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# Surgical Management of Traumatic Acute Subdural Hematoma in Adults: A Review

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## Abstract

**Traumatic acute subdural hematoma (ASDH) is a major clinical entity in traumatic brain injury (TBI). It acts as a space occupying lesion to increase intracranial pressure, and is often complicated by co-existing lesions, and is modified by cerebral blood flow (CBF) changes, coagulopathy, and delayed hematomas. Because of its complicated pathophysiology, the mortality of ASDH is still remaining high. In this review article, its epidemiology, pathophyiology, surgical treatment, and salvage ability are described. With regard to epidemiology, as the population ages, growing number of elderly patients with ASDH, especially patients with prehospital anticoagulant and antiplatelets, increase. Concerning pathophysiology, in addition to well-known initial intracranial hypertension and subsequent ischemia, delayed hyperemia/hyperperfusion, or delayed hematoma is being recognized frequently in recent years. However, optimal treatments for these delayed phenomenons have not been established yet. With regard to surgical procedures, all of craniotomy, decompressive craniectomy, and initial trephination strategies seem to be effective, but superiority of each procedure have not been established yet. Since Glasgow Coma Scale (GCS) scores, age, papillary reaction, and computed tomographic findings are strongly correlated to outcome, each factor has been investigated as an indicator of salvage ability. None of them, however, has been defined as such one. In future studies, epidemiological changes as population ages, management of delayed pathophysiology, superiority of each surgical procedures, and salvage ability should be addressed.**

Key words: acute subdural hematoma, traumatic brain injury, surgical management, indication, salvage ability

## Introduction

Acute subdural hematoma (ASDH) represents a major clinical entity in traumatic brain injury (TBI), diagnosed on computed tomography (CT) as extraaxial, hyperdense, crescent lesion between the dura, and brain parenchyma. ASDH is an acute space occupying lesion to increase intracranial pressure (ICP), and is often complicated by co-existing intracranial lesions, including a variety of diffuse injuries, contusional hematomas, and edemas. In addition, it is also modified by subsequent phenomenons such as both global and focal ischemia beneath hematoma, reactive hyperemia/hyperperfusion, coagulopathy, delayed hematomas, and so on. Because of its complex pathophysiology, mortality of ASDH has been remaining high, in spite of advances in emergency medical systems, diagnostic modalities, and treatment techniques.[1–8]

As the population ages, neurosurgeons confront with a growing number of elderly with TBI including ASDH, often suffering from delayed intracranial hematoma in association with prehospital anticoagulant and antiplatelet. Such elderly-specific condition has been widely recognized in recent years, but managements have not been well established. In this article, we briefly review the epidemiological and pathophysiological aspects of ASDH to focus on surgical management for it.

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## Epidemiological Overview

The frequency of ASDH has been proposed as approximately 10–20% of patients admitted with TBI.[1–3] When focusing on patients with severe TBI, Glasgow Coma Scale (GCS) score of 8 or less, approximately 60% of patients are accompanied by various extents of ASDH.[4] In Japan, age distribution

of ASDH has a couple of peaks for the younger [15–30 years old (y.o.)] and the older (45–80 y.o.) groups. In comparison with those in the United States, elderly patients are more frequent; approximately 70% of patients are over 45 y.o. and 40% are over 65 y.o.[8] As the population ages, ASDH is also increasing in its frequency.[9]

From analysis of Traumatic Coma Data Bank (TCDB) in the United States and Japan Neurotrauma Data Bank (JNTDB), most ASDHs are caused by motor-vehicle related accidents (MVAs) and falls,[10,11] Frequency of assaults or other mechanisms is significantly less. MVA is most frequent in the younger (15–30 y.o.) age group. Falls are most frequent in the older (45–80 y.o.) age group.[3] Studies with comatose patients have demonstrated MVA as the mechanism of injury in most of ASDH,[5,6,12] suggesting MVA causes not only as more severe injury but also accompanying diffuse axonal injury.

About from 35% to 80% of patients with ASDH present with GCS score of 8 or less.[1,2,13,14] A talk and deteriorate have been described in 6% of patients with GCS score of 3–15[15] or 13% of patients with GCS score of 3–8,[16] excluding prehospital deterioration. Mortality in patients with GCS score of 3–15 has been ranged from approximately 30–60%,[2,15,17,18] or with GCS score of 8 or less as 55–70%.[5,6,12,19,20]

## Pathophysiological Overview

ASDH is an acute space occupying lesion to increase ICP, and is often complicated by co-existing intracranial lesions, including a variety of diffuse injuries, contusional hematomas, and edemas. In addition, it is also modified by subsequent phenomenon, as follows:

### I. Cerebral blood flow (CBF) changes

In patients with ASDH, it has been demonstrated that Cerebral blood flow (CBF) markedly decreases immediately after injury.[21,22] Since the early CBF reduction occurs under the condition of normal or restored systemic blood pressure and arterial oxygenation, existence of nonsystemic causes are suggested. This CBF reduction is proposed as resulting from cerebral perfusion pressure (CPP) decrease due to ICP increase. Dysautoregulation,[23] cerebral vasospasm,[24] and reduced metabolic demand[25] due to primary brain injury may be also accounted for CBF reduction. Such secondary ischemia is a target of ICP- or CPP-oriented treatment in cases with ASDH.

Subsequent to CBF reduction, hyperemia/hyperperfusion frequently occurs in the cortex located beneath an evacuated ASDH, and prolongs to be associated with unfavorable outcomes.[26] Hyperemia/hyperperfusion may induce reperfusion injury initiated by oxygen free radicals, for example. Such hyperemia/hyperperfusion often occurs in the elderly.[27] ICP- and/or CPP-oriented management may not be suitable for hyperemia/hyperperfusion. The optimal treatment of hyperemia/hyperperfusion has not been established yet. Recently, early induction of hypothermia before craniotomy has been reported to have a potential to improve outcome in cases with evacuated traumatic intracranial hematomas.[28] Since hypothermia has been known to reduce reperfusion injury in animal models,[29] early induction of hypothermia before hematoma removal may have a potential to protect brain from reperfusion injury induced by hyperemia/hyperperfusion in cases with ASDH.

### II. Coagulopathy

It has been known that various degree of coagulopathy frequently occurs in patients with isolated blunt TBI,[30,31] as 22.7% upon emergency room (ER) arrival.[32] Brain tissue injury stimulates tissue factor pathway of coagulation, resulting in systemic bleeding tendency shortly after TBI.[33] Coagulopathy may influence hemostasis, delayed expansion, or occurrence of intracranial hematoma, leading to unfavorable outcomes. Recently, a high serum D-dimer level has been demonstrated to be associated with a poor outcome in patients with traumatic intracranial hematoma.[34] The role of D-dimer has not been fully understood; there may be a certain potential to affect outcome in TBI including ASDH.

### III. Delayed deterioration

Delayed deterioration, so-called "talk-and-deteriorate", often occurs in elderly patients with TBI including ASDH. The brain atrophies with age, and more intracranial spaces are created to compensate blood accumulation and brain swelling before symptoms appear. Delayed deterioration occurs within 6 hours after trauma in majority of cases being caused by expansion of ASDH, delayed traumatic intracerebral hematoma (DTICH), progression of contusional hematoma, and edema.[11,16] Since such deterioration is often rapid and critical, early detection and surgical intervention have been strongly recommended prior to inevitable deterioration.

As another entity of delayed deterioration, delayed posttraumatic acute subdural hematoma (DASH) has been recently recognized in elderly patients on anticoagulants following mild TBI. The patients initially have normal neurological examinations and normal CT, developing an ASDH with rapid deterioration from 9 hours to 3 days after the head injury. It has been suggested that elderly

patients on anticoagulants should be admitted for observation.[35]

Contralateral acute epidural hematoma following removal of ASDH is a rare but devastating intra- and post-operative complication.[36,37] The frequency of this occurrence has been reported as 6–30% in surgically treated ASDH. The mechanisms have not been fully understood. Initial decompressive craniectomy on the contralateral side has been attributed to release of a tamponade effect, usually produced by increased ICP and by post-traumatic arterial hypotension. Therefore, immediate postoperative CT scan has been recommended to evaluate this rare, but, potentially lethal complication in patients with ASDH and a contralateral skull fracture.[36] Gradual decompression with an initial burr-hole craniotomy followed by a decompressive craniectomy has been suggested to avoid this complication; however, its efficacy has not been established.[37]

**IV. Prehospital oral anticoagulant and antiplatelet**

As the population ages, it is increasingly common for elderly patients to encounter with TBI including ASDH. Many of them are being treated in the outpatient setting with anticoagulant or antiplatelet prior to injury. In cases with TBI, prehospital treatment with antiplatelet and anticoagulant has been associated with an increased risk of intracranial hematoma enlargement or delayed hematoma, leading to risk for increased morbidity and mortality.

Prehospital antiplatelet has been associated with significantly increased mortality and unfavorable outcome in TBI patients,[38–40] or with higher rates of neurosurgical interventions as well as more episodes of rebleeding.[41] Although several reports suggest no association of prehospital antiplatelet with increased mortality or unfavorable outcome,[42,43] the available data suggest that patients on antiplatelet therapy may have a higher risk of mortality and morbidity after TBI, especially in cases with intracranial hematoma including ASDH.[44] Issues of withdrawal and/or reversal of antiplatelet are still controversial. It has been demonstrated that platelet transfusion had no impact on rate of intracranial hematoma progression, neurological decline, or neurological outcomes in patients with TBI. Discontinuation of antiplatelet markedly increases risk of thromboembolism, especially in patients with coronary heart disease. The risk of coronary thrombosis after withdrawal of antiplatelet is greater than the risk of surgical bleeding.[45] In contrast, the risk of stroke seems relatively low as approximately 2% within 30 days after discontinuation of antiplatelet.[46] Thus, whether to withdraw or reverse antiplatelet in TBI may be decisions that depend on a variety of circumstances, such as extent of hematoma, necessity of surgical treatment, or indication for prehospital use of antiplatelet agent.[44]

Prehospital use of warfarin, seems to be a trend toward increased mortality in elderly patients regardless of injury severity.[47] The admitted prothrombin time-international normalized ratio (PT-INR) is directly correlated with mortality.[48] Specific replacement therapy, including vitamin K, fresh frozen plasma, or VII factor, facilitates successful clot evacuation without bleeding complications.[49] As antiplatelet agent, withdrawal and reversal of anticoagulant increases the risk of thromboembolism. Whether to withdraw or reverse may depend on a variety of circumstances.

## Surgical Aspects

**I. Indications**

Theoretically, intracranial hypertension due to ASDH may lead to transtentorial cerebral herniation and secondary ischemic injury of the brain. The purpose of surgery is to release from herniation and to reduce secondary ischemic injury minimally. Thus, ASDH causing significant mass effect, which may be effectively reduced by surgery, is supposed to be an indication for surgery.[50] In general, initial surgical indication may be based on the patients GCS score, pupillary examination, and CT findings. Neurological deterioration[50] and/or increase in ICP may also be an important factor in delayed decision.[51]

In a study analyzing CT findings in initially nonsurgically treated patients, it has been demonstrated that an midline shift greater than 5 mm in patients with a GCS score of lower than 15 on the initial CT was significantly related to the failure of nonsurgical treatment.[39] In another study, significant differences in clot thickness and hematoma volume have also been demonstrated between the operative and the nonoperative groups in patients with GCS scores 13–15. In addition, all patients with an initial hematoma thickness greater than 10 mm required surgery.[52] On the basis of the above literature review, a clot thickness greater than 10 mm, or a midline shift greater than 5 mm are suggested as critical parameters for surgery to remove ASDH, regardless of the GCS scores.[51]

As an indication for elective surgery, ICP monitoring may be important to make decision for surgery. Servadei et al.[2] developed a protocol to select comatose patients for nonoperative management. The criteria used to select comatose patients for nonoperative management were clinical stability or improvement during the time from injury to

evaluation at the hospital, hematoma thickness less than 10 mm and midline shift less than 5 mm on the initial CT, and ICP monitoring. Surgery was performed if the ICP exceeded 20 mmHg. Under this protocol, 15 of 65 comatose patients were treated nonoperatively, and 2 patients were identified as elective surgery based on increasing ICP and intracranial hematoma development. Since this protocol worked successfully, the authors concluded that nonoperative treatment can be safely used for a defined group of comatose patients with ASDH.

**II. Timing of surgery**

Theoretically, time from injury to surgery may be an important factor to rescue brain from secondary ischemia due to compression and/or intracranial hypertension by ASDH. In general, prompt evacuation of ASDH has been recommended in patients with indications for surgery.[50,51] This recommendation may be supported by the following studies. Seelig et al. demonstrated a 30% mortality in patients with ASDH operated within 4 hours after injury and a 90% mortality in those more than 4 hours after injury. It has also been demonstrated that the duration from injury to surgery was significantly shorter in patients with a functional recovery than with a fatal outcome, suggesting the importance of prompt evacuation of subdural hematoma.[5,53] It has also been reported that there were trends indicating that earlier surgery improved outcome, although the time from injury to operative evacuation of ASDH with regard to outcome morbidity and mortality were not statistically significant even when examined at hourly intervals.[6] However, the relationship between the time from injury to surgery and the outcome may be difficult to study, because patients operated in sooner time may have more severe injuries than those with delayed surgery.[51] Actually, most studies focusing on the time between injury and surgery have failed to demonstrate a correlation with outcome.[1,2,10,12,18,54]

On the other hand, time from TBI to surgery may not be as important as time from clinical deterioration to surgery. Haselberger et al. reported relationship between the time interval from onset of coma to surgery and outcome. They demonstrated that the outcome of patients operated within 2 hours after clinical deterioration is significantly better than those operated longer than 2 hours.[55] These results suggest that prompt evacuation of ASDH is indicated even after deterioration, including delayed deterioration such as talk and deteriorate.[50,51] The therapeutic time window has not been established, however, prompt surgery within 2 to 4 hours after clinical deterioration have a better outcome than those who undergo delayed surgery.[51]

In situations of elective surgery, timing of ICP increase beyond the threshold is suggested as the timing of surgery. (See "Indications" paragraph 3 for details.)

**III. Surgical technique**

Commonly used surgical techniques for the evacuation of ASDH include cranioplastic craniotomy, large decompressive craniectomy, trephination/craniostomy, or combination of these procedures. In reality, surgical techniques are not specified in most papers, and the effectiveness of surgical procedures is not addressed. For example, some institutes use decompressive craniectomies in all ASDH,[12] whereas other institutes use cranioplastic craniotomies,[6] or various procedures are used on different occasions.[10,17,55] The choice of surgical procedures may depend on the surgeon's expertise, training, neurological status of patients, duration from deterioration, pre-operative radiological findings, degree of intraoperative brain swelling, and availability of operating theater. In principle, the purpose of surgery is to release brain from intracranial hypertension to suppress secondary injury minimally. Thus, large craniotomy with hematoma evacuation may be principle.[50] When intra- and/or post-operative brain swelling is strongly expected, decompressive craniectomy may be suitable. It is supported by a recent publication which has demonstrated that decompressive craniectomy significantly improve outcome in patients with refractory intracranial hypertension due to extensive contusion, compared to routine craniotomy.[56] However, as it has been known that bony decompression result in apparent exacerbation of edema,[57] the superiority of decompressive craniectomy to craniotomy is still controversial.[58–60]

As an alternative strategy, emergency trephination has been widely accepted as an initial procedure for ASDH presented with impending herniation.[61–63] Trephination is a quick and easy technique to reduce ICP by evacuating hematoma. However, hematoma evacuation may often result in partially, ICP reduction may be often temporary, and hemostasis may not be obtained occasionally. Thus, emergency trephination should be followed by craniotomy or craniectomy. In recent years, trephination has been also applied as a minimum invasive procedure for elderly or patients with certain risks for craniotomy or craniectomy. As another aspect of trephination, hematoma irrigation with trephination therapy (HITT) has been also applied.[64] The concept of this procedure is to evacuate subdural hematoma as much as

possible, and to handle parenchymal injury under physiological condition, in order to reduce unfavorable effect of decompressive craniectomy, such as exacerbation of edema. The above-mentioned initial trephination strategies seem to have relatively good outcome, however, the superiority to other procedures are still controversial.

## Salvage Ability

Several factors have been identified to correlate with the outcome in patients with severe head injuries, including age, GCS score, pupil reactivity, and CT findings.[65–67] There may be a tendency to treat less aggressively because unfavorable outcomes are highly expected in patients with GCS score of 3, bilateral fixed and dilated pupils, or elderly. Questions have been raised as to whether these patients should be treated aggressively, or whether they have a chance of a meaningful recovery. An accurate early prediction of either survival or mortal appeared to be important to allow informed consent and to decide the aggressiveness of treatment.

With regard to advanced age, it has been reported that patients older than 65 years are statistically correlated with poorer outcomes.[6] Most patients older than 70 years with GCS of 9 or less die, or rarely have a poor outcome.[68,69] No patients older than the age of 75 years who had extensor posturing preoperatively, flaccid to pain, or had unilateral or bilateral fixed and dilated pupils, makes a meaningful recovery (GOS 3–5).[69] On the contrary, it has been reported that 1 of 28 comatose elderly patients survived with a GOS score of 4 or 5.[6] Multivariate analysis in patients operated for ASDH does not identify age as an independent predictor of outcome.[5,10,14] These results suggest that there may be a certain relationship between poor outcome and advanced age, but it seems to be impossible to predict death accurately on the basis of advanced age with certainty.

Concerning GCS score of 3, at presentation, has been associated with a significantly poor outcome.[70] In particular, mortality rates approaches 100% when the GCS score of 3 is associated with bilateral fixed and dilated pupils.[71] However, recent report in patients with GCS score of 3 demonstrate that good functional outcome is achieved in 25.5% of patients presenting with bilateral reactive pupils, 27.6% of patients presenting with a unilateral fixed and dilated pupil, 7.5% patients presenting with bilateral fixed but not dilated pupils, and 1.4% of patients presenting with bilateral fixed and dilated pupils, in blunt head trauma.[72] Similar results are also demonstrated by the analysis of JNTDB.[73] Even limited to ASDH, a recent report demonstrates that good functional outcome is achieved in 8% of patients with GCS score of 3.[74] Patients presenting with GCS score of 3 may be treated aggressively initially since a good functional outcome can be obtained in some cases.

## Conflicts of Interest Disclosure

The authors have no personal, financial, or institutional interest in any of the drugs, materials, or devices in the article. The authors who are members of the Japan Neurosurgical Society (JNS) have registered online Self-reported COI Disclosure Statement Forms through the website for JNS members.

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