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Impact of coagulopathy on the management and outcome of chronic subdural hematoma

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ABSTRACT

Background: Chronic subdural hematoma (CSDH) is one of the commonest conditions encountered in neurosurgical practice. It is a disease more commonly seen in the elderly, and its incidence is expected to rise as the population of persons above 65 years increases. With improving survival and an increasing ageing population, the use of antithrombotic medications to prevent and treat cardiovascular diseases appears to be on the rise. Though trauma is the leading cause of CSDH, these pharmacologic agents alter coagulation and have been associated with the rising recurrence of CSDH. There appears to be a complex relationship between CSDH and coagulopathy, which can lead to rebleeding or recurrence after surgical evacuation of the hematoma.

Aim/Objectives: To determine the relationship between the presence of coagulopathy and outcome in patients who underwent burr hole drainage of CSDH.

Methodology: This was a retrospective cohort study on all patients who underwent drainage of CSDH at the University of Benin Teaching Hospital over a 19-year period from June 2006 to May 2025. Clinical data were obtained from a computerised log of patients' records and analysed using STATA software version 12.

Results: One hundred and forty patients were studied. The male-to-female ratio was 4:1, and most patients were above 60 years (55.5%). Fifty-one per cent (51%) had coagulopathy - 10.07% were on antiplatelet medications and 0.71% were on warfarin. Seventy-two per cent (72%) of patients with coagulopathy had a history of trauma. Bilateral CSDH was found in thirty-six per cent (36%) of patients with coagulopathy. The patients who had coagulopathy had lower mean hematoma volumes, longer hospital stay, and accounted for all the patients (4) who required ICU admission. The presence of coagulopathy did not alter the rate of recurrence, reoperation, and GOS at discharge.

Conclusion: There is a high incidence of coagulopathy in patients who have CSDH, and most of them would require prompt evacuation irrespective of haematoma volume. Pre- and post-operative substitution of coagulation factors is associated with very good outcomes despite poor neurological status at presentation.

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Introduction

Chronic subdural hematoma (CSDH) is one of the commonest conditions encountered in neurosurgical practice. It is the slow-growing encapsulated fluid collection of blood and blood degradation products in the potential space between the dura mater and arachnoid coverings on the brain surface.[1,2] Since its first description by Wepfer in 1657, our understanding of the pathophysiology and clinical features has evolved rapidly.[3] The propagation of CSDH and its recurrence is known to result from fibrinolytic hyperactivity, which occurs within the hematoma capsule and systemic blood, amongst other mechanisms.[4] This ensuing coagulopathy contributes significantly to outcome following surgical evacuation.[5]

Globally, it is a disease more commonly seen in the elderly, with an incidence of about 8-58 patients per 100,000 population.[6] The prevalence of CSDH is expected to rise as the elderly population increases.[7]

There appears to be a complex relationship between CSDH and coagulopathy. On the one hand, CSDH can lead to coagulopathy, and on the other hand, patients with coagulopathy are at higher risk for developing CSDH, i.e., coagulopathy is a known risk factor for CSDH.[8,9] There are several recognized causes of coagulopathy, which include anticoagulant or antiplatelet therapy, liver disease, chronic kidney disease with dialysis, renal failure, or hematologic conditions that may impair coagulation, such as hemophilia. Thus, even mild trauma can lead to significant bleeding in such individuals.

In CSDH patients in whom no recognized causes for coagulopathy exist, the presence of CSDH itself is also known to cause coagulopathy.[10] Though less commonly discussed, large or recurrent CSDHs may consume clotting factors or activate fibrinolysis, leading to a secondary coagulopathy. Several studies suggest fibrinolytic activity within the hematoma membranes contributes to ongoing bleeding and expansion of the hematoma.[1] Several mechanisms have been proposed in the development of secondary coagulopathy in CSDH. These include hyperfibrinolysis in chronic subdural membranes, which produce tissue plasminogen activator (tPA), promoting fibrinolysis and preventing clot stabilization.[4] In CSDH, there is local consumption of coagulation factors within the hematoma cavity as well as inflammatory processes in the membranes, both of which may alter coagulation profiles. A secondary disseminated intravascular coagulopathy (DIC) - like picture may present in severe or recurrent cases, though it is rare. There is excessive activation of both coagulation and fibrinolysis, which are important in the progressive enlargement of CSDH.[10] The excessive activation in coagulation is mainly through the extrinsic pathway.[10] Higher levels of plasminogen at initial operation have been identified in patients subsequently experiencing CSDH recurrence. This suggests that markers of hyperfibrinolysis may be able to predict those at highest risk of recurrence and therefore help guide treatment and follow-up.[1]

The age-long treatment for CSDH, irrespective of the institution, has been evacuation through burrhole craniostomy, besides other surgical procedures.[8,11] However, surgical evacuation alone will not suffice in patients who have coagulopathy-associated CSDH, whether or not there was antecedent trauma. Thus, treatment of coagulopathy and surgical evacuation of CSDH have to be addressed in tandem in this group of patients, either preoperatively, postoperatively, or both.[12] In patients with substitution of coagulation factors, outcome has been found to be worse if patients only had post - operative correction.[4]This suggests that for safe and effective treatment of patients with coagulopathy - associated CSDH, there should be both pre-operative and post-operative substitution of coagulation factors to avert complications such as recurrence, reoperations, prolonged hospital stay, and even increased mortality following surgical evacuation.

We investigated the association between coagulopathy and outcome in CSDH patients in the University of Benin Teaching Hospital, Nigeria, a referral centre for most neurosurgical cases in our subregion.

Materials and methods

This was a retrospective cohort study on all patients who underwent surgery for CSDH at the Neurosurgical unit of the University of Benin Teaching Hospital over a 19-year period from June 2006 to May 2025. All patients were admitted via the Accident and Emergency Department, outpatient clinics, or were referred from other units within the hospital. They all underwent full neurosurgical evaluation before admission, and diagnosis was

confirmed by cranial computerized tomographic (CT) scans or Magnetic Resonance Imaging (MRI).

All the patients had burr-hole drainage of the haematoma. Single or double burr holes were sited based on the discretion of the attending surgeon. Standard frontal and parietal burr holes were made using the Hudson brace and perforator. Copious irrigation of the subdural space was done with warm antibiotic-impregnated saline until the effluent was clear. They were nursed flat post-operatively on our neurosurgical wards until mobilized, except for very few ill patients who went to the intensive care unit (ICU). No surgical drains were used postoperatively.

Out of 511 patients operated on within the study period, complete records were found in only 140 patients, with a data retrieval ratio of 27.4%. Data obtained from case notes was entered into a predesigned proforma, which was then uploaded into a computer database. These included demographic, clinical, radiological, and laboratory profiles. The history of trauma, anticoagulation therapy, diabetes, cardiac, renal, and hematologic conditions was particularly noted. We determined whether anticoagulation had been used, and all patients had a clotting profile done on admission.

Coagulopathy was defined as a derangement in clotting profile, low platelet count, use of antithrombotic medications, hematological disorder with an increased risk of bleeding, hepatic failure, or hemodialysis. Platelet counts <100,000 per microliter (normal 100,000-400,000 per microliter), Prothrombin time >15 sec (11-15 seconds), Partial Thromboplastin Time with Kaolin >45 seconds (20-45 seconds), and International Normalized Ratio (INR) >1.2 were taken as abnormal. Antithrombotic medications recorded included low-dose aspirin, clopidogrel, and warfarin. All patients who had coagulopathy were given fresh frozen plasma and tranexamic acid peri-operatively.

The patients were divided into two groups: those with and without coagulopathy. We compared, in the two groups, the patients' age, pre-operative Glasgow Coma Score (GCS), history of trauma, tobacco and alcohol use, and radiologic findings. Intraoperative hematoma volumes, reoperation, recurrence rates, need for ICU admission, duration of hospitalization, and Glasgow outcome score at discharge were also studied. Routine post-operative CT in CSDH was not performed unless indicated by neurologic deterioration or failure to improve neurologically following surgery.

Data were analyzed using STATA software version 12. Continuous variables expressed in means (± SD) were tested using Student's t-test for the difference between group means of CSDH patients without versus with coagulopathy. Categorical variables were compared using the Chi-squared test or Fisher's exact test (if any cell in a category was < 5). P-value < 0.05 was considered statistically significant.

Results

The case file records for one hundred and seventy-five patients were obtained; however, only one hundred and forty had complete data. The mean age was 60.34 years. Most patients were above 60 years (55.5%). There were one hundred and thirteen males and twenty-seven females (M: F = 4:1). The male-to-female ratio for patients without coagulopathy was 7:1, while for those with coagulopathy, it was 3:1. (See Table 1)

Table 1. Laboratory variables and the use of anti-thrombotic medications in CSDH patients with coagulopathy

Variables (n)	Frequency	Percentage
Platelet count, n= 115		
<100,000/μL	8	6.96
Prothrombin time, n= 136	37	27.21
>15 seconds		
PTTK, n= 135		
>45 seconds	23	17.04
INR, n=126		
>1.20	40	31.75
Anticoagulant, n=140		
Warfarin	1	0.71
Antiplatelet, n=139		
Clopidogrel	4	2.88
Low dose aspirin	9	6.47
Clopidogrel and low dose aspirin	1	0.72

μL: microlitre; PTTK: Partial Thromboplastin Time with Kaolin; INR: International Normalized Ratio

Only 14 patients had a history of antithrombotic use: warfarin, 1(0.71%); clopidogrel, 4(2.88%); low-dose aspirin, 9(6.47%); and both clopidogrel and low-dose aspirin, 1(0.72%). (See Table 2).

In the two groups of patients, trauma appeared to be the commonest aetiologic factor for the CSDH (71.77%). Approximately 50 % of patients took

alcohol, and 14.39% used tobacco, but there was no observable difference in the presence or absence of coagulopathy. Patients who had coagulopathy were more likely to present with lower GCS scores, though this was not statistically significant (p=0.079). Most patients, however, presented with mild deterioration in the level of consciousness with a GCS of 13-15. The presence of coagulopathy did not affect the appearance of bilateral lesions(p=0.189).

Table 2. CSDH patients without versus with coagulopathy by demographic, aetiology, lifestyle, clinical and radiologic variables

Variables	n (%) Total= 140	No Coagulopathy n= 69 (49.29%)	Coagulopathy n=71 (50.71%)	P-value
Age (years), n=137	2(1.46)	0	2	0.879
0-10	1(0.73)	0	1	π
11-20	3(2.19)	2	1	
21-30	13(9.49)	7	6	
31-40	16(11.68)	8	8	
41-50	26(18.98)	12	14	
51-60	76(55.47)	37	39	
Above 60	60.34(17.16)	60.27(15.11)	60.40(18.98)	
Age, Mean (SD), years				0.963 3μ
Gender, n= 140	27(19.29)	9	18	0.065
Female	113(80.71)	60	53	α
Male				
Aetiology, n= 124	89(71.77)	43	46	0.615
Trauma	35(28.23)	17	18	α
Others				1.000 π
Alcohol, n= 140	69(49.29)	33	36	0.733
No	71(50.71)	36	35	α
Yes				
Smoking, n=139	119(85.61)	59	60	0.705
No	20(14.39)	9	11	α
Yes				
GCS at presentation, n=128	7(5.47)	2	5	0.079
3-8	31(24.22)	10	21	π
9-12	90(70.31)	48	42	
13-15				
Bilateral haematoma, n= 135	93(68.89)	49	44	0.189
No	42(31.11)	17	25	α
Yes				
Side on CT, n= 134	50(37.31)	21	29	**0.047**
Left	42(31.34)	27	15	**α**
Right	42(31.34)	17	25	
Bilateral				

CSDH: Chronic subdural Haematoma; π: Fisher's exact test; SD: Standard Deviation; μ: Student's t-test between group means; α: Chi-squared test

In patients with coagulopathy, more left-sided lesions (42%) were observed compared to the right side (21%), while in patients without coagulopathy, more right-sided lesions (41%) were seen compared to the left (32%). The difference in sidedness was statistically significant (p=0.047). (See Table 3)

Table 3. CSDH patients without versus with coagulopathy by intraoperative findings and outcomes

Variable	n (%) Total= 140	No Coagulopathy n= 69 (49.29%)	Coagulopathy n= 71 (50.71%)	P-value
Haematoma volume, ml (n=122)	8(6.56)	6	8	0.600
0-50	62(50.82)	28	34	π
51-100	22(18.03)	13	9	
101-150	17(13.93)	10	7	
151-200	8(6.56)	3	5	
201-250	2(1.64)	1	1	
251-300	3(2.46)	2	1	
Above 300	132.77(85.74)	137.30(95.99)	127.93(73.76)	0.548 6
Mean (SD) ml				
Recurrence, n= 139	131(94.24)	65	66	0.983
No	8(5.76)	4	4	α
Yes				1.000 π
Re-operation, n= 135	1(0.74)	0	1	0.549
Craniectomy	5(3.70)	2	3	α
Old burr hole	129(95.56)	65	64	1.000
No				π
ICU Care, n= 140	136(97.14)	69	67	**0.045**
No	4(2.86)	0	4	**α**
Yes				0.120 π
GOS at discharge, n= 139	1(0.72)	0	1	0.620
	0(0)	0	0	π
Death	1(0.72)	1	0	
Persistent vegetative state	4(2.88)	1	3	
Severe disability	133(95.68)	66	67	

Moderate disability				
Good outcome				
Duration of hospitalization, n=114	15.45(9.00)	14.13(6.64)	16.52(10.46)	0.1603
Mean (SD) days				

CSDH: Chronic subdural haematoma; π: Fisher's exact test; SD: Standard Deviation; μ: Student's t-test between group means; α: Chi-squared test; ICU: Intensive Care Unit; GOS: Glasgow Outcome Score; α: range.

Table 3 compares the mean hematoma volumes, recurrence, reoperation rate, ICU admissions, GOS at discharge, and duration of hospitalization in patients with and without coagulopathy. The mean hematoma volumes measured intraoperatively were 137.30ml for patients without coagulopathy and 127.93ml for patients with coagulopathy. In the no-coagulopathy group, there were 2 outliers with 500 and 600 ml of intraoperative haematoma volume. These two patients had CSDH complicating the insertion of ventriculoperitoneal shunts. When these outliers were removed and data reanalyzed, the no-coagulopathy group mean (n=61) was 123.77 (SD 59.80) ml and the Coagulopathy group mean (n=59) was 127.93 (SD 73.76) ml. The combined mean (n=120) was 125.81 (SD 66.78) ml with no statistically significant difference observed between the two groups (p= 0.7345).

Overall, the recurrence rate was 5.76%. The presence of coagulopathy did not affect the rate of recurrence(p=0.983). Not all recurrent cases were operated. In coagulopathy-associated CSDH, all recurrences were significant enough to require reoperation. In one patient, a limited craniectomy was done to evacuate the clot.

Only patients with coagulopathy required ICU admission, and this accounted for 2.86% of the total number of patients seen (p=0.045). One (0.72%) death was recorded in a patient with coagulopathy. GOS at discharge was a good outcome in 95.6% with no difference observed in the presence or absence of coagulopathy.

The mean duration of hospital stay was 16.52 days in patients with coagulopathy and 14.13 days in patients without coagulopathy. The difference was not statistically significant (p=0.1603)

Discussion

Defining coagulopathy can be challenging due to the several hematological parameters involved.[13] In the study of coagulopathy associated with CSDH, common investigations include simple tests ranging from clotting profile and complete blood counts to complex ones such as clotting factor assay and thromboelastography.[10]

The incidence of chronic subdural hematoma rises sharply with advancing age, irrespective of aetiology.[14, 9,15,16,17] Our study shows that coagulopathy associated with CSDH was also common in the 6th decade, and the male-to-female ratio was 3:1. There was no statistically significant difference in the mean ages of patients with and without coagulopathy (p=0.963). This likely meant that the pattern of age-related hemostatic changes, comorbidities, liver/kidney dysfunction, and local hematoma-driven fibrinolysis in the hematoma membrane was similar. [18,19,20,21]

In this study, it was found, surprisingly, that only 10.71% of patients operated for CSDH were on antithrombotic agents: more patients were on antiplatelets compared to warfarin. It was expected that a higher number of elderly patients would be on cardio- and neuroprotective antithrombotic medications. This strongly contrasted previous studies, which observed a higher incidence of antithrombotic use, which ranged between 29 and 43%.[22,23,24].Thus, with more than fifty percent (50%) of patients having coagulopathy, the strong association between coagulopathy and CSDH is thus underscored.

In large studies, approximately two-thirds of patients had a history of trauma.[25] Stroobandt et al found a trauma rate of 80%.[26] This was similar to our finding in which 71.8% were ascertained to have a traumatic origin. This was in contrast to Konig et al, who found trauma in only 48%.[12] This rate depends, to a large extent, on the number of patients in whom a history of trauma could be recalled.

The presence of coagulopathy appeared to play a role in the level of consciousness at presentation. More patients with coagulopathy in our study tended to present with lower GCS scores. As expected, this should relate to larger hematoma volumes. This correlated well with our study in which we found that patients with coagulopathy had higher mean hematoma volumes only after correcting for outliers (127.93 ml vs 123.77 ml ; p=0.7345). Is it possible that other factors besides the volumetric effect are responsible for the lower GCS at presentation?

The incidence of bilateral hematoma in patients with coagulopathy in our study was 36.2%. This was much higher compared to previous reports, which ranged from 17.4% to 25.52%.[12,27,28]. Our findings, however, appear to be congruent with those of Oyama et al, who postulated that bilateral CSDHs tended to occur more frequently in patients with coagulation abnormalities.[29] There is, however, no clear-cut consensus on the role of anticoagulation in the bilateral occurrence of CSDH. Garba Sunday et al[30] in their review article, noted that anticoagulation contributed to bilateral CSDH, while Yu-Hua Huang[28] found no association. In our environment, in which routine brain scans are not done and late presentation is common, this may have contributed to the increased frequency of bilateral lesions seen.

In our study, we found that left-sided lesions were more common. Patients with coagulopathy had twice as many left-sided lesions as right-sided lesions. In patients without coagulopathy, right-sided lesions were more common. This difference was statistically significant (p=0.047). MacFarlane et al had previously noted that left-sided lesions were more common.[31] With the higher occurrence of left-sided lesions, it is important to ask if this is a result of a biological or anatomical process. Right-sided (non-dominant) lesions are generally not associated with communication difficulties or impairment of dominant hand function but rather more subtle symptoms and signs, such as inattention and geographical dyspraxia. This may therefore lead to their underdiagnosis rather than a true increase in left-sided lesions. Coagulopathy resulting in larger hematomas and more significant neurologic deterioration may explain why left-sided lesions were more commonly diagnosed.

With burr hole evacuation of CSDH, there is usually a rapid improvement in the clinical status of patients. Out of seven patients who presented with a GCS<8, only four eventually required ICU admission. Only patients with coagulopathy were admitted into the ICU, and for this reason, they tended to have a longer duration of hospitalization. The longer duration of hospitalization, including ICU care, means that patients with coagulopathy had higher costs of treatment, which has a significant impact in a low-resource setting.[32]

Recurrence of CSDH after the first burr hole craniostomy is not rare. The rate of clinically significant recurrence that required surgical evacuation ranged from 2-37% after initial burr hole evacuation with or without postoperative drain.[33,34] Its causes range from patient-specific risk factors, radiologic risk factors, surgical risk factors, and subdural fluid characteristics.[33] In our study, the recurrence rate was 5.54% and there was no statistically significant difference between the presence or absence of coagulopathy (p=0.983). This is similar to findings by Vinredai et al in their review paper, where they observed that the use of anticoagulants or antiplatelet did not lead to an increase in the rate of recurrence of CSDH.[33] Toshiyuki et al also found that there were no significant differences in the incidence of radiographic deterioration or reoperation of ipsilateral or contralateral hematomas between patients with and without antithrombotic therapy after surgical treatment of unilateral CSDH.[24] Forster et al, however, observed that perioperative antithrombotic therapy led to higher recurrence and worse outcomes.[35] Our study also showed that the presence of coagulopathy did not lead to a higher reoperation rate (p=0.549). This is probably because patients with laboratory documented coagulopathy associated with CSDH had pre- and post-operative substitution of coagulation factors.

Coagulopathy has been identified as an independent risk factor for mortality.[5]Mortality rates in CSDH patients have been reported to be as high as 16.7%.[15] In this study, only one mortality (0.72%) was observed, and it was in a patient with coagulopathy. The perioperative correction of coagulopathy in patients undergoing surgery may have significantly reduced its impact. This makes it extremely important that all patients with CSDH should have coagulation screening done. When derangements are seen, these should be corrected as early as possible before surgery. The overall outcome following surgery for CSDH is, however, favourable.[25]

The presence of coagulopathy in chronic subdural haematoma (CSH) requires correction of coagulation to facilitate surgery.[12] In virtually all our patients who had derangements in clotting profile, tranexamic acid was commenced, and fresh frozen plasma (FFP) was given before and after surgery. Fresh whole blood was also given when FFP was not available or the patient was noted to have low hematocrit. In hospitals where facilities for advanced coagulation studies, including clotting factor assays,

could be done, substitution of the deficient factor pre- and postoperatively has been found to improve outcome.[12] Fresh frozen plasma is rich in factors II, V, VIII, IX, X, XI, and antithrombin III and is readily available in our environment. Since there is a high prevalence of coagulopathy in CSDH patients, it is recommended that, in emergency cases, FFP be administered if there is not enough time to carry out laboratory tests. This is especially important in low-resource settings where delayed referral and late presentation are common.

Limitations of the Study

Due to challenges with diagnostic laboratory capability, the definition of coagulopathy was narrow.[36] It is thus possible that some patients with subclinical coagulopathy would have been missed, leading to a lower incidence of coagulopathy. The relatively small sample size may have impacted the power of the study.

Conclusion

There is a high incidence of coagulopathy in patients who have CSDH requiring surgical evacuation, irrespective of haematoma volume. Our results suggest that patients with coagulopathy are more likely to present on the left side, have lower admission GCS scores, require ICU admission after surgery, and have longer hospital stays. The presence of coagulopathy did not increase the frequency of bilateral lesions, clinical recurrence after surgery, or need for reoperation. This therefore highlights the need for pre- and post-operative substitution of coagulation factors, which improves outcomes despite poor neurological status at presentation.

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