Those patients who survive the ictus of aneurysm rupture harbor substantial risks of neurological morbidity, functional disability, and cognitive dysfunction. Although the pervasiveness of cognitive impairment is widely acknowledged as a long-term sequela of aneurysmal subarachnoid hemorrhage (aSAH), the mechanisms underlying its development are poorly understood. The onset of aSAH elicits activation of the inflammatory cascade, and ongoing neuroinflammation is suspected to contribute to secondary complications, such as vasospasm and delayed cerebral ischemia. In this review, we analyze the extant literature regarding the relationship between neuroinflammation and cognitive dysfunction after aSAH. Pro-inflammatory cytokines appear to play a role in maintaining normal cognitive function in adults unaffected by aSAH. However, in the setting of aSAH, elevated cytokine levels may correlate with worse neuropsychological outcomes. This seemingly dichotomous relationship between neuroinflammation and cognition suggests that the action of cytokines varies, depending on their physiologic environment. Experimental therapies which suppress the immune response to aSAH appear to have a beneficial effect on cognitive outcomes. However, further studies are necessary to determine the utility of inflammatory mediators as biomarkers of neurocognitive outcomes, as well as their role in the management of aSAH.