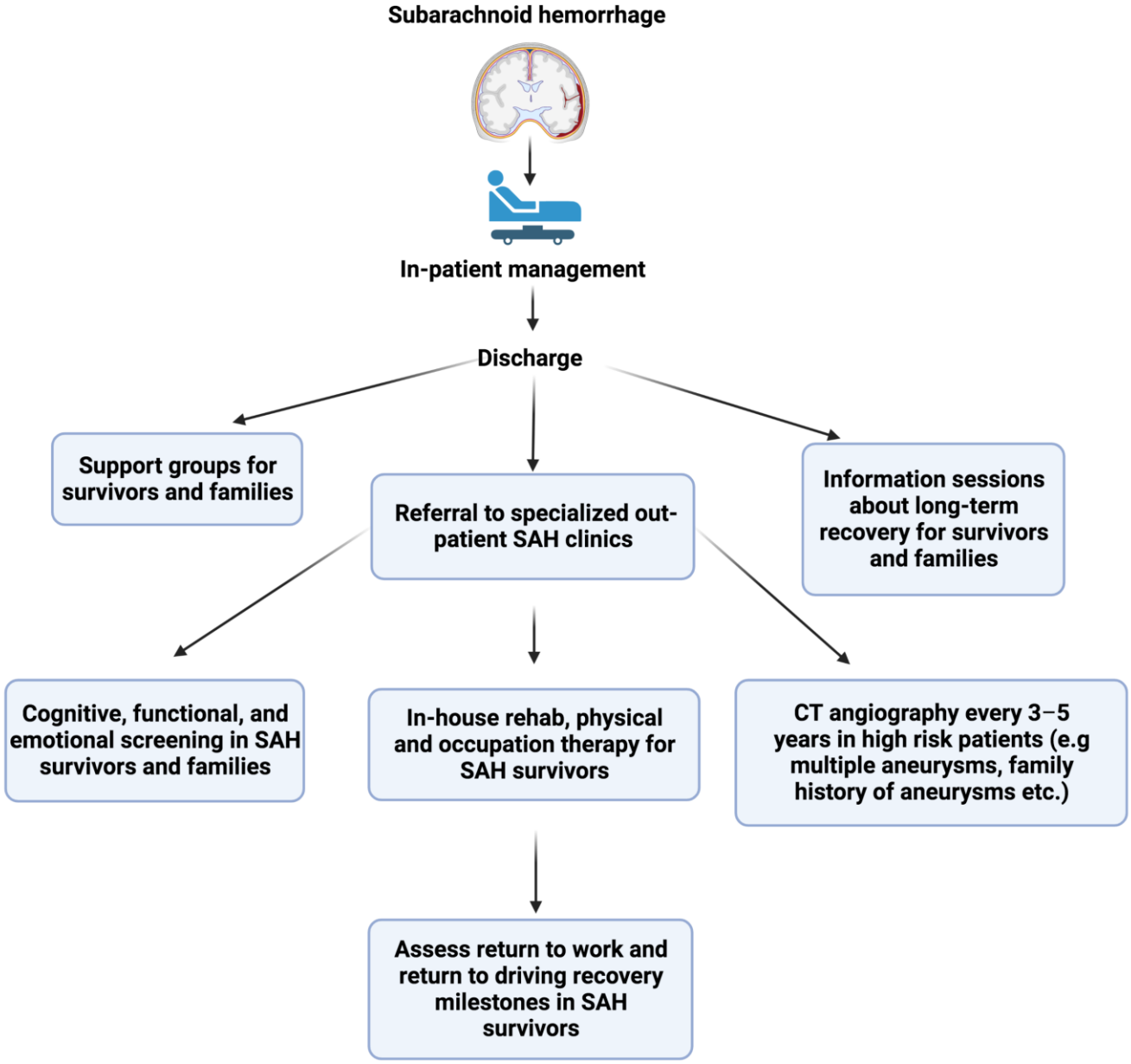


**Figure 1.** The proposed rehabilitative paradigm for SAH patients. Image credit: Biorender.

## 5. Subarachnoid Hemorrhage Prognostic Biomarkers and Imaging Modalities

Ongoing research is being conducted to identify neurological prognostic biomarkers for SAH patients. Many studies have proposed neuro-specific markers in humans that include S100B, glial fibrillary acid protein (GFAP), neuron-specific enolase (NSE), microtubule-associated protein tau (MAPT), neurofilament-light (NF-L), amyloid-B-protein, apolipoprotein E (ApoE), and ubiquitin C terminal hydrolase 1 (UCHL1) [94]. An increase in almost all markers indicated poorer outcomes except for Amyloid-B-protein and ApoE, in which reductions indicated poorer outcomes (Table 1).

**Table 1.** Prognostic biomarkers and outcomes correlation in the setting of SAH.

| Biomarker | Comments | Study References |
|---|---|---|
| S100B | Increased; correlated to poorer outcomes in the acute setting. | [95,96] |
| NSE | Increased; correlated to poorer outcomes such as increased mortality and vasospasm. | [97,98] |
| GFAP | Increased; correlated to higher mortality and poor functional outcomes. | [99,100] |
| MAPT | Increased; correlated to poorer outcomes in the acute setting, increased hypoxic brain injury, and poor functional outcomes at 12 months. | [95,101] |
| NF-L | Increased; correlated to early cerebral ischemia and poor neurological outcome at 6 months. | [102,103] |
| UCHL1 | Increased; sub-acute period correlated with increased neuronal loss and poorer outcomes. | [104,105] |
| Amyloid-B-protein | Decreased; correlated to poorer long-term functional outcomes. | [106,107] |
| ApoE | Decreased; correlated to increased inflammation and poorer outcomes. | [107,108] |

Abbreviations: glial fibrillary acid protein (GFAP), neuron-specific enolase (NSE), microtubule-associated protein tau (MAPT), neurofilament-light (NF-L), apolipoprotein E (ApoE), and ubiquitin C terminal hydrolase 1 (UCHL1).

Several imaging modalities are also currently being studied as potential prognostic tools to determine outcomes after SAH. Several studies have identified findings that correlate with poorer outcomes in SAH aside from the initial prognostic indicators, such as the size of the hemorrhage. A study by Fragata et al. found that observed increases in apparent diffusion coefficients (ADC) and decreases in fractional anisotropy (FA) correlated with poor functional outcomes following SAH [109]. Another study by Abdel-Tawab et al. found that an increase in mean transit time (MTT) in computed tomographic perfusion (CTP) correlated with an increase in delayed cerebral ischemia and poorer outcomes [110].

Following SAH, there is an increased risk for recurrence [92]. One study compared life expectancy, quality-adjusted life years (QALY), hemorrhage recurrence, and costs associated with the screening strategies. Screening was found not to be cost-effective post-SAH every five years and reduced QALY [111]. The data supported a benefit from CT angiography screening every 3–5 years in post-ictus patients who are female, were young at ictus ($\leq$35–40 years old), had multiple aneurysms, had a family history of SAH or autosomal dominant polycystic kidney disease, and had a decreased QoL due to fear of recurrence [92,111]. Beyond that, it is generally not recommended to mandate screening but to allow the patient to decide after being fully educated on the risks and benefits of screening [92]. While imaging modalities are well-defined for diagnosis, further research into the prognostic value of extensive imaging and biomarkers could provide increased insight into the functional outcomes of SAH patients and help better guide treatment decisions earlier in the course of care.

Although there are no current guidelines for prognostic biomarkers and imaging modalities at the time of this review, there are well-defined prognostic scales currently in use for SAH patients. A common prognostic scale is the Glasgow Coma Scale (GCS), which evaluates eye opening, motor, and verbal responses. Derivatives of the GCS include the World Federation of Neurological Surgeons Committee Scale (WFNS) and the Prognosis on Admission of Aneurysmal Subarachnoid Hemorrhage (PAASH). Both scales show similar predictability in outcomes for SAH patients [112]. The Hunt and Hess grading system is also frequently used. The Hunt and Hess scale classifies SAH in grades 1 to 5 based on presenting symptoms; however, this grading scale has been scrutinized due to its ambiguity and its utility for use in prognosis [113,114]. The Fisher and modified Fisher scales (mFs)

are also used and are based on CT findings at the time of admission [115]. These scales only apply to vasospasm risk rather than clinical outcomes. The VASOGRADE scale was recently developed using a combination of the WFNS and mFs to predict better delayed cerebral ischemia (DCI) and provide insight into poorer acute functional outcomes [116]. The Ogilvy and Carter scale was introduced to predict surgical outcomes. The Ogilvy and Carter scale is based on the Hunt and Hess scale, the Fisher scale, and other factors such as the age and size of the aneurysm [117].

Newer scoring models have also been introduced, such as the Poor-grade Aneurysmal Subarachnoid Hemorrhage Prognostic Scoring System (PASHPSS), which integrates mFs, WFNS, age, conservative treatment, DCI, shunt-dependent hydrocephalus, and cerebral herniation [118]. The PASHPSS scale showed promising results in prognostic prediction but has yet to be clinically implemented or further evaluated. Another newer scoring model includes the "TAPS" model, which incorporates mFs, age, WFNS, the Graeb score of intraventricular hemorrhage, white blood cell count, and surgical clipping parameters [119]. The "TAPS" model showed positive results in prognostic predictability and is currently enrolled in a clinical trial to validate these findings (NCT04785976). While these scales exist and are in use clinically, there is much variability in their use by each institution. At the time of this review, there is no standardized prognostic scale solely for SAH, and further research is required.

## 6. Conclusions

The advancement of SAH treatment means more patients now survive SAH; however, the SAH survivor population is burdened by a high prevalence of neuropsychiatric and cognitive impairments that impact recovery, social and physical independence, and the ability to return to work. Thus, serious attention and rehabilitative strategies are crucial in addressing these treatable medical issues that the SAH survivor faces. Further research into the best rehabilitative approach is also needed to provide effective mechanisms that optimize outcomes post-SAH.

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