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(CASE REPORT)

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Case report: 82-year old patient with chronic subdural hematoma (SDH)

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Abstract

Background: Chronic Subdural Hematoma (CSDH) is commonly found on elderly and appears to be increasing within the last decades. Not only associated with substantial morbidity and mortality, CSDH also increases financial burden, thus appropriate treatment is required for better outcomes.

Case Presentation: 82-year-old patient is admitted with the chief complaint of decrease in consciousness since 12 hours before admission. The family also complained an untreated left hemiparesis since one week prior to the admission. Patient also complained pain when straightening the right leg before losing consciousness.

Summary: CSDH is a common neurosurgical case amongst elderly. Burr-hole drainage is the preferred technique to treat the condition. Follow up after the treatment is recommended as recurrence is quite common.

Keywords: Chronic Subdural Hematoma; CT scan; External Ventricular Drainage; Burr-hole drainage; Head trauma

1. Introduction

Chronic Subdural Hematoma is defined as a collection of blood and blood breakdown products within intracranial subdural space which liquefies over time. CSDH has an incidence of 17/100,000/year, which increases with age. It is commonly found on elderly and it's incidence appears to be increasing within the last decade [1–4].

It is a complex disease with pathophysiologic cycle involving both traumatic and inflammatory components. Not only associated with substantial morbidity and mortality, CSDH also increases financial burden, thus appropriate treatment is required to ensure better outcomes. The management might involves careful observation and surgical treatment, with burr-hole drainage being the preferred treatment [2,4].

2. Case report

82-year-old patient is admitted with the chief complaint of decrease in consciousness since 12 hours before admission. The family also complained an untreated left hemiparesis since one week prior to the admission. Patient also complained pain when straightening the right leg before losing consciousness. History of nausea, vomiting, seizure, fever and past medical conditions were denied. However, there was indeed a history of falling down 2 months' prior, head hitting the floor in the process.

Initial physical examination is shown below on Table 1**.** High blood pressure was observed along with pin point pupil, and left lateralization on the neurological examination.

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Table 1 Physical examination

Parameter	Result
Vital Signs	GCS 314 Blood pressure: 190/84 mmHg Heart Rate: 78x/min Respiratory Rate: 20x/min Temperature: 36°C SpO2: 96% on RA
Head/Neck	Anemia (-) Icterus (-) Dyspnea (-) Pin point pupil
Thorax	Pulmo: ves/ves, Rh -/-, Wh -/- Cor: S1>S2 reguler, murmur (-), gallop (-)
Abdomen	Soefl, bowel sound (+)
Extremity	Edema -/-, cold warm extremities, CRT <2 second
Neurological Examination	Neck stiffness: - Brudzinski I/II/III: - Motoric: Left lateralization Sensoric: hard to evaluate Cranial nerve function: hard to evaluate

Head CT scan without contast and chest X-ray is performed on the same day of admission (4 October 2023). CT scan shows chronic SDH on right frontotempoparietal region + left herniation subfalcine to midbrain 1,2 cm. Chest X-ray shows normal no anomaly. ECG is also performed and shows no anomaly.

Serum electrolyte, kidney function, hepar function, complete blood count, and blood glucose was examined on the time of admission shown in **Table 2**.

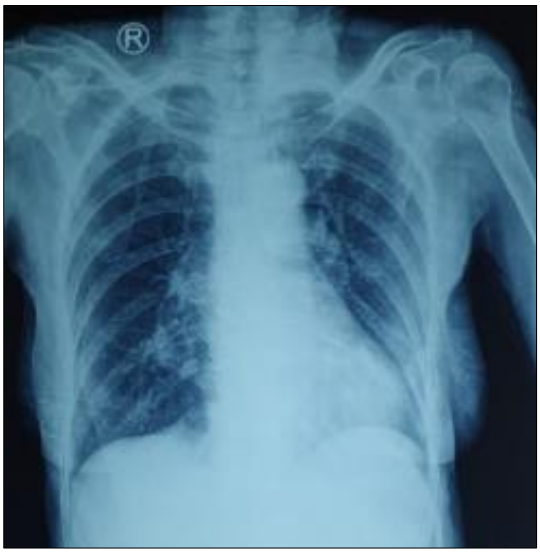


Figure 1 Patient CXR (4/10/2023) shows no anomaly

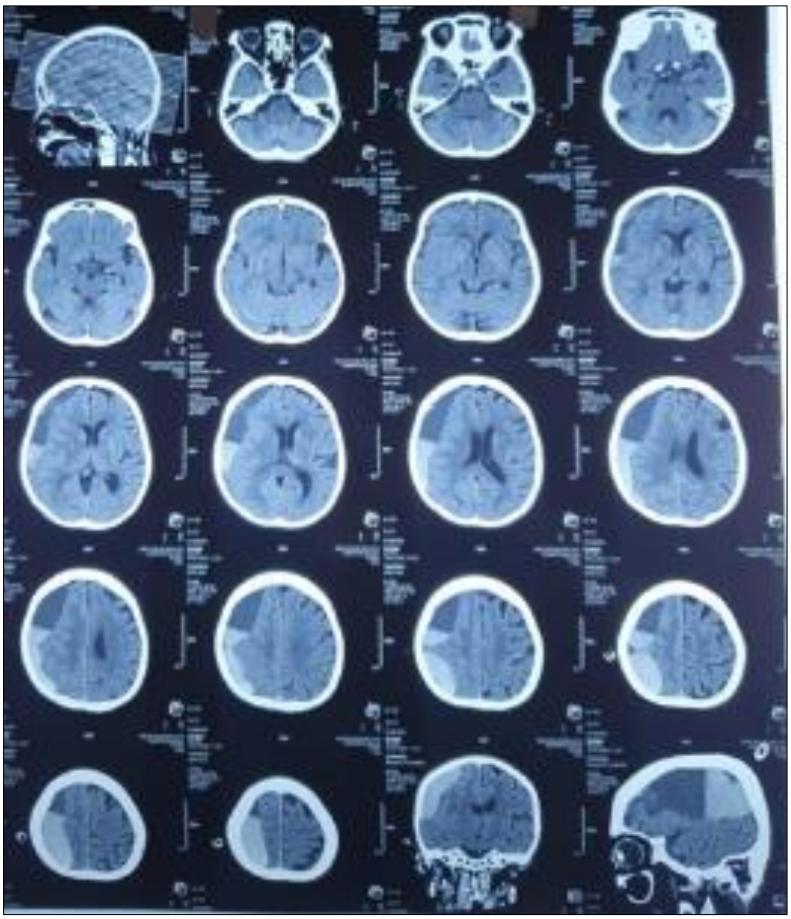


Figure 2 CT Scan without contrast showing chronic SDH on right frontotempoparietal region + left herniation subfalcine to midbrain 1,2 cm

Table 2 Serum electrolyte, kidney function, hepar function, complete blood count, and blood glucose

	04/ 10	**05/ 10**	**13/ 10**	**Unit**	**Normal Value**
Complete Blood Count					
Hb	13.6		10.3	g/dL	12 – 17
WBC	11.6		17.84	$X10^3$ µL	4,5 – 11
HCT	39.6		28.8	%	38 – 51
Thrombocyte	292		348	$X10^3$ µL	150 – 450
Kidney Function					
Ur/Cr	46/ 0.98			mg/dL	17 – 45 / 0,5 – 1,5
Liver Function					
SGOT	35			U/L	L<37/ P<31
SGPT	16			U/L	L<41 / P<31
Hematostatic Function					
PT		15.4		detik	10” – 14”
APTT		36.4		detik	25” – 35”

Serum Electrolyte					
K+	3.9			mmol/L	3,4 – 5,3
Na+	135			mmol/L	135 – 150
Ca	1.2			mmol/L	1,00 – 1,40
Cl	99			mmol/L	98 – 107/ 96 – 114
GDA	124			mg/dL	70 – 120
Viral Marker					
HbsAg		Neg			
Anti HCV		Neg			
HIV Rapid 1		Neg			

Patient is diagnosed with decrease of consciousness due to chronic subdural hematoma presented with left hemiparesis. Initial treatment was administered before surgery was performed. Bedrest with head trunk up 30 degrees was advised. O2 2 lpm through nasal cannula was administered. Patient received IVFD NaCl 0,9% 1500 ml/24 hour, IV Ceftriaxone 2x1 gram for 5 days, IV Metamizole if fever is present, IV Ranitidine 1x1 ampule. Nasogastric tube is administered, and PO medications include folic acid and complex vitamin B each 1 tablet per day, Piracetam 2x800 mg and Candesartan 2x16 mg. While waiting for surgery, patient blood pressure increases to 223/118 mmHg despite receiving oral antihypertensive therapy, thus IV Nicardipine 5 mg/hour was administered with the target of systolic blood pressure bellow 160 mmHg. Systolic blood pressure continued to fluctuate between 180 – 195 mmHg, thus Captopril 2x50 mg, Spironolactone 1x100 mg, and Amlodipin 2x10 mg was administered.

Even though burrhole drainage was already scheduled, patient's family initially refused the surgery thus patient is treated conservatively for 14 days. At the 15th day of hospitalization, family agreed to the procedure. Patient was treated with Anbacim 2g as prophylaxis antibiotic prior to the surgery.

Following the surgery, patient condition improved. Hemiparesis and difficulty to communicate persists, however GCS increases to 434 which further increases to 435 on the next day. Vital signs were stable. Patient was then discharged on the 5th day post-surgery.

3. Discussion

Within this case is presented 82-year old female with history of head trauma 2 months prior to admission. This aligns with the reported risk factors which includes old age and history of head trauma. CSDH indeed occurs primarily on patients of older age, the reason might be related to age-related general brain athropy, increasing risk for falls, and frequent administration of antithrombotic medications. Other risk factors include: male, anticoagulant administration, and alcoholism [1,2].

Etiology and pathogenesis had evolved over time. Recent understanding suggest subclinical traumatic injury might open dural border layer leading to the activation of reactive inflammatory cascade, thus releasing angiogenic factors. This ultimately results in the development of inflammatory membranes and delicate neovessels, which over time generate a constant fluid and blood exudation [3].

CSDH consists of 3 clinical phases. The initial traumatic event, which initiates the cycle of creating CSDH, might be one or more subclinical or clinical traumatic episodes. Within the case reported here, this occurs 2 months prior to the time of admission. After this, there is an asymptomatic latency period during which a number of mediators, including as fibrin, angiopoietin-2, type 1 and type 3 procollagen, vascular endothelial growth factor, and other cytokines and chemokines, stimulate the growth of CSDH. After the intracranial vault's compensatory systems are overpowered by the growing hematoma, a period of symptoms eventually starts [3].

Symptoms might include headaches, limb weakness, gait abnormalities, and cognitive impairment. Usually symptoms are evident within 3 weeks to 3 months after the onset of head injury without brain damage [4–6]. Within this case, headache and limb weakness is present, followed by decrease consciousness. History of head trauma is also present 2 months prior.

Severity can be classified into 5 grades, ranging from 0 to 4 based on their clinical presentation: [7]

- Grade 0: Normal neurologic condition
- Grade 1: The patient is focused and awake, with only minor symptoms (like a headache) and no neurological impairment (like reflex asymmetry).
- Grade 2: There is drowsiness and neurological impairment, for example hemiparesis
- Grade 3: Stuporus with severe focal symptoms, such as hemiplegia, but nevertheless responsive to painful stimuli
- Grade 4: Comatose without motor response towards noxious stimuli; decorticate or decerebrate posturing.

For patient with history of head trauma, selective CT scan without contrast for patient with neurological deficit is generally suggested. For patient with mild head trauma (GCS 13-15) with clinical decision indication, moderate (GCS 9-12) or severe (GCS 3-8) and penetrating head trauma, head CT scan without IV contrast is usually appropriate [8].

Cranial imaging provides the basis for diagnosis, mostly non-contrast CT scan which displays hypodensity, isodensity or mixed density lession. It has also been stated that extended recumbency can cause a layered kind of hematoma density by separating the fluid and blood components. The blood components are reflected in the CT density coefficients. More substantial components sink due to gravity if the patient stays in the laying position for an extended period of time. The likelihood of elderly adults lying down for extended periods of time and engaging in less physical exercise is higher, such as patient in this case. Thus, gravity separates the blood components, and the CT image shows layered density [6,9].

However, in SDH itself, there are three hypotheses of mixed density pathophysiology. The first being the difference between solid clot and liquid blood on hyper-acute bleeding. The second is resolving hematoma during transitional period, which appears as a peripheral hypodensity within central hyperdensity. Hypodensity is faster in peripheral region because fibrinolysis in CSF is activated from the outside. The third hypothesis is repeated microhemorrhage. It is difficult for insidious rebleeding to create mixed density as opposed to increasing the density. Repeated trauma may also lead to acute haemorrhage which create hyperdense layer or lump within hypo/isodense hematoma [9].

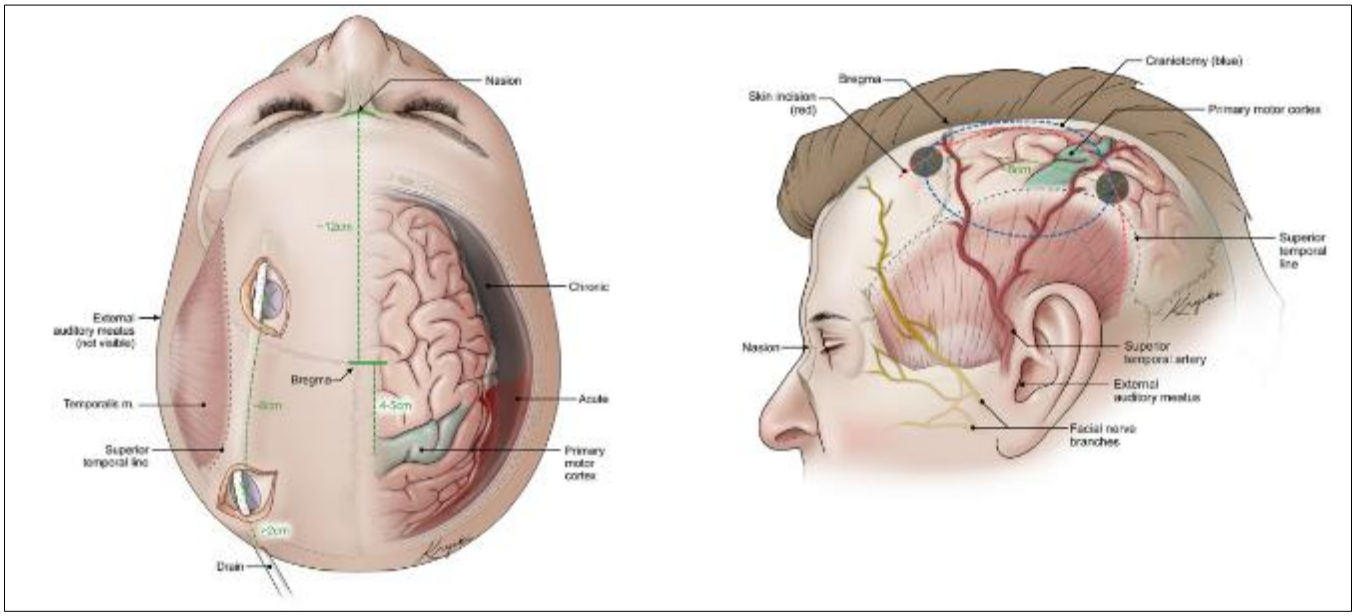
This varying radiodensity might differentiate lesion age. Acute lesion have high radiodensity comparing to serebrum, subacute lesion (up to the age of two week) is isodense compared to cerebrum, while chronic lesion (>3 weeks) is hypodense compared to the cerebrum. CT findings may also describe unilateral or bilateral lesion, location (tipically on the convexity), thickness, and the presence of membrane. However, MRI is more sensitive on determining internal components (such as intrahematoma membranes) and size, which can be further enhanced by contrast to detect fresh bleeding, hemolysis, and hemoglobin [4–7].

On asymptomatic patients, some CSDH might resolve spontaneously, thus careful observation might be sufficient. Other conservative treatments include intra cranial pressure control, reversal of anticoagulant and several serial exams can be performed [3,6].

Surgical treatment, namely burr-hole craniostomy (BHC) with (subdural) drainage remains as the preferred procedure on symptomatic patients to reduce intra cranial pressure. 1-2 burr holes are drilled into the skull while the patient is sedated followed by saline irrigation. For large CSDH, two burr-holes are typically positioned on the anterior (in front of corona suture) and posterior (approximately 8 cm posterior to the first hole, on tuber parietalis). Both holes are positioned cranial to the superior temporal line, which typically aligns with the hematoma's cranio-caudal centre and minimises the possibility of harming expressive brain regions (such as the operculum or primary motor cortex). The chance of damaging a bridging vein increases and the amount of gravity-induced passive hematoma outflow decreases if the placement is more superiorly and medially. An outer membrane that may be present after the dura is opened in a cruciate manner needs to be coagulated. It is not recommended to cut through the outer membrane past the burr-hole margins, and there is insufficient evidence to justify accessing the inner membrane. The vascularized membrane may bleed, and if it does so far from the surgical site, it may be difficult to stop the bleeding [7].

Further research are still needed for uncertainties on the ideal location for drain placement (subdural, subgaleal, or subperiosteal), the number of burr holes that should be used, and the value of intraoperative irrigation. According to preliminary analyses, after burr hole surgery, subperiosteal drains may result in lower rates of infection, recurrence, and drain misplacement than subdural drains; additionally, subgaleal drains seem to have the same efficacy rates as subdural drains, but results in a lower incidence of parenchymal injury. The complication rate for BHC ranges from 8% to 43%, according to data from randomized controlled trials [3].

However, if haemorrhage can't be stopped with continued irrigation, burr-hole can be converted to craniotomy. When the two burr-holes are combined into a craniotomy, the skin incision may raise the following issues: (1) The upper part of the hematoma and cavity between the superior temporal line and the superior sagittal sinus is difficult to access, (2) preserving subcutaneous vascularization and superior temporal artery branches, (3) preserving the frontal branch of the facial nerve. The skin incision in the figure bellow has been suggested to overcome these considerations [7].



A) The burr-hole locations are highlighted, taking the motor cortex into account. The authors' suggested placement of the subcutaneous/subgaleal drain is depicted here. B) The lateral image showed the skin incision for a craniotomy conversion in addition to the same Burr-hole placements. The advantages of this skin incision are illustrated by emphasising anatomical markers.

Figure 3 Burr Hole Drainage Location

Other procedures such as twist drill craniostomy (TDC) are also available. However, it was shown from meta-analyzes that BHC has better outcomes and lower complication compared to craniotomy and TCD. Therefore, nowdays craniotomy were only recommended on solid, ssified, calcified, or multiple membranous hematoma [6].

Bedrest are suggested after the surgery, as mobilization immediately after surgery are shown to be related to high recurrence rate. This finding remains controversial and needed further research [6].

However, on conditions such as the presented case, where consent was not acquired from the family members, careful observation should be performed [4,7].

Alternative strategies such as administration of high-dose glucocorticoids have been suggested.[5] In some institutions, patients are administered dexamethasone with the intention of blocking inflammatory changes in the subdural area, which could hinder the growth and endurance of hematomas.[10] However a recent study comparing Dexamethasone treated and placebo treated group on 748 patients. At six months, dexamethasone medication produced more adverse events and fewer favorable outcomes than placebo; however, the dexamethasone group underwent fewer repeat surgeries [4]. This was later reinforced by a multicentered randomized trial in Netherlands, finding that dexamethasone therapy for CSDH resulted in worse outcomes than placebo, including higher infection rate, hyperglicemia, and delirium [10]. Within this case, patient is not treated with Dexamethasone at all, even during her conservative therapy period. Intracranial pressure is managed by controlling blood pressure.

Even though acute SDH primarily happens due to rupture of bridging veins leading to blood collection within the dural space, 30% of the cases might also be secondary to cortical arteries rupture, thus it is suggested that systolic blood pressure control might be beneficial. Blood pressure management varies as there has been no specific consensus regarding this matter. So far, a trial showed that systolic blood pressure reduction leads to decreased hematoma expansion. Previously, American Stroke Association recommended to lower SBP in patients with SBP >200-220 mmHg with target approximately ~140 mmHg, ranging from 150 – 220 mmHg. However, additional studies are still needed on this matter [11].

ACE Inhibitors has been suggested to reduce recurrence risk in CSDH patient, however contradicting studies has also been reported. Tranexamic acid can also be administered as an attempt to treat recurrent CSDH, resulting in hematoma resolution by halting fibrinolysis and kinin-kallikrein inflammatory system. Statin also exhibit anti-inflammatory effects by facilitating blood vessel repair by recruiting endothelial progenitor cells [3,6].

Recurrence is quite common, ranging from 5% to approximately 30%.[1,3] Routine CT imaging following the surgery has been suggested to evaluate re occurrence. Higher recurrence rates are associated with CSDH that have isodense, hyperdense components or a separated or laminar architecture type [5,12].

Prognosis can also be predicted by scoring, such as Subdural Hematoma in the Elderly (SHE) which predicts 30-day mortality rate in elderly (>65 years) which relies on age, admission GCS, and hematoma volume as shown in **Table 3** bellow. 30-day mortality rate in CSDH increase from 12.2% in SHE-score 0 to 33.3% in SHE-score 3, with area under the curve of 0.80 [3].

Table 3 SHE score

Components		**Score**
Age >80 yo	No	0
	Yes	1
GCS on admission	5-12	1
	3-4	2
Hematoma volume >50 ml	No	0
	Yes	1

Prognosis can also be predicted by scoring, such as Subdural Hematoma in the Elderly (SHE) which predicts 30-day mortality rate in elderly (>65 years) which relies on age, admission GCS, and hematoma volume as shown above [3].

4. Conclusion

Within this case is presented 82-year-old patient is admitted with the chief complaint of decrease in consciousness since 12 hours before admission. CSDH is diagnosed through a non-contrast head CT scan. CSDH is a common neurosurgical case amongst elderly. Burr-hole drainage is the preferred technique to treat symptomatic CSDH with increasing ICP. Follow up after the treatment is recommended as recurrence is quite common.

Compliance with ethical standards

Disclosure of conflict of interest

No conflict of interest to be disclosed.

Statement of ethical approval

The present research work does not contain any studies performed on animals/humans subjects by any of the authors.

Statement of informed consent

Informed consent was obtained from individual included in this study

References

[1] Rauhala M, Helén P, Huhtala H, Heikkilä P, Iverson GL, Niskakangas T, et al. Chronic subdural hematoma—incidence, complications, and financial impact. Acta Neurochir. 2020 Sep, 162(9):2033–43.

[2] Blaauw J, Jacobs B, Den Hertog HM, Van Der Gaag NA, Jellema K, Dammers R, et al. Neurosurgical and Perioperative Management of Chronic Subdural Hematoma. Front Neurol. 2020 Jun 19, 11:550.

[3] Feghali J, Yang W, Huang J. Updates in Chronic Subdural Hematoma: Epidemiology, Etiology, Pathogenesis, Treatment, and Outcome. World Neurosurgery. 2020 Sep, 141:339–45.

[4] Hutchinson PJ, Edlmann E, Bulters D, Zolnourian A, Holton P, Suttner N, et al. Trial of Dexamethasone for Chronic Subdural Hematoma. N Engl J Med. 2020 Dec 31, 383(27):2616–27.

[5] Miah IP, Tank Y, Rosendaal FR, Peul WC, Dammers R, Lingsma HF, et al. Radiological prognostic factors of chronic subdural hematoma recurrence: a systematic review and meta-analysis. Neuroradiology. 2021 Jan, 63(1):27–40.

[6] Tamura R, Sato M, Yoshida K, Toda M. History and current progress of chronic subdural hematoma. Journal of the Neurological Sciences. 2021 Oct, 429:118066.

[7] Nouri A, Gondar R, Schaller K, Meling T. Chronic Subdural Hematoma (cSDH): A review of the current state of the art. Brain and Spine. 2021, 1:100300.

[8] Shih RY, Burns J, Ajam AA, Broder JS, Chakraborty S, Kendi AT, et al. ACR Appropriateness Criteria® Head Trauma: 2021 Update. Journal of the American College of Radiology. 2021 May, 18(5):S13–36.

[9] Park HR, Lee KS, Shim JJ, Yoon SM, Bae HG, Doh JW. Multiple Densities of the Chronic Subdural Hematoma in CT Scans. J Korean Neurosurg Soc. 2013, 54(1):38.

[10] Miah IP, Holl DC, Blaauw J, Lingsma HF, Den Hertog HM, Jacobs B, et al. Dexamethasone versus Surgery for Chronic Subdural Hematoma. N Engl J Med. 2023 Jun 15, 388(24):2230–40.

[11] Plowman K, Lindner D, Valle-Giler E, Ashkin A, Bass J, Ruthman C. Subdural hematoma expansion in relation to measured mean and peak systolic blood pressure: A retrospective analysis. Front Neurol. 2022 Oct 17, 13:1026471.

[12] Stanišić M, Hald J, Rasmussen IA, Pripp AH, Ivanović J, Kolstad F, et al. Volume and densities of chronic subdural haematoma obtained from CT imaging as predictors of postoperative recurrence: a prospective study of 107 operated patients. Acta Neurochir. 2013 Feb, 155(2):323–33.