

Case Report

Decision-making for emergency craniotomy in traumatic intracranial hemorrhage with severe thrombocytopenia and coagulopathy in a patient with suspected acute leukemia: A case-based review

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ABSTRACT

Background: Emergency craniotomy for traumatic intracranial hemorrhage is time-dependent, yet surgery may be futile when profound thrombocytopenia and coagulopathy prevent hemostasis and severe brain swelling is already present.

Case Description: A young adult man with traumatic intracranial hemorrhage rapidly deteriorated to coma (Glasgow coma scale-3). Admission laboratories showed white blood cell count 72,300/ μ L, platelet count 12,000/ μ L, and prothrombin time-international normalized ratio 1.59, raising concern for an underlying hematologic disorder, including acute leukemia, with coagulopathy. Head computed tomography demonstrated a large frontal intraparenchymal hemorrhage with traumatic subarachnoid hemorrhage, intraventricular extension, and imaging features consistent with severe diffuse brain swelling. After intubation, emergency craniotomy was undertaken with perioperative transfusion of red blood cells, fresh frozen plasma, and platelets. Diffuse bleeding and marked brain swelling prevented durable hemostasis, and the patient died.

Conclusion: When neurotrauma meets hematologic catastrophe, the decision to operate should incorporate both neurologic salvageability and the realistic probability of achieving usable hemostasis within the available time. Early estimation of attainable platelet correction, together with rapid coagulation assessment including fibrinogen when available, parallel hematology support, and a damage-control operative plan with pre-specified stopping rules may help frame surgery as a time-limited trial rather than a binary choice.

Keywords: Acute leukemia, Coagulopathy, Damage control neurosurgery, Thrombocytopenia, Traumatic intracranial hemorrhage

INTRODUCTION

Emergency surgery for traumatic intracranial hemorrhage is a time-dependent life-saving intervention aimed at relieving mass effect and limiting secondary brain injury, and the surgical indications for traumatic parenchymal lesions and the management principles for severe traumatic brain injury (TBI) have been summarized in several guidelines.^[5,6,39] These recommendations,

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however, rest implicitly on the assumption that some degree of hemostasis can be achieved. In patients who already have severe thrombocytopenia or coagulopathy on arrival, failure of intraoperative hemostasis can itself be fatal, and additional bleeding and brain swelling caused by the procedure may rapidly destabilize both systemic physiology and the intracranial environment.

In practice, a platelet count (Plt) of at least 100,000/ μ L is widely shared as the target for intracranial hemorrhage requiring neurosurgical intervention, and national guidance in Japan likewise recommends maintaining a similar level in traumatic intracranial hemorrhage.^[11,25,41] At the same time, in TBI, low admission Plts and coagulopathy are associated with hematoma progression and death,^[15,26,38] and in emergency hematoma evacuation performed in patients with preoperative thrombocytopenia, surgery may technically be completed while severe brain edema still determines the outcome.^[43] It is therefore not enough to know the guideline threshold. One also has to think, at the same time, about how far the count can be raised within a short period and what kind of operation should be chosen if it cannot be raised enough.

The situation becomes even more complicated when the blood tests obtained on arrival raise suspicion of a hematologic disorder, including acute leukemia. In addition to thrombocytopenia, disseminated intravascular coagulation (DIC), hypofibrinogenemia, hyperfibrinolysis, and microcirculatory dysfunction related to hyperleukocytosis may overlap and contribute to the onset or progression of intracranial hemorrhage and to a high case-fatality rate.^[7-9,20,37] Few reports directly address real-time neurosurgical decision-making when traumatic intracranial hemorrhage coexists with suspected hematologic catastrophe before a definitive hematologic diagnosis is available. The present case combined severe head trauma with already limited neurologic salvageability and severe thrombocytopenia and coagulopathy first recognized at the time of transfer. In this article, we organize the decision points from initial trauma care to the decision for emergency surgery as a case-based review based on the CAsE-BAsed REview sTandards (CABARET),^[2] and we describe the case using elements of the CARE guideline.^[12]

CASE REPORT

A 23-year-old man with no known hematologic disease was found collapsed outside his home on the day of presentation, with bleeding from the right frontal region. The last-known-well time was unknown. When emergency medical services (EMS) arrived, he had aphasia with Japan Coma Scale (JCS) I-3. During transport, he deteriorated to JCS III-100 and developed right hemiparesis. At the referring hospital, the vital signs were heart rate 120/min, blood pressure 150/90 mmHg, peripheral oxygen saturation

(SpO₂) 96%, pupils 3 mm/3 mm, and consciousness JCS III-200. Intracranial hemorrhage was diagnosed, and he was transferred to the receiving tertiary hospital.

On arrival at the receiving tertiary hospital, consciousness was JCS III-300 and Glasgow coma scale (GCS)-3, with pupils 3 mm/3 mm. Respiratory rate was 26/min, heart rate 110/min, blood pressure 165/88 mmHg, and temperature 37.5°C. Because respiratory status was poor, endotracheal intubation was performed shortly after arrival and mechanical ventilation was started.

The timeline is summarized in Table 1.

Initial laboratories were notable for white blood cell (WBC) count 72,300/ μ L, Plt 12,000/ μ L, and prothrombin time-international normalized ratio (PT-INR) 1.59, raising concern for an underlying hematologic disorder, including acute leukemia, with coagulopathy.

Laboratory data obtained on arrival are summarized in Table 2.

Head computed tomography (CT) showed a large intraparenchymal hemorrhage centered in the left frontal lobe and intraventricular hemorrhage, with associated traumatic subarachnoid hemorrhage. Hemorrhagic lesions were also present in the left and right temporal lobes. The sulci were diffusely effaced, and the basal cisterns were poorly visualized, strongly suggesting diffuse brain swelling. The preoperative head CT is shown in Figure 1.

Anesthesia was started approximately 80 min after arrival, and emergency craniotomy for hematoma evacuation and decompression began about 2 h after arrival. Perioperative transfusion consisted of 4 units of red blood cells, 4 units of fresh frozen plasma (FFP), and 20 units of platelets. During surgery, hemostasis was difficult due to a diffuse bleeding tendency, and marked brain swelling was present. Surgery ended about 4 h after arrival. Because the postoperative general condition was extremely poor, repeat CT was not performed. He was managed in the high care unit, but death was confirmed later the same day.

Postoperative laboratories showed Plt 54,000/ μ L with persistent coagulopathy (PT-INR 1.78).

DISCUSSION

Literature search methods

This article is a focused, non-systematic case-based review anchored to the clinical questions raised by the case. To make the review process transparent, we referred to CABARET,^[2] and the case description was organized with elements of the CARE guideline.^[12] PubMed/MEDLINE was searched on February 28, 2026, mainly in English, and relevant domestic guidance was checked by hand in the original portable document format (PDF) documents.

Table 1: Timeline of clinical course (relative timing).

Relative time	Phase	Key findings/interventions
Presentation day	Found collapsed outside home	Emergency call; last-known-well time unknown
At scene	EMS assessment	JCS I-3; aphasia
During transport	Transport	JCS III-100; right hemiparesis
Arrival at referring hospital	Initial evaluation	HR 120/min; BP 150/90 mmHg; SpO ₂ 96%; pupils 3 mm/3 mm; JCS III-200; intracranial hemorrhage diagnosed; transferred to receiving tertiary hospital
Arrival at receiving tertiary hospital	Initial evaluation	JCS III-300; pupils 3 mm/3 mm; RR 26/min; HR 110/min; BP 165/88 mmHg; temperature 37.5°C
Shortly after arrival	Airway management	Endotracheal intubation; mechanical ventilation started
Approximately 80 min after arrival	Anesthesia	Induction started
About 2 h after arrival	Surgery	Emergency craniotomy started (hematoma evacuation and decompression)
About 4 h after arrival	Surgery	Procedure ended
Shortly after surgery	Anesthesia	Anesthesia ended
Later the same day	Outcome	Death confirmed

EMS: Emergency medical services, JCS: Japan coma scale, RR: Respiratory rate, HR: Heart rate, BP: Blood pressure, SpO₂: Oxygen saturation by pulse oximetry

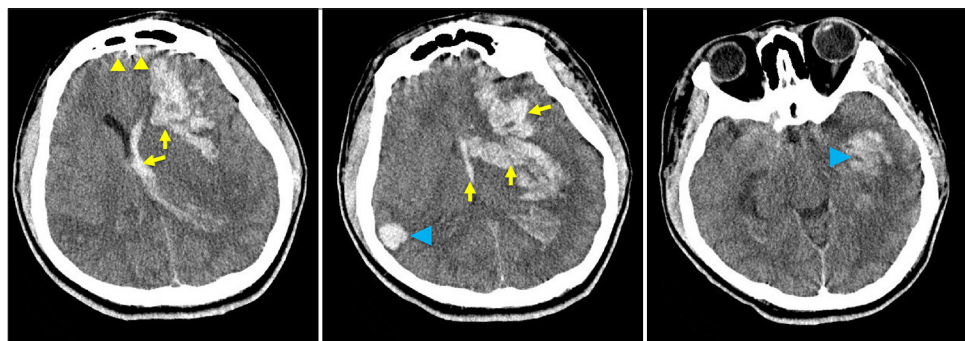


Figure 1: Preoperative noncontrast head computed tomography (CT). The left and middle axial CT images show a large left frontal intraparenchymal hemorrhage with intraventricular extension (yellow arrows). Traumatic subarachnoid hemorrhage is indicated by yellow arrowheads in the left image. Additional hemorrhagic lesions in the temporal lobes are indicated by blue arrowheads in the middle and right images. Diffuse sulcal effacement and poor visualization of the basal cisterns suggest severe diffuse brain swelling.

The search focused on three domains: traumatic intracranial hemorrhage with thrombocytopenia or coagulopathy in relation to surgery; leukemia or other hematologic malignancy with intracranial hemorrhage and hemostasis; and damage control neurosurgery (DCNS) or staged surgery. Priority was given to primary sources and guideline documents that directly informed bedside decision-making in this case.

Priority-core literature domains are summarized in Table 3.

Estimating neurologic salvageability and stating the purpose of surgery

In this case, the first question should not have been simply, “Should we operate on this hemorrhage?” but rather, “What

do we want to achieve if we operate?” Surgical indications for traumatic parenchymal lesions are usually framed in terms of mass effect, progressive neurologic deterioration, and raised intracranial pressure,^[5,6,39] and in progressive hemorrhagic lesions, early intervention may be the only chance of survival. In addition, in traumatic intracerebral hemorrhage, early progressive hemorrhagic injury often changes the clinical course and management,^[44] and acute traumatic subdural hematoma with thrombocytopenia has been reported to enlarge more readily before surgery.^[42]

In this patient, aphasia was present when EMS arrived, right hemiparesis appeared in the ambulance, and consciousness worsened rapidly from JCS I-3 to III-100, III-200, and III-300 during the short interval from first contact to transfer from the referring hospital to the receiving hospital. This suggested

Table 2: Laboratory findings from the arrival sample and the postoperative sample.

Parameter	Available at decision	Later result from arrival sample	After surgery	Unit
WBC	72,300	–	107,550	/μL
RBC	2.85×10 ⁶	–	2.42×10 ⁶	/μL
Hb	8.5	–	7.4	g/dL
Ht	27.5	–	22.9	%
Plt	12,000	–	54,000	/μL
PT-INR	1.59	–	1.78	-
D-dimer	30.0	–	30.0	as reported
Fibrinogen	–	110	–	mg/dL
FDP	–	133.0	–	as reported
Peripheral smear abnormal cells	–	95.4	–	%
AST (GOT)	41	–	84	IU/L
ALT (GPT)	37	–	34	IU/L
Gamma-GTP	78	–	64	IU/L
LDH	1,450	–	1,929	IU/L
ALP	95	–	74	IU/L
BUN	18.1	–	23.3	mg/dL
Creatinine	0.90	–	1.97	mg/dL
Uric acid	6.20	–	7.80	mg/dL
Na	135	–	134	mEq/L
K	3.3	–	5.6	mEq/L
Cl	96	–	98	mEq/L
Ca	9.1	–	–	mg/dL
CRP	15.8	–	13.4	mg/dL
NT-proBNP	561	–	–	pg/mL

WBC: White blood cell count, RBC: Red blood cell count, Hb: Hemoglobin, Ht: Hematocrit, Plt: Platelet count, PT-INR: prothrombin time-international normalized ratio, FDP: Fibrin/fibrinogen degradation products, AST: Aspartate aminotransferase, ALT: Alanine aminotransferase, gamma-GTP: Gamma-glutamyl transferase, LDH: Lactate dehydrogenase, ALP: Alkaline phosphatase, BUN: blood urea nitrogen, Na: sodium, K: Potassium, Cl: Chloride, Ca: Calcium, CRP: C-reactive protein, NT-proBNP: N-terminal pro-B-type natriuretic peptide. Postoperative sample obtained after surgery; exact sampling time was not recorded. Fibrinogen, fibrinogen degradation products, and peripheral smear findings were derived from the arrival sample but were reported after the initial operative decision. A dash (–) denotes a result not yet available at the operative decision (first result column), a result already shown in the preceding column and therefore not repeated (middle column), or a postoperative result unavailable for this report (last result column). Dashes do not denote zero

Table 3: Priority-core literature domains used in this case-based review.

Domain	No. of papers	Main contribution to this review
Traumatic brain injury coagulopathy/transfusion/surgery	15	Platelet thresholds, hematoma progression, perioperative hemostasis, fresh frozen plasma/prothrombin complex concentrate/rotational thromboelastometry, and operative risk under coagulopathy
Leukemia/hematologic malignancy-associated intracranial hemorrhage	18	Incidence, prognostic factors, transfusion limits, acute promyelocytic leukemia-specific early hemorrhage, and survival after intracranial hemorrhage
Damage control/surgical strategy	4	Abbreviated neurosurgical goals, staged strategy, and civilian applications of neuro-damage control

that a mass lesion with focal signs had moved rapidly toward brainstem dysfunction. Nonoperative management was therefore strongly expected to lead to death within a short time, and it is hard to deny that emergency craniotomy was a reasonable option.

At the same time, coma with a GCS of 3 on arrival and severe brain swelling indicated that the neurologic chance of survival was already extremely low. In intracranial hemorrhage requiring intensive care unit admission in patients with acute leukemia, stupor/coma is independently associated with poor

outcome,^[16] and across the broader literature on intracranial hemorrhage associated with hematologic malignancy, low GCS is consistently linked to poor prognosis.^[7,31] What needed to be shared before surgery, therefore, was not only whether an indication was present, but whether the purpose of surgery was avoidance of herniation, short-term physiologic stabilization, or assessment of irreversibility.

Hemostatic target (Plt 100,000/ μ L) and whether it could be reached

The idea that Plt 100,000/ μ L should be targeted in intracranial hemorrhage or neurosurgical procedures has been repeated in domestic and international guidance.^[11,25,41] Observational studies also support an association between low Plts and progression of traumatic intracranial hemorrhage,^[38] and a 2025 meta-analysis on admission coagulopathy and hematoma progression in TBI reported that abnormalities in Plt, PT-INR, fibrinogen, and D-dimer were associated with progressive hemorrhagic injury.^[26] In the setting of emergency craniotomy, however, the more practical question is not the target itself but how far one can get within the limited time available.

Using the assumptions adopted in the Ministry of Health, Labor, and Welfare guidance, namely, circulating blood volume of 70 mL/kg, a splenic sequestration correction factor of 2/3, and at least 0.2×10^{11} platelets per unit of platelet concentrate, the predicted platelet increment immediately after transfusion can be approximated from body weight (BW) (kg) and the number of administered units U:^[25]

By rearranging the same formula, the number of units required to achieve a target increment can also be approximated.^[25]

In this case, the recorded BW was 98 kg, and even under ideal conditions the expected increase from 20 units of platelet concentrate was only about 39,000/ μ L. Starting from an initial Plt count of 12,000/ μ L, reaching 100,000/ μ L would therefore have required about 45 units even under ideal assumptions, which made the actually administered 20 units insufficient from the outset. In fact, the postoperative Plt count was 54,000/ μ L, close to the theoretical value of approximately 51,000/ μ L. This means that the Plt rose only about as much as predicted, yet still did not reach the range, in which surgery could be considered relatively safer. In other words, the point of failure in this case was not platelet refractoriness alone, but the fact that, given the patient's body size and starting value, the target was difficult to reach from the beginning.

That a higher Plt does not translate straightforwardly into a better outcome has also been shown in intracranial hemorrhage in patients with leukemia. Chern *et al.* reported that even with aggressive transfusion targeting Plt 50,000/ μ L

after intracranial hemorrhage in leukemia patients, only some patients reached the target, and reaching the target itself was not simply associated with 30-day mortality.^[9] A nested case-control study on platelet transfusion and the occurrence of intracranial hemorrhage in acute leukemia likewise suggested that bleeding risk cannot be explained by Plt alone.^[10]

For that reason, "Plt 100,000/ μ L" remains an important guide in trauma neurosurgery. But when this level is unlikely to be reached within the available time, as in the present case, it should not be fixed as a yes-or-no threshold for surgery. It needs to be incorporated into decisions about lower-burden operative strategies and into the design of a time-limited trial. New approaches are beginning to appear, such as randomized trials examining early transfusion of cold-stored platelets,^[27] but the evidence is not yet at a stage where it can be applied directly to cases like this one with suspected hematologic disease.

Do not focus on Plt alone: Overlap between traumatic coagulopathy and leukemia-related coagulopathy

At the time of operative decision-making, the actionable data were marked thrombocytopenia, leukocytosis, PT-INR prolongation, D-dimer 30.0, and lactate dehydrogenase (LDH) 1,450 IU/L. Outsourced results that became available later showed fibrinogen 110 mg/dL, fibrin/fibrinogen degradation products (FDP) 133.0, and 95.4% abnormal cells on the peripheral blood smear. These later results are used here for retrospective phenotypic reinterpretation rather than to retroactively judge the bedside decision. Taken together, they make it more likely that the case involved not only simple thrombocytopenia but also a hemostatic disorder beyond platelets and an underlying hematologic disease, including acute leukemia. Coagulopathy after TBI is itself an independent adverse prognostic factor,^[15,47] and the European guideline also emphasizes that major bleeding and coagulopathy should be addressed in parallel.^[35]

The same overlap matters when one thinks about intraoperative brain swelling. Uncorrected traumatic coagulopathy has been strongly associated with severe brain swelling during decompressive surgery,^[45] and high preoperative FDP may predict severe hypotension after dural opening.^[19] Serial studies in craniotomy for TBI have also shown that early hypofibrinogenemia and hyperfibrinolysis or fibrinolysis shutdown are associated with in-hospital mortality,^[32] and even in isolated head trauma, fibrinolytic markers such as D-dimer correlate with outcome.^[40] In this case, the fibrinogen level of 110 mg/dL and FDP 133.0 that became available later strongly support, at least in retrospect, the presence of consumptive coagulopathy with fibrinolysis.

Once acute leukemia is added to the picture, the situation becomes even more complicated. Large retrospective studies of intracranial hemorrhage associated with hematologic malignancy have shown high short-term mortality, and prolonged prothrombin time, subarachnoid hemorrhage, multiple lesions, and low GCS have repeatedly been identified as adverse factors.^[7,8,20,31,48] In acute leukemia, reported risk factors for intracranial hemorrhage include hyperleukocytosis, prolonged prothrombin time, low fibrinogen, and low albumin,^[21,24,52] and in *de novo* acute leukemia the combination of high WBC count and high FDP has been described as a strong risk factor for the early intracranial hemorrhage.^[17] The WBC count of 72,300/ μ L and FDP 133.0 in the present case fit this high-risk phenotype. In addition, the peripheral smear was composed of 95.4% abnormal cells, with only very small numbers of mature neutrophils, monocytes, and lymphocytes, and with nucleated red cells present. These findings are difficult to explain by reactive leukocytosis alone and, in retrospect, support the bedside impression at arrival that a hematologic disorder including acute leukemia might be present.

Acute promyelocytic leukemia (APL) is a representative condition in which intracranial hemorrhage is a major cause of early death, and fibrinogen below 1.5 g/L, hyperleukocytosis, prolonged international normalized ratio, and high LDH are associated with early death and fatal intracranial hemorrhage.^[13,14,37] No data in this case prove APL. Still, the fibrinogen level of 110 mg/dL is below the 1.5 g/L threshold, and, together with FDP 133.0, marked leukocytosis, and elevated LDH, it supports retrospective concern for a leukemia-associated coagulopathic phenotype. APL remained an important differential diagnosis, but it could not be confirmed. Once leukemia was suspected from the arrival blood tests, there was value in thinking about hemostatic optimization on that assumption while contacting hematology in parallel.

The temporal sequence in this case, however, cannot be fixed in only one direction. Bleeding from the right frontal region and traumatic subarachnoid hemorrhage support an interpretation centered on trauma, but the last-known-well time was unknown, and severe thrombocytopenia, prolonged PT-INR, hypofibrinogenemia, high FDP, and a peripheral smear dominated by abnormal cells were all documented retrospectively. It therefore remains possible that intracranial hemorrhage related to a hematologic disorder, including acute leukemia, occurred first, causing impaired consciousness or a fall, and that the frontal injury and traumatic subarachnoid hemorrhage followed.^[7,8,13,14,16,17,20,21,24,37,48,52] In other words, this case is better understood not as a simple choice between “pure traumatic intracranial hemorrhage” and “pure leukemia-associated intracranial hemorrhage,” but as a mixed condition in which trauma was superimposed on an

intrinsic bleeding tendency, or intrinsic bleeding precipitated a traumatic fall.

What should run in parallel: Goal-directed hemostatic resuscitation

In recent years, the question of what should run in parallel in TBI with failed hemostasis has increasingly been organized under the concept of goal-directed coagulation management. In a study implementing rotational thromboelastometry in craniotomy for TBI, management was more responsive than management based on standard coagulation tests alone with respect to modifiable factors such as blood product use and progressive hemorrhagic injury.^[33] Another report suggested that adding prothrombin complex concentrate to FFP shortened the time to craniotomy.^[18]

Even so, a strategy of simply “giving more FFP” is not straightforward. A prospective trial of low-dose early FFP in severe TBI was stopped early because it showed an increase in delayed traumatic hematoma,^[50] and another retrospective study found that greater perioperative FFP transfusion was associated with death or poor functional outcome.^[51] In a case with severe coagulopathy like the present one, therefore, increasing FFP should not be treated as automatically correct. What needs to be supplemented should be judged from PT-INR, fibrinogen, viscoelastic testing, and the speed of bleeding.

The roles of recombinant activated factor VII (rFVIIa) and thromboelastometry as emergency rescue measures have been discussed for some time,^[1] and rFVIIa has been reported before emergent craniotomy in coagulopathic blunt trauma.^[4] These are not treatments that can be generalized as standard care, but rescue options that have to be weighed against thromboembolic complications, cost, and limited evidence.

Applied to this case, the minimum three items that needed to be defined during the initial resuscitation were Plt, PT-INR, and fibrinogen, although fibrinogen was not immediately available in this case. If possible, D-dimer/FDP and viscoelastic testing should have been added to identify whether the main bottleneck lay in platelet replacement, fibrinogen replacement, or coagulation factor replacement. In fact, the later results of fibrinogen 110 mg/dL and FDP 133.0 suggest that the bottleneck was not platelet deficiency alone but also hypofibrinogenemia and marked fibrinolytic activation or degradation product accumulation. Put differently, a one-line understanding that “if Plt rises, surgery becomes safer” was not enough. Whether fibrinogen could be replaced, how quickly, with what product, and how the operative plan and withdrawal criteria should be reduced if replacement could not be achieved should have been central to the preoperative family discussion and team agreement.

A reduced operative plan: Where DCNS fits

When hemostasis is unlikely to be achieved, the operative plan should not be built backward from an ideal “complete” procedure. The priority is to avoid increasing the bleeding burden and surgical insult, and to minimize the procedure step by step. DCNS, proposed by Rosenfeld, is the idea of performing only the minimum intervention needed to sustain life in a short time and prioritizing restoration of overall physiologic status.^[34] The concept later spread from military and resource-limited settings to civilian trauma,^[23,36] and more recent reports place decompressive hemicraniectomy for severe cranial injury in civilian trauma within a damage control framework.^[3]

What became problematic during surgery in this case was not so much that a local bleeding point could not be found, but that diffuse oozing and severe brain swelling progressed at the same time. Reports linking uncorrected traumatic coagulopathy to severe brain swelling,^[45] together with reports that brain edema can become the main determinant of outcome in surgical cases with preoperative thrombocytopenia,^[43] suggest that pursuing complete evacuation may instead widen the bleeding surface.

Accordingly, in similar cases, the practical points that should be shared before surgery are as follows: (i) whether the aim is limited to avoidance of herniation; (ii) at what point escalation of the procedure should stop; and (iii) whether “closing” should be accepted not as defeat but as part of the plan when hemostatic indicators do not improve intraoperatively. In practical terms, stopping rules might include failure to obtain macroscopic hemostasis after abbreviated decompression, uncontrolled diffuse oozing despite transfusion escalation, or swelling that precludes safe closure. DCNS does not guarantee a good outcome in a case like this, but it provides a framework that reduces hindsight regret and makes team decision-making explicit.

How this case fits within the literature on intracranial hemorrhage associated with hematologic malignancy

The literature on intracranial hemorrhage associated with hematologic malignancy provides another angle from which to view this case. In a cohort study published in *BMC Medicine*, 30-day mortality was high, but the results also suggested that selected patients may derive some benefit from surgery.^[7] On the other hand, studies of platelet transfusion management after intracranial hemorrhage in leukemia patients show that reaching the target Plt count does not make outcome improvement self-evident,^[9,28] and studies predicting intracranial hemorrhage in acute leukemia place WBC count, coagulation abnormality, and low fibrinogen above Plt in terms of importance.^[21,24]

Studies of chronic and acute subdural hematoma in patients with hematologic malignancy likewise show that surgery is

not always contraindicated, but low Plt and low hemoglobin are associated with poor outcome, and the reoperation rate is high.^[46] In other words, “do not operate because it is a hematologic disease” and “operate because there is a hematoma” are both too simple. Urgency on the neurocritical side and the likelihood that hemostasis can actually be achieved have to be weighed at the same time.

Recent papers have also tried to stratify prognosis in acute leukemia-associated intracranial hemorrhage. Older work proposed a risk score model for fatal intracranial hemorrhage,^[20] and more recent studies have reported associations between the Chinese DIC Scoring System score and 30-day mortality^[49] as well as a new risk prediction model in adults with acute leukemia.^[22] These models, however, were developed mainly for intracranial hemorrhage arising in the natural history of hematologic malignancy and cannot be applied directly to traumatic intracranial hemorrhage. In the present case, they are better read as showing that a cluster of risk factors, namely, hyperleukocytosis, coagulopathy, low fibrinogen, and coma, may further worsen hemostatic failure after trauma.

The clinical picture of intracranial hemorrhage in acute leukemia also varies across centers and disease subtypes. An epidemiologic study from Thailand,^[29] a single-center study from China,^[52] a risk study in adults with acute leukemia,^[24] and an intensive care unit cohort^[20] all indicate that outcome changes markedly according to the type of hemorrhage and the general condition. When traumatic subarachnoid hemorrhage, intraparenchymal hemorrhage, intraventricular hemorrhage, coma, and coagulopathy are all present together, as in this case, the patient is likely to fall into the most unfavorable part of the spectrum even within leukemia-associated intracranial hemorrhage.

What can be said when the case is reconsidered as a time-limited trial

Bringing these points back to the present case, at least three things could have been said before surgery. First, from the neurologic side, nonoperative care was likely to be fatal within a short time, so emergency craniotomy was a reasonable option. Second, from the hemostatic side, reaching Plt 100,000/ μ L was not realistic either theoretically or practically, and the later findings of fibrinogen 110 mg/dL and FDP 133.0 suggest that hypofibrinogenemia and fibrinolytic abnormality would probably have remained even if the Plt count rose. Third, for that reason, the operation needed to be positioned not as definitive surgery but as a time-limited trial with an explicit purpose, time frame, and withdrawal criteria.^[30]

This formulation is not meant to justify the operation, nor to deny it. The most practical lesson that can be taken back to the bedside from this case is to restate, under the information

limits that existed at the time, what was known, what was not known, and which targets appeared realistically reachable.

This manuscript is based on the available clinical information and the literature reviewed. Fibrinogen, FDP, and peripheral smear findings were derived from the arrival sample but became available only after the preoperative decision, so the interpretation in this paper remains vulnerable to hindsight bias. In addition, the mechanism of injury was not witnessed and the last-known-well time was unknown, so the temporal sequence cannot be settled with certainty: trauma may have been the main cause of the intracranial hemorrhage, or hemorrhage related to hematologic disease may have come first and led to a fall and frontal injury. Activated partial thromboplastin time, blood gas data (pH and lactate), serial calcium and temperature data, and viscoelastic testing were not confirmed, so the relative contributions of traumatic coagulopathy and hematologic disease-related coagulopathy cannot be separated quantitatively. Imaging was not analyzed quantitatively for hematoma volume, degree of midline shift, or basal cistern compression. Finally, bone marrow examination, immunophenotyping, and cytogenetic or molecular testing were not available, so the subtype of acute leukemia, especially a disease-specific process such as APL, could not be confirmed.

CONCLUSION

In traumatic intracranial hemorrhage with severe thrombocytopenia and PT-INR prolongation that raise concern for an underlying hematologic disorder, the decision about emergency craniotomy should weigh neurologic salvageability against the realistic chance of achieving usable hemostasis within the available time. When platelet correction is unlikely to reach a safer range and additional coagulopathy is suspected or later confirmed, surgery is better framed as a time-limited trial with parallel hematology support, a reduced operative plan, and explicit stopping rules rather than as a binary choice between full intervention and no intervention.

Ethical approval: The Institutional Review Board approval is not required.

Declaration of patient consent: The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given consent for their images and other clinical information to be reported in the journal. The patient understands that the patient's names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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