Korean J Neurotrauma. 2020 Oct;16(2):113-125
https://doi.org/10.13004/kjnt.2020.16.e43
pISSN 2234-8999·eISSN 2288-2243

KJNT

Review Article

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Neurocritical Management of Traumatic Acute Subdural Hematomas

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OPEN ACCESS

Received: Apr 16, 2020
Revised: Sep 9, 2020
Accepted: Sep 9, 2020

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Conflict of Interest
The authors have no financial conflicts of interest.

ABSTRACT

Acute subdural hematoma (ASDH) has been a major part of traumatic brain injury. Intracranial hypertension may be followed by ASDH and brain edema. Regardless of the complicated pathophysiology of ASDH, the extent of primary brain injury underlying the ASDH is the most important factor affecting outcome. Ongoing intracranial pressure (ICP) increasing lead to cerebral perfusion pressure (CPP) decrease and cerebral blood flow (CBF) decreasing occurred by CPP decrease. In additionally, disruption of cerebral autoregulation, vasospasm, decreasing of metabolic demand may lead to CBF decreasing. Various protocols for ICP lowering were introduced in neuro-trauma field. Usage of anti-epileptic drugs (AEDs) for ASDH patients have controversy. AEDs may reduce the risk of early seizure (<7 days), but, does not for late-onset epilepsy. Usage of anticoagulants/antiplatelets is increasing due to life-long medical disease conditions in aging populations. It makes a difficulty to decide the proper management. Tranexamic acid may use to reducing bleeding and reduce ASDH related death rate. Decompressive craniectomy for ASDH can reduce patient's death rate. However, it may be accompanied with surgical risks due to big operation and additional cranioplasty afterwards. If the craniotomy is a sufficient management for the ASDH, endoscopic surgery will be good alternative to a conventional larger craniotomy to evacuate the hematoma. The management plan for the ASDH should be individualized based on age, neurologic status, radiologic findings, and the patient's conditions.

Keywords: Acute subdural hematoma; Decompressive craniectomy; Surgical endoscopy; Tranexamic acid; Intracranial pressure; Disease management

INTRODUCTION

Acute subdural hematoma (ASDH) has been a major part of traumatic brain injury (TBI). It is a common clinical entity for the neurosurgeons and usually results in a poor prognosis. ASDH is easily diagnosed on the brain computed tomography (CT) as extra-axial hyperdense crescent mass between the dura and the brain parenchyma. Intracranial hypertension may be followed by acute space occupying hematoma and brain edema because of accompanying brain damages. The hematoma usually arises from relatively low-pressure venous bleeding due to damaged venous veins, which may be traumatic or spontaneous. Arterial etiology may contribute in a small portion. But, high incidence of cortical artery rupture has been

reported.[66,87] ASDH occurs frequently under acceleration conditions,[30,61] resulting from linear brain acceleration within the skull causing stretch injury to veins or arteries.

ASDH is often complicated by diffuse axonal injury, global and focal ischemia, reactive hyperemia, and delayed hematoma expansion.[4,50,68] Ischemic brain damage adversely affects outcome and morbidity. To reduce ischemic brain damage, the early reduction of the brain shift, as well as control of increased intracranial pressure (IICP), must be mandatory.[4,31,78,85,91] Regardless of the complicated pathophysiology of ASDH, the extent of primary brain injury underlying the ASDH is the most important factor affecting outcome.[65]

The pathophysiology of ASDH varies with age, and that currently employed resuscitation and treatment methods have differentially improved the outcome for younger patients.[39] And a large cohort study for traumatic ASDH demonstrated a lower mortality rate than those of previous reports.[79] Earlier diagnosis with CT imaging and treatment improvement from regionalization of care in centers of excellence may affect the lower mortality. ASDH is the most common diagnosis in the elderly over 65 years in the nationwide databank.[26] It is higher chance of taking antiplatelets/anticoagulants and having medical illness and brain atrophy in the old ages. It could make a neurosurgeon in agony to decide the critical care of the traumatic ASDH. Herein, we review the critical care and the surgical managements for the traumatic ASDH and discuss the preoperative considerations in the various situations and the optimal way to get the best outcome.

PATHOPHYSIOLOGY OF ASDH

ASDH lead to accumulation of fresh hematoma on subdural space and complexed variable intracranial conditions as IICP, edema, cerebral blood flow (CBF) change, parenchymal contusion or direct injuries. In early stage of ASDH, CBF decreasing occurred markedly.[80,98] However, blood pressure or arterial oxygenation were non-pathologic condition in early ASDH stage. Ongoing intracranial pressure (ICP) increasing lead to cerebral perfusion pressure (CPP) decrease and CBF decreasing occurred by CPP decrease. In additionally, disruption of cerebral autoregulation, vasospasm, decreasing of metabolic demand may lead to CBF decreasing.[23,70]

ASDH may occur systemic coagulopathy.[18,58] Because, brain injury stimulates coagulation pathway and lead to systemic bleeding tendency.[97] Systemic coagulopathy after ASDH may occur hemostasis change, hematoma expansion, intracranial hematoma and lead to poor outcome. Kuo et al.[57] reported D-dimer level associated with outcome in patients with head trauma. However, D-dimer levels don't reflect patient's outcome perfectly.

Delayed deterioration occurs in elderly patient who got ASDH frequently. Elderly patient got atrophic brain volume and more large subdural space. Extra-subdural space may compensate increasing of hematoma and brain swelling before neurological deterioration. Delayed deterioration may occur often within 24 hours after trauma and it caused by hematoma expansion, traumatic delayed intracerebral hematoma, increasing cerebral contusion hematoma and edema. Recently, many of elderly patients took antiplatelet agent or anticoagulant due to cardiovascular or cerebrovascular disease. Neurosurgeons should define that antiplatelet agent or anticoagulant is new risk factor of delayed deterioration in elderly patient with head trauma. Despite elderly patient show normal CT scan initially, ASDH may

https://doi.org/10.13004/kjnt.2020.16.e43

detect with delayed deterioration from 9 hours to 3 days after trauma. Elderly patient who got head trauma should be observed closely and be taken serial CT scan.[44,88]

CRITICAL CARE OF ASDH

General management

Management of increased ICP is the key element for head trauma and the situation with increased ICP happens frequently in the ASDHs. Increased ICP (over 22 mmHg) may lead to poor outcome of TBI patients.[12,98] Thus, various protocols for ICP lowering were introduced in neuro-trauma field.

Head elevation of 30 degrees with cervical collar loosening can improve venous return and help to decreasing ICP.[8,67] Hyperventilation can decrease ICP by inducing vasoconstriction. However, respiratory alkalosis may lead to exacerbate secondary ischemia.[20,64,90] Muizelaar et al.[71] treated patient who got TBI with hyperventilation ($PaCO_2$ 25 mmHg) for 5 days. In this randomized study, hyperventilation group had a worse outcome than non-hyperventilation group. Mayer et al.[67] reported $PaCO_2$ of 30 mmHg is best target of hyperventilation. Guideline recommended hyperventilation as a temporizing measure for the reduction of elevated ICP.[17] The hypertonic or hyperosmolar fluid therapy can occur osmotic gradients and lead to fluid shifting from brain intracellular space to circulation system.[22] However, it can use only temporary until surgical treatments engaged.[27] Administration of intravenous steroid has no benefit evidence for ASDH patients. Rather, use of steroid occurred harmful effect for ASDH patient in large scale, prospective randomized multicenter study.[25] Hypothermia is challenging for treatment of ASDH patient. Mild therapeutic hypothermia after cardiac arrest and myocardial infarction showed improving patient's survival rating and increasing favorable neurologic outcome.[33,72,84] However, hypothermia for TBI does not establish their definitive availability. Hypothermia has been known to reperfusion injury in animal model.[49] However, result of hypothermia for TBI showed different aspect in clinical field. Recently, Eurotherm3235 trial reported that therapeutic hypothermia plus standard care to reduce ICP did not result in outcomes better than those with standard care alone in patients with an ICP of more than 20 mmHg after TBI.[10] In brain trauma foundation TBI guideline, hypothermal treatment for TBI patients trend to non-improving outcomes when was used early (within 2.5 hour after trauma), short term (48 hours) and prophylactic.[17] Shaefi et al.[81] analyzed recent 8 randomized controlled trials (RCTs) of hypothermia therapy for TBI published since 2007. The majority of these trials did not identify improvement with the use of hypothermia, though there were subgroups of patients that may have benefited from hypothermia. More well-designed study generally does not favor use of hypothermia for severe TBI. They recommended that empiric hypothermia should be avoided and hypothermia may use in very limiting conditions as for keeping body temperature control at their focus with the intend to avoid hyperthermia.[81] Mild hypothermia may try to be applied in TBI patients who may get some benefits.

Seizure control

Use of anti-epileptic drugs (AEDs) for ASDH patients have controversy. Rabinstein et al.[76] reported that 25% of ASDH patients experienced seizure attack. Seizure can occur increasing ICP or increasing brain metabolic demands.[93] As a most widely cited RCT trial, Temkin et al.[93] reported that use of phenytoin reduces seizure incidence from 14% to 4%. In clinical field, phenytoin/fosphenytoin is commonly used. Because it can be administrated

https://doi.org/10.13004/kjnt.2020.16.e43

by intravenous lines and can be changed tablet easily. More, it cannot effect to deep sedation.[8] Recently, levetiracetam arise as a 1st choice of AEDs with equivalent efficacy[55] and levetiracetam demonstrated equal effectiveness compare with phenytoin on seizure prevention after TBI.[101] Valproate may effect psychiatric or behavioral problems.[43,77] Usage of AED after TBI may reduce early stage seizure. However, late stage of seizure cannot prevent.[83] If TBI patient have not seizure during a week or a month after TBI, prophylactic AED may stop.

Anticoagulant/antiplatelet agent

Usage of anticoagulant or antiplatelet agent is increasing due to life-long medical disease conditions in ageing populations. Usage of anticoagulant or antiplatelet agent have a risk as coagulopathy or increasing bleeding tendency. Hematoma expansion is well recognized risk factor of life threatening in ASDH and coagulopathy is one of most important risk factors for ASDH expansion.[15] Patients who take oral anticoagulation have a 4–15 folds increased subdural hematoma (SDH) risk and may expand hematoma easily, increase risk of death, have worse outcome. Appropriate reversal of coagulopathy is essential medical management to improve ASDH.[28,99] Warfarin such as vitamin K antagonist is typical anticoagulation agent and patients who take warfarin should take 10 mg vitamin K bolus with 4-factor prothrombin complex concentrate 25–50 μ/kg(depend on international normalized ratio).[32] Recombinant factor VIIa and fresh frozen plasma is useful antidote of warfarin in emergent ASDH situation.[42] Recently, usage of non-vitamin K antagonist oral anticoagulant (NOAC) is increasing due to difficulty of warfarin monitoring. However, NOAC, such as dabigatran, apixaban, edoxaban and rivaroxaban haven't antidote for reversal of coagulopathy.[29] United States Food and Drug Administration approved idarucizumab (praxbind)[6] and andexanet alfa[7] for NOAC antidote. It needs to more investigation for effectiveness. If TBI patient who take anticoagulation agent doesn't need surgical treatment immediately, should discontinue anticoagulation agent as soon as possible, use effective antidot and keep close observation of neurological deterioration. If TBI patient who take anticoagulation agent need surgical treatment immediately, surgeon should recognize of coagulopathy during surgery and risk factors. ASDH patients who take antiplatelet agent should discontinue antiplatelet medicine quickly. Because antiplatelet agent has no antidote and reversal of coagulopathy take several days. Platelet transfusion had been considered for reversal of platelet function recover. However, PATCH trial showed that platelet transfusion in ASDH lead to worse outcome and it is associated with increased 3 months death.[13] Resumption of anticoagulant/antiplatelet agent after ASDH surgery is not clearly defined. Generally, it may resume 3–7 days after the surgery and the resumption time is dependent on each patient's medical risk.

Tranexamic acid (TXA) for ASDH

TXA is a synthetic material made by lysine amino acids, which effects anti-fibrinolytic role by binding to plasminogen and preventing plasminogen-fibrin interactions and clot dissolution.[47,94] TXA is used to reducing bleeding in trauma and off label uses widely.[47] According to TAX's pharmacodynamics, TAX may increase the possibility of thromboembolic complications. However, clinical trials reported no increasing the thromboembolic incidence.[5] In 2010, very meaningful study was reported about TXA as effect of TXA on death, vascular occlusive events, and blood transfusion in trauma patients with significant hemorrhage (CRASH-2) trials.[1] This study designed for various traumatic patients (non-intracranial trauma). This RCT was undertaken in 274 hospitals in 50 countries. All-cause mortality was significantly reduced with TXA using groups and the risk of death due to bleeding was significantly reduce. Vascular occlusive events did not differ significantly. Base on CRASH-2 trials results, CRASH-2 trial collaborators announced that possibility of

https://doi.org/10.13004/kjnt.2020.16.e43

TXA effect for TBI and need further study for TXA use in TBI. According to CRASH-2 data, effect of TXA on death, vascular occlusive events, and other morbidities in patients with acute TBI (CRASH-3) trials designed for TBI and included patient with TBI only.[2] Overall head injury related death rate was similar in both groups (TXA 18.5% vs. placebo 19.8%). because severe head injury with or without hematoma is independent risk factor of death by itself, TXA cannot influence to head injury related death rate on severe injured patients. So, they analyzed with subgroup after except severe injured group. In patients with mild to moderate head injury, head injury related death rate reduced with TXA use. In additionally, they analyzed effect of TXA by time to treatment. Early treatment was more effective than later treatment in patients with mild to moderate head injury. The risk of vascular occlusive event was similar in both groups. CRASH-3 trials showed possibility of TXA in head injury patients. If TXA is used as soon as possible after trauma, TXA may reduce death rate on mild to moderate TBI patients without increasing occlusive vascular event risk. In additionally, Ebrahimi et al.[24] reported that TXA may reduce surgery related bleeding in patient with SDH and EDH. Mean amount of bleeding during surgery was lower in receiving TAX groups compared to the placebo groups. Base on various reports, appropriate administration of TXA may reduce TBI related death rate without complications. More, if TBI patients need to surgery, surgery related bleeding may reduce and may reduce blood transfusion.

Expanding subdural hematoma at the subacute stage

A subacute SDH (saSDH) is chronologically defined as a hematoma that evolves from an ASDH within 4 to 21 days of head injury. A problematic saSDH is a rare complication in the usual course of an ASDH.[35,46,92] The clinical significance of saSDH results from expanding the hematoma and making the intracranial hypertension. Expanding saSDHs can be defined as initially non-operated ASDHs that show rapid neurological deterioration and expansion of the hematoma volume in the subacute stage.[35] The exact mechanism of expanding saSDH is still not elucidated. Several factors, such as older age, bleeding tendency, thicker initial hematoma, lower blood hemoglobin level, and higher blood leukocyte count may be involved.[35,46,59] And the saSDH may be a different disease entity from chronic subdural hematoma and the cerebrospinal fluid may take a role in the increase in the mass effect.[11,69]

Expanding saSDHs have been reported to occur in approximately 10% to 30% of ASDHs treated conservatively.[35,45,89] The clinical deterioration usually happens rapidly. The clinical concern and surgical intervention of expanding saSDHs usually occurs around 13 days after head trauma.[35,92] Various surgical methods were proposed for the saSDH, including craniotomy, craniectomy, and burr-hole procedures. It could be said that the aim of surgery is rapid and safe recovery from the symptoms. From the point of this view, minimal surgery could be considered. Ha et al.[35] reported that only 1 case in the 23 cases of expanding saSDHs was treated with craniotomy and the others with burr-hole or twist-drill craniotomy with closed drainage. And no revision surgery was required. The saSDH could be said in the state of liquefaction, so the simple closed drainage is enough to relieve the clinical symptoms.

Expanding saSDH is worthwhile to be kept in mind as a pitfall in non-surgical management of ASDH. Brain CT around the 13th day after head trauma for the ASDH patient should be taken and older patients with relatively large ASDH should be carefully monitored whether an expanding saSDH happens.

https://doi.org/10.13004/kjnt.2020.16.e43

SURGICAL MANAGEMENT OF ASDH

Decompressive cranial surgery has a long history and we can find legacy of neurosurgery from archaeologic evidences. This surgical procedure has been very useful procedure in TBI.[3,9,34,75] Decompressive craniectomy can reduce patient's death rate. However, it does not mean that decompression can be help to good functional outcome always.[21] ASDH is space occupying lesion and may accompany brain edema. Character of ASDH may lead to chain reaction with brain herniation and secondary brain injury. Indication of decompressive surgery may be severe mass effect of traumatic brain. Because purpose of decompressive surgery is prevention of brain herniation and secondary brain injury.[63] Surgical decision should consider that CT scan, Glasgow Coma Scale (GCS) score, neurological deterioration, IICP sign.[16] Surgical decision was associated with timing of surgery. Timing of surgery may be an important role to save un-injured brain tissue from secondary ischemia due to IICP.[48] However, it is very difficult to study that find relationship between timing of surgery and outcome. Because, severe injured patient may undergo surgery more early periods. In fact, many of study failed demonstration between timing of surgery and patient's outcomes.[39,53,54,65,86,96] Kim et al.[52] reviewed 16 literatures about the impact of time to surgery in TBI. Five literatures (31.1%) found that outcome was affected by the timing of surgery and 11(68.7%) was not. The 75% of literatures reported that timing of surgery was not significant in severe TBI situation. At present situation, these literatures may reflect paradoxically that surgical timing associate with various conditions and it is not an easy to decide appropriate surgical timing. As a result, IICP is most important indication for timing of surgery and surgical decision. ICP monitor is used for surgical timing traditionally. What else we may use serial CT scan for surgical timing.[88]

Decompressive craniectomy or craniotomy

Surgical approach for ASDH is preferred treatment option. However, whether decompressive craniectomy or craniotomy is suitable procedure for ASDH have controversial.[62] Several studies reported efficacy of decompressive craniectomy which have brain parenchymal injury or swelling.[82,95] Either, craniotomy for ASDH may be alternative procedures for ASDH without brain parenchymal injury or swelling. Because decompressive craniectomy have their own complication risk factor of additional cranioplasty surgery.[60,74,102] Phan et al.[73] compared procedure outcome of decompressive craniectomy and craniotomy in ASDH patients. Poor outcome was higher (60.1% vs. 50.1%, p=0.004) in the craniectomy group. They analyzed their own study and compare with other studies. When comparing the preoperative characteristics of the craniotomy vs. craniectomy groups, craniectomy groups have more lower GCS score and high number of poor prognosis. As a result, they insisted that craniotomy and craniectomy groups are not comparable.[73]

There are meaningful 2 trials for decompressive craniectomy. The DECompressive CRAniectomy (DECRA) investigated effect of early decompressive craniectomy in brain swelling.[37] However, surgical groups associated with poor outcome. The most disputable issue has been the enroll indication of ICP ≥20 mmHg for ≥15 minutes within 1 hours as indication for surgery. Honeybul et al.[38] contended 20 mmHg was too low and 15 minutes too short to decide operation and surgical morbidity may offset potential improvement by decompressive craniectomy. Trial of decompressive craniectomy for traumatic intracranial hypertension (RESCUEicp) had different inclusion criteria from the DECRA trial which was a higher ICP (25 mmHg for 1–12 hours, despite maximal medical treatment).[40] RESCUEicp trial showed that surgical groups reduced mortality more than medical groups and surgical

https://doi.org/10.13004/kjnt.2020.16.e43

groups increased survivors who had vegetative state (8.5% vs. 2.1%) or severely disabled (21.9% vs. 14.4%). Interpretation of RESCUEicp trial's results should be careful. If TBI patients survive as maximal medical treatment, patients have a chance that may recover independently. However, if patient's survive with decompressive craniectomy, patients may have high possibility of severe disability.[38] As a result, decompressive craniectomy may reduce mortality effectively, however, craniectomy have a own surgical risk factor as post operation hematoma due to large surgical flap, infection due to large surgical wound, need additional cranioplasty surgery. Surgeon may consider craniotomy for ASDH patients who haven't combined severe brain parenchymal contusion, haven't severe brain swelling, have atrophic brain on elderly.

Endoscopic surgery for ASDH

Decompressive craniectomy is open required in the patient of an ASDH with brain edema. However, craniotomy is a less invasive alternative that may avoid the complications and the later cranioplasty associated with decompressive craniectomy in certain situations.[60] If the craniotomy is a sufficient management for the ASDH, endoscopic surgery through a small craniotomy will be an alternative to a conventional larger craniotomy to evacuate the hematoma. And endoscopic surgery can save time and reduce blood loss from the large craniotomy.

In the subdural pathologies, endoscopic approaches have been used to treat the empyema and the chronic hematoma.[14,36] And endoscopic removal of ASDH have been reported recently.[19,41,51,56,100] But, no well-designed comparative study or large case series is reported and clinical experiences are limited up to now. And also, indications for the endoscopic surgery have not been well established.

Although the endoscopic surgery for an ASDH could be performed in very selected patients, it will be more implemented in the future. Possible candidates for the endoscopic procedure are 1) ASDH with mass effect, 2) sufficient decompression after only removing the hematoma, 3) absence of severe coagulopathy, and 4) brain atrophy observed. So, this surgery will be good in the elderly with ASDH. Endoscopic evacuation of traumatic ASDHs was achieved through a burr-hole.[56,100] But, it could not give enough window to accommodate the surgical instruments. And complete hemostasis may not be achieved with burr-hole procedure and rebleeding was reported after surgery.[95] Hwang and Shin[41] proposed normal small craniotomy point for the endoscopic surgery of traumatic ASDHs. They made a small craniotomy around the superior temporal line at the coronal suture and reported the surgical outcomes as a minimally invasive surgery. Flexible endoscope, even in low resolution, can be a good tool to get optimal and safe evacuation of the ASDH.[51] Endoscopic evacuation of traumatic ASDH will be a novel procedure as a minimally invasive surgery in appropriately selected cases.

CONCLUSIONS

Outcome of ASDHs have been dismal because of its combination of diffuse axonal injury and accompanying intracranial hypertension. When the surgical treatment is required, large craniotomy and/or craniectomy is inevitable. But, the incidence rate in the elderly increased, the different management policies are emerging. Antiplatelets and anticoagulants become bigger issues not to expand the hematoma and in preparing the hematoma evacuation surgery. And minimally invasive surgery including burr-hole surgery and endoscopic surgery may be a good

https://doi.org/10.13004/kjnt.2020.16.e43

option to evacuate the ASDH. The management plan for the ASDH should be individualized based on age, neurologic status, radiologic findings, and the patient's medical conditions.

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