Manuscript

**Psychological distress after Subarachnoid Haemorrhage: A systematic review and meta-analysis**

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***Abstract***

**Objective:** Psychological distress is a common complication in patients after Subarachnoid haemorrhage (SAH) which often has significant impact on the prognosis. The objective of this study was to determine the pooled prevalence of anxiety symptoms and depressive symptoms in patients after SAH and identify relevant risk factors.

**Methods:** The study adopted a systematic review and meta-analysis protocol. Multiple databases including EMBASE, Medline, PsychInfo, and Web of Science were searched for publications before 1st January 2020. Screening, data extraction, and quality assessment were undertaken following the PRISMA guidelines for preferred reporting of systematic reviews and meta-analysis. The random-effects model was used to calculate pooled prevalence rates. Meta-analysis was conducted using Comprehensive Meta-analysis software. The review protocol was registered on PROSPERO (CRD42020182594).

**Results:** 42 studies reporting anxiety symptoms and 64 studies reporting depressive symptoms were included. The pooled short term(<3 years) and long term(≥3 years) prevalence rates of anxiety symptoms were 31.4%(95% CI: 23.6%, 40.4%) and 40.4%(95% CI: 31.6%, 49.8%), respectively, whereas the pooled short term and long term prevalence rates of depressive symptoms were 25.2%(95%CI:17.8%, 34.5%) and 35.8%(95%CI:28.6%, 43.6%), respectively. Gender and pre-existing psychiatric conditions were identified as potential risk factors.

**Conclusions:** The high prevalence of anxiety symptoms and depressive symptoms after SAH highlights the need for appropriate assessment and management of psychological stress in patients after SAH. Further research is warranted to explore potential underlying mechanisms and to develop holistic interventions that incorporate understanding of both the biological and psychological impact of SAH.

***Main text***

**1 Introduction**

Spontaneous subarachnoid haemorrhage (SAH) is a devastating condition, with high rates of death and disability98. It is most often caused by rupture of a brain aneurysm. With improving treatment there are increasing numbers of patients surviving with what is graded as a good outcome on conventional outcome scales. However, these scales severely underestimate psychological distress and cognitive dysfunction which lead to extensive problems reintegrating back into society 99-101.

 Psychological distress commonly refers to the individualized patient response to illness among which anxiety symptoms and depressive symptoms are most commonly reported as long-term complications of SAH1,3. Previous work by Morris and colleagues reported approximately 40% of patients had moderate to severe levels of anxiety symptoms 16 months after SAH, whereas approximately 20% experienced moderate to severe levels of depressive symptoms1. These psychological distresses can persist for long periods of time after SAH and have negative impact on quality of life2, 3, 4, even in those who have made a full neurological recovery5. The largest study conducted in the UK via SAH support groups with 414 participants, reported that around 77.5% SAH patients experienced psychological distress – both anxiety symptoms and depressive symptoms, an average of 3 years after SAH8. Anxiety symptoms are the most commonly reported psychological distress. This frequently relates to fears over haemorrhage recurrence 4, or fears about their ability to return to everyday life 6 which are based on their own beliefs about SAH treatment and prognosis. We have noted these sentiments in qualitative work with patient groups and development of SAH outcome scales. 114 Commonly quoted fears are “will it happen again?” or “Who will look after my family if I can’t”. Studies have also reported an increased prevalence of depressive symptoms and insufficient coping alongside a reduced quality of life in SAH survivors 7. In addition, personality trait and psychiatric morbidity prior to SAH35 may also contribute to psychological distress after SAH. Leventhal's common-sense model may be a useful framework for eliciting and understanding patients’ beliefs about SAH and treatment and how they contribute to changes in outcomes over time105.

 One potential major biological risk factor for developing psychological distress after SAH is neuroinflammation. While inflammation plays a key role in brain injury and poor outcomes after SAH, growing evidence also supports the neuroinflammatory pathway underlying both depression and anxiety9-11, 102-103 . Therefore, the initial, acute neuroinflammatory response in SAH may be a significant predisposing factor to psychological distress as inflammatory cytokines can signal the brain to cause neurotransmitter disturbances, hypothalamic-pituitary-adrenal axis dysfunction, and neuroplasticity changes which can affect mood and behaviour104. Therefore, SAH acting as both a physiological and a psychological trauma predisposes individuals to both short- and long-term psychological distress12-15.

 Although there is a large literature reporting the prevalence of psychological distress, studies have produced disparate ﬁndings and little consensus. There has been no substantial review into the published data and there is no examination of risk factors associated with it. Therefore, the current study aimed to separately evaluate the pooled prevalence of anxiety symptoms and of depressive symptoms among patients after SAH. In addition, potential risk factors linked to these psychological distresses were studied.

**2. Methods**

Methods for this systematic review followed the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA)16 recommendations. The review protocol was registered on PROSPERO (ref: CRD42020182594)

**2.1 Search strategy:**

The electronic databases of EMBASE, Medline, PsychInfo and Web of Science were used to select relevant studies published before 1st January 2020. The Population, Intervention, Comparison, Outcomes and Study (PICOS) framework was used to guide the search strategy. The search terms used included “subarachnoid haemorrhage OR SAH” and “anxiety” and “depression OR depressive symptoms”. We also examined reference lists of eligible articles in order to identify additional relevant studies. Two reviewers (MB and Quang-Vinh Banh) screened paper titles and abstracts using the Rayyan systematic review app developed by the Qatari Computing Research Institute17 which provided a platform to include and exclude papers with ease. The majority of papers MB and QVB agreed upon however, differences were discussed and resolved between MB and RH.

**2.2 Inclusion and exclusion criteria for study selection**

Studies were included based on the following inclusion criteria: 1) cohort, case-control, cross-sectional or case reports with at least 10 cases. 2) reported numbers of study populations of SAH and those who developed symptoms of anxiety or/and depression after SAH, as well as reported measures of anxiety or/and depression. 3) included individuals over the age of 18 who met the diagnostic criteria for SAH. 4) were published in English language. 5) method or criteria for assessing anxiety or/and depression was a clinical diagnosis or standardized questionnaire. After title and abstract screening, full texts were read and checked to assess eligibility.

**2.3 Data extraction**

Data were extracted using a pre-piloted structured form. Studies were grouped according to their respective searches for “anxiety” and “depression”. Bibliographic data; key study characteristics – including study design, sample size and mean age of participants, aetiology of SAH, severity of SAH, interventions, follow up time points, tools used to measure anxiety symptoms/depressive symptoms; results and the methodological quality of the study were extracted from each study.

**2.4 Quality assessment and critical appraisal**

After reviewing recommendations for observational studies18-20, the one developed by Hawker et al., 2002 21 was adopted for this particular review, as it provides flexibility to assess the quality of a range of studies – cohort, case-control and cross-sectional – which are all represented in this review. The tool is based on a scoring system where nine components of the paper are allocated points on a scale of good – very poor. The nine components assessed are – abstract and title, introduction and aims, method and data, sampling, data analysis, ethics and bias, results, transferability, implications and usefulness 21. The scoring system was defined as – ‘good’ providing 4 points while ‘very poor’ provides 1 point. This produces a combined score for a paper between 9 and 36 points – with the higher the score the better the quality of the paper. Despite the development of this tool, Hawker et al., did not define ‘quality grades’ and as such, the grades outlined and applied by Lorenc et al.22 were used: high quality (A), 30-36 points; medium quality (B), 24-29 points, low quality (C), 9-24 points. The risk of bias of each study was analysed and included as part of the quality assessment tool. In addition, funnel plot of standard error by Hedges’g was used to indicate any potential publication bias.

**2.5 Data analysis and synthesis**

Not all studies provided measures of anxiety symptoms and depressive symptoms at the same time points. Therefore data were synthesized based on specific, specified time points. The prevalence of anxiety symptoms and depressive symptoms was calculated at 3 months, 6 months, 1 year, 2 years and 3 or more years. Follow up was further dichotomised into short term (less than 3 years) and long term (3 years or more). The pooled prevalence statistics were calculated using the random effects model by the Comprehensive Meta- Analysis software23. 95% confidence intervals were also calculated. I2 test was used to indicates the percentage of variance in the meta-analysis that is attributable to study heterogeneity, with 25%, 50% and 75% considered to indicate low, medium and high heterogeneity respectively. Potential risk factors linked to the prevalence of anxiety symptoms or depressive symptoms were identified and summarised.

**3. Results**

**3.1 Study selection**

Our search strategy yielded an initial 542 papers relating to anxiety symptoms after SAH and 1196 papers for depressive symptoms after SAH. After abstract and title screening and duplicate removal, this was reduced to 78 papers and 97 papers respectively and the subsequent full text articles were retrieved. After full text screening, a total of 42 papers relating to anxiety symptoms and 64 papers relating to depressive symptoms were included in this review. 36 papers were in the results for both the anxiety and depression searches but were considered and extracted separately in order to minimise risks of bias during data extraction24. Figures 1a and 1b provide PRISMA flow charts16 outlining the search strategy and the study selection process.

**3.2 Anxiety symptoms**

Forty-two studies involving a total of 5950 patients (mean age 51.8 years) reporting anxiety symptoms in SAH were included in this analysis. The characteristics of these studies are presented in Table 1.

**3.2.1 Prevalence of anxiety symptoms – short term**

Five studies reported anxiety symptoms 3 months after SAH. Ackermark et al25 had the highest reported number (51.6%) of individuals with anxiety symptoms after SAH, while Powell et al.5 had the lowest, with 15.3%. The pooled 3-month prevalence of anxiety disorders was 32.2% (95% CI: 18.8%, 49.3%). In comparison, 6 studies reported patients suffering with anxiety 6 months after SAH. Von Vogelsang et al.26 had the highest number (59%) while Fontanella et al.27 had none. The pooled 6-month prevalence of anxiety symptoms was 19.2% (95% CI: 6.5%, 44.9%). At the 1 year follow up time point, 6 studies reported anxiety symptoms after SAH. Von Vogelsang et al.26 had the highest number (55.6%), with Hellawell et al.28 reporting 6.8%). The pooled prevalence at 1 year was 40.5% (95% CI: 35.3%, 46%). Finally, 6 studies reported patients with anxiety symptoms at 2 years follow up, with the highest prevalence reported by von Vogelsang et al.,29(60.2%).The pooled 2-year prevalence of anxiety symptoms after SAH was 47.6% (95% CI: 35.8%, 59.6%). When considering all studies reporting anxiety under 3 years, the pooled prevalence was 31.4% (95% CI: 23.6%, 40.4%) (see Figure 2a).

**3.2.2 Prevalence of anxiety symptoms – long term**

Due to the amount of variation in follow up time points after 3 years, and the perception that there is relatively little change in condition of patients after this timepoint it was decided that studies reporting anxiety symptoms after 3 years would be grouped together. In total, 9 studies reported anxiety symptoms at time points between 3 years and 10 years, with a mean follow up time of 6.47 years. Persson et al.30 reported the highest proportion of patients suffering from anxiety symptoms (76.9%) while Dulhanty et al.,31 reported the lowest (23.6%) . The pooled long-term prevalence of anxiety symptoms after SAH is 40.4% (95% CI: 31.6%, 49.8%) (see Figure 2b).

 The meta-analysis revealed high heterogeneity across studies reporting short-term anxiety symptoms (Tau2 = 0.772, df = 22, p < 0.0001; I2 = 92%) and long-term anxiety symptoms (Tau2= 0.285 ; df= 8 (p< 0.0001); I2= 91%), respectively.

**3.3 Depressive symptoms**

Sixty-four studies involving a total of 8834 patients (mean age of 52.2 years) reporting depressive symptoms after SAH were included in this review. Following data extraction, the characteristics of these studies are presented in Table 2.

**3.3.1 Prevalence of depressive symptoms – short term**

Only three studies reported depression or depressive symptoms 3 months after SAH. Ackermark et al.25 reported the highest prevalence of 38.7% and Powell et al.32 the lowest (8.1%. The pooled prevalence of depressive symptoms at 3 months after SAH was 16.2% (95% CI 4.7%, 43.3%). At 6 months, 5 studies reported depressive symptoms, with the study by Von Vogelsang et al.29 reporting the highest prevalence (67%), while Hellawell et al.28 reported the lowest (4.5%). The pooled prevalence of depressive symptoms at 6 months was 26.7% (95% CI 7.8%, 60.9%). Nine studies reported depressive symptoms at 1 year after SAH. Boerboom et al.,33 reported the highest (77.6%) while Hellawell et al.28 the lowest (4.5%). The pooled prevalence of depressive symptoms 1 year after SAH at was 27.5% (95% CI 15.1%, 44.9%). Seven studies reported depressive symptoms 2 years after SAH. Von vogelsang et al.29 reported the highest number of patients reporting depressive symptoms (65.9%), while Al Yassin et al.14 reported the lowest (3%). The calculated pooled prevalence for depressive symptoms 2 years after SAH was 24.6% (95% CI 11.2%, 45.6%). When considering all studies reporting depressive symptoms under 3 years, the pooled prevalence was 25.2% (95% CI: 17.8%, 34.5%) (see Figure 3a).

**3.3.2 Prevalence of depressive symptoms – long term**

Similar to the long-term anxiety symptoms results, studies looking at the levels of depressive symptoms at time points over 3 years after SAH were considered together. 15 studies reported results at time points ranging from 3.25 years to 12 years with an overall mean follow up time of 6.12 years. Persson et al.3 had the fewest patients reporting depression or depressive symptoms (16.6%), while Boerboom et al.34 had the highest (78.9%. The pooled prevalence of depressive symptoms over 3 years after SAH was found to be 35.8% (95% CI 28.6%, 43.6%) (see Figure 3b).

 The meta-analysis revealed high heterogeneity across studies reporting short-term depressive symptoms (Tau2 = 1.111, df = 23, p < 0.0001; I2 = 96%) and long-term depressive symptoms (Tau2= 0.335 ; df= 14 (p< 0.0001); I2= 90%), respectively.

* 1. **Risk factors related to anxiety and depression after SAH**

Limited data was presented for risk factors for anxiety symptoms or depressive symptoms. 3 studies25, 26, 35 suggested female gender is a risk factor for developing anxiety symptoms after SAH whereas 2 studies36, 37 stated female gender was a risk factor for developing depressive symptoms post-SAH.

 Although several studies excluded patients based on previous psychological comorbidities, a selection of studies – 3 anxiety papers25, 35, 38, 3 depression papers36, 39, 37 – discussed the impact of pre-existing psychiatric disorders on the likelihood of developing psychological distress after SAH. Caeiro et al.36 outlined that those individuals with previous mood disorder reacted less constructively to an unexpected life event, while Hedlund et al.37 argued that a lifetime history of psychiatric morbidity results in increased risk of new psychiatric symptoms post SAH. A further 5 anxiety studies15, 40, 41, 42, 29 and 3 depression studies12, 43, 44 indicated a link between previous psychiatric morbidity and psychological distress post SAH, while the majority stated this as crucial for future research. In addition, only 1 study25 mentioned age as a potential risk factor for psychological distress.

* 1. **Quality assessment**

**3.5.1 Methodological quality assessment**

All 70 papers were assessed for methodological quality using the Hawker et al.21 tool. The results for anxiety and depression can be seen in table 3 and 4 respectively. Overall, the majority of papers in both groups were assessed as being ‘high quality’ – anxiety (n = 30) and depression (n = 16). Several studies were considered ‘medium quality’ – anxiety (n = 9) and depression (n = 8) and fewer considered ‘low quality’ – anxiety (n = 3) and depression (n = 4). The studies scored as ‘low quality’ were still considered as part of this review however more awareness was paid during data synthesis.

Risk of bias was considered in the quality assessment tool by Hawker et al.21. Although there was no scored assessment of individual bias for each study, several common biases became apparent during data extraction. Selection and reporter bias were the most common amongst the studies included in this review.

**3.5.2 Funnel plots assessing publication bias**

In addition to the Hawker et al. quality assessment tool, funnel plots for each set of studies were created to evaluate publication bias. All funnel plots showed marked asymmetry indicating heterogeneity amongst the studies due to selection, reporter or multiple publication bias mentioned above. Also the asymmetry appears to be consistent over the short and long term and across anxiety and depression studies. See 4 funnel plots in Figure 4a, 4b, 4c, and 4d.

**4. Discussion**

To our knowledge, this is the largest systematic review and meta-analysis evaluating precise estimates of the prevalence rates of anxiety symptoms and depressive symptoms in short-term and long-term after SAH, respectively. The review also identified potential risk factors linked to psychological distress after SAH which could help to identify those SAH patients who are particularly vulnerable to psychological distress. The review highlights the need for appropriate assessment and management of psychological stress in patients after SAH.

**4.1 Prevalence of anxiety symptoms**

In the short term, a variety of factors could contribute to the development of anxiety symptoms after SAH including biological factors such as possible organic damage to brain tissue in the limbic system45 and the initial, acute neuroinflammatory response to bleeding; and psychological factors including beliefs about illness and treatment such as worry of reoccurrence and fear of returning to social functions or everyday life.1,4, 6 Our meta-analysis revealed that the prevalence of anxiety symptoms appears to increase in the short term, with consecutive time points at 3 months, 6 months, 1 year and 2 years post SAH. Studies reported both short and long term anxiety symptoms revealed a growing number of individuals suffering from anxiety symptoms which appears to continue after 3 years. The higher prevalence of anxiety symptoms found in the long term after SAH, indicates continued impact on patients’ lives including negative consequences on their ability to self-care15, capacity to reintegrate and participate into social events, return to work13 and their ability to improve their overall quality of life2.

**4.2 Prevalence of depressive symptoms**

The prevalence of depressive symptoms in the short term appears to slowly increase over time after SAH and be most significant more than 3 years after the event. Studies that reported both short and long term depressive symptoms revealed the number of participants suffering from depressive symptoms appear to increase into long term. The cause of depressive symptoms amongst individuals after SAH may be a direct result of biological factors – the damage to brain tissue and persisting symptoms45; psychological factors – a reactivation of previously diagnosed mood disorders35, 37 or social factors – difficulty in returning to work or previous activities4, 46. A recent review by Tang et al.97 found similar prevalence and persistence of depressive symptoms however did not distinguish between short and long term symptomatology or other psychological distress such as anxiety. Supporting this, Kreitschmann-Andermahr et al.7 and Hedlund et al.35 both highlight the increased prevalence of depressive symptoms in patients after acute SAH. The increased depression may impact their coping strategies, cause sleep disturbances and cognitive impairment, and subsequently impair their overall quality of life.

 Overall, the findings from this review provide a new, collective insight into both the short-term and long-term prevalence of anxiety symptoms and depressive symptoms in individuals after SAH. The initial development and the relatively steady continuation of both anxiety symptoms and depressive symptoms results in long-term consequences for individuals, affecting their social participation, return to work and their overall quality of life. This review highlights the need for early and appropriate assessment for anxiety symptoms and depressive symptoms which may help to stratify a subgroup of patients who may benefit from interventions targeting psychological distress and improve overall health and quality of life after SAH.

**4.3 Risk factors for psychological distress**

A much larger number of female SAH patients were included in the current review, consistent with high prevalence of SAH in women47. This remains as an important risk to consider, as women have consistently higher prevalence rates of both anxiety symptoms and depressive symptoms48, 49. Although a few studies have looked at the *gender differences* as risk factors for SAH, not many have considered the association between gender differences and likelihood of developing psychological distress after SAH. The significantly higher proportion of women reported across all studies indicates the high vulnerability to anxiety and depression in female SAH patients. However, more research is warranted to confirm the gender difference. In addition, some studies suggest that individuals with *pre-existing psychiatric disorders* could be more likely to develop psychological distress. The traumatic nature of an SAH may trigger previous symptoms or exacerbate already present symptoms thereby intensifying the amount of psychological distress in these vulnerable individuals Future work to examine other potential risk factors such as patients’ beliefs about SAH and treatment and how they contribute to short and long term recovery adopting Leventhal's common-sense model is warranted105.

*The severity of SAH* as a potential risk factor for psychological distress after SAH should be considered. However, the majority of studies primarily included patients with less severe SAH scores or those with good neurological outcomes, it is difficult to examine the link between the severity of SAH and the development of psychological distress based on existing evidence. While inflammation plays a key role in the pathology of SAH, growing evidence also supports the neuroinflammatory pathway underlying both depression and anxiety which makes it an important risk factor to consider. Inflammation is a key component in the acute and chronic phases of neuronal injury50 after SAH. Studies have shown that a neuroinflammatory response post SAH contributes to early brain injury, cerebral vasospasm and delayed neurological deterioration51 with an extended time course of inflammation post SAH52-56. The growing evidence of inflammation in the pathogenesis of anxiety and depression strongly indicates that inflammatory responses after SAH may put patients in high risk of developing psychological distress. More research is needed to explore the neuroinflammatory mechanisms as such evidence may reveal new intervention targets. In particular, the benefits of anti-inflammatory agents for psychological distress after SAH remain to be explored. A high prevalence of psychological distress also occurs in a range of other physical illnesses including cardiovascular disease, gastrointestinal disease, pulmonary disease and chronic pain106-110. While shared underlying biological mechanism may contribute to the comorbidity, further research is needed to examine how patients’ beliefs about their illness and treatment may mediate this comorbidity and how best to predict and intervene psychological distress in patients suffering from these physical illnesses.

**4.4 Strengths and limitations**

This study is the first systematic review and meta-analysis to examine the pooled prevalence rates of anxiety symptoms and depressive symptoms in patients after SAH. A comprehensive and inclusive search strategy was employed during the review process. The majority of studies were being evaluated as high or medium quality, which provides strong confidence when interpreting findings and drawing conclusions. This study was also able to systematically review reported risk factors linked to anxiety symptoms and depressive symptoms after SAH in literature. However, there are several limitations to be considered. Firstly, while the most common cause of spontaneous SAH is a ruptured aneurysm, it can also be caused by a variety of rarer pathologies such as arteriovenous malformations or coagulopathy, and is idiopathic in a significant number of cases. SAH patients irrespective of cause were included in this review although a majority of studies focused on aneurysmal SAH. Some clinical features of the study population including the degree of bleeding, delayed brain ischemia, apoptosis, some complications in the acute period, and functional neurological outcomes which also impact on psychological distress111-113, have been reported recently but were not examined in the current review. Secondly, there is a selection bias across studies. In many studies, only patients with good neurological outcomes discharged from hospital and living independently were studied. This is in combination with self-selection bias whereby a functional and cognitive threshold for patients to participate in studies was indicated. Thirdly, there is a reporting bias as many studies used self-reported scales to determine individuals’ levels of anxiety and depression with varied diagnostic thresholds. Although these are straightforward tools to collect large amounts of data, the self-reported nature of them may allow for the overestimation or underestimation of symptoms and lack of consideration for previous or current treatments. Fourthly, there is a wide range of response rates across studies, with lower rates reported in older studies and higher rates in more recent studies using more comprehensive recruitment methods and improved analysis techniques. The variation in sample sizes, particularly those that are small, inherently minimises the power of the prevalence results in this review. Alongside this, some studies had grouped the anxiety and depression data together which led to difficulties uniformly extracting data. There was not enough data to perform a separate review on PTSD, therefore some anxiety and depression results may be representative of PTSD symptoms post SAH. Finally, there is a high heterogeneity across studies due to variations in study designs, outcome measures, study populations, durations of follow-up and varied data analysis approaches.

**5. Conclusions**

This is the largest systematic review and meta-analysis evaluating precise estimates of the prevalence rates of anxiety symptoms and depressive symptoms in both short-term and long-term after SAH, respectively. The findings highlight the need for appropriate assessment and management of these psychological distress in patients after SAH. Further research is warranted to explore potential underlying mechanisms such as the neuroinflammatory hypothesis and to develop holistic intervention programmes that incorporate understanding of both biological and psychological impact of SAH on the prognosis.

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