

Recurrent Chronic Subdural Hematoma Complicating HIV/HCV Cirrhosis: A “Repair Window” Therapeutic Hypothesis – Case Report

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Case Report

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Abstract

Background

Chronic subdural hematoma (CSDH) management is particularly challenging in patients with dual abnormalities of coagulation and immune function, such as those with HIV/HCV-related liver cirrhosis and thrombocytopenia. The high risk of recurrence necessitates innovative perioperative strategies, but evidence for any specific approach is lacking.

Case Presentation

We report a case of a 43-year-old male with HIV/HCV-related cirrhosis, hypersplenism, and severe thrombocytopenia, who experienced multiple early recurrences of CSDH following two separate burr hole drainage procedures. After the second recurrence of a left-sided hematoma, a multidisciplinary team implemented a combined therapeutic regimen during the hypothesized early postoperative “repair window.”

Intervention and Outcomes

The regimen, initiated immediately after the repeated burr hole drainage, consisted of: 1) recombinant human thrombopoietin (rhTPO) 300 U/kg subcutaneously daily for 7 days; 2) oral atorvastatin 20 mg daily; and 3) adjunctive hyperbaric oxygen therapy (HBOT) at 2.0 ATA for 60 min daily for 20 sessions. Following this combined therapy, the hematoma resolved completely. The patient remained recurrence-free throughout a 12-month follow-up period. However, due to multiple confounders (prior surgeries, variable adherence, concurrent treatments), causality cannot be established.

Conclusion

This hypothesis-generating observation suggests that for high-risk CSDH patients with profound coagulopathy, the early postoperative period may constitute a decisive “repair window” that warrants targeted investigation. A proactive, multimodal strategy targeting hemostatic correction, membrane stabilization, and microenvironment optimization during this window may disrupt the cycle of re-bleeding, but this requires validation in future controlled studies.

1. Introduction

Chronic subdural hematoma (CSDH) is a common neurosurgical condition, typically managed effectively with burr hole drainage. However, in patients with underlying systemic disorders affecting hemostasis and immune function, the clinical course can become protracted and recalcitrant to standard therapy ^[1, 2]. Co-infection with human immunodeficiency virus (HIV) and hepatitis C virus (HCV) leading to liver cirrhosis presents a particularly complex scenario. This combination creates a “perfect storm” of coagulopathy (from thrombocytopenia, impaired synthesis of clotting factors, and portal hypertension), systemic inflammation, and potential blood-brain barrier dysfunction, all of which may promote

hematoma expansion, impede natural resolution, and drive recurrence^[3–7]. The management of CSDH in this population requires a paradigm shift beyond simple surgical evacuation to address these foundational pathophysiological disturbances.

Here, we present a challenging case of recurrent CSDH in a patient with HIV/HCV-related cirrhosis. The failure of repeated surgical interventions led to the conceptualization of a targeted timing strategy during the early postoperative phase—termed the “repair window.” This report does not aim to validate any therapeutic protocol. Instead, it provides a hypothesis-generating observation of a multimodal timing strategy in an extreme case, with detailed limitations disclosed.

2. Results

2.1 Clinical baseline and repeated hospitalizations

A 43-year-old male was admitted with a chief complaint of “headache and dizziness for 5 days after a fall.” He had a complex medical history, including long-term substance abuse, HIV/HCV co-infection diagnosed over ten years earlier, and long-term antiretroviral therapy (lamivudine, efavirenz, tenofovir disoproxil fumarate) with poor adherence. Hypertension was diagnosed one year prior; he was not on any antihypertensive medication.

First Hospitalization

On admission, the patient was alert (GCS 15) with no focal neurological signs. Laboratory tests revealed severe thrombocytopenia (platelets $45 \times 10^9/L$) and coagulopathy. Head CT showed an acute/subacute right fronto-temporo-parieto-occipital subdural hematoma with mild midline shift (Fig. 1A). Conservative management was initiated (mannitol, glycerol/fructose, human immunoglobulin, continuation of antiretrovirals, and lacidipine for blood pressure). A chest CT suggested liver cirrhosis, but the patient declined further hepatic investigations.

On day 3, consciousness deteriorated to drowsiness (GCS 14). Repeat CT revealed hematoma enlargement (Fig. 1B). Due to neurological worsening, a right parietal burr hole evacuation with subdural drain was performed. Intraoperative drainage was uneventful. Postoperatively, human intravenous immunoglobulin (IVIG) and apheresis platelets were given. Urokinase (20,000 U/day) was irrigated through the drain for four days. Oral atorvastatin (20 mg nightly) was started to promote absorption. A follow-up CT on postoperative day 14 showed significant hematoma reduction (Fig. 1C). The patient insisted on discharge against medical advice, with instructions to continue atorvastatin and antiretrovirals.

Second Hospitalization

About one week later, he was readmitted with recurrent headache and dizziness. CT confirmed recurrence of the right-sided CSDH (Fig. 2A). Platelets were $55 \times 10^9/L$. The family declined surgery.

Management consisted of atorvastatin (20 mg nightly) and hyperbaric oxygen (HBOT: 2.0 ATA, 60 min daily for 20 sessions over 4 weeks). Liver ultrasound confirmed cirrhosis with splenomegaly. Mildly elevated transaminases were treated with hepatoprotectives. After 20 days, symptoms improved and CT showed marked hematoma reduction (Fig. 2B). He was discharged with platelets $60 \times 10^9/L$ and advised to continue atorvastatin, antiretrovirals, and antihypertensives.

Third Hospitalization

Nine months later, he presented with headache, dizziness, and right-sided weakness (grade 4/5). GCS was 15. Head CT revealed a new left fronto-temporo-parietal CSDH with significant mass effect (Fig. 3A). Laboratory findings: platelets $43 \times 10^9/L$, fibrinogen 1.8 g/L, leukopenia ($3.3 \times 10^9/L$). The patient admitted non-adherence to medications after the prior discharge. An urgent left parietal burr hole evacuation and drain placement was performed. Postoperatively, limb strength recovered immediately, and CT showed marked improvement (Fig. 3B). However, on postoperative day 7, consciousness declined to stupor. Repeat CT showed recurrence and enlargement of the left-sided hematoma (Fig. 3C), necessitating an emergency re-do burr hole drainage on the same side. This second surgery was designated day 0 for the “repair window” intervention.

2.2 Multimodal intervention within the postoperative repair window (from Day 0 onwards)

- - rhTPO: 300 U/kg subcutaneously once daily, started on day 0 and continued for 7 consecutive days. Target platelet count $> 80 \times 10^9/L$. No thrombotic events or acute liver function deterioration occurred.
- - HBOT: 2.0 atmospheres absolute (ATA); 20 min compression, 60 min 100% oxygen breathing, 20 min decompression; once daily. Initiated on day 1, continued for 20 sessions over 4 weeks (5 days/week). The patient tolerated all sessions without otologic or claustrophobic adverse events.
- - Atorvastatin: 20 mg orally nightly, continued throughout hospitalization and advised post-discharge.

Platelets rose from $43 \times 10^9/L$ (day 0) to a peak of $201 \times 10^9/L$ on day 5, with transient maintenance above $150 \times 10^9/L$, and coagulation parameters improved (see Table 1 for full timeline). Antiretroviral, hepatoprotective, and antihypertensive therapies were continued.

After approximately 40 days, the patient met criteria for clinical cure and was discharged. Pre-discharge CT showed near-complete resolution of the left-sided CSDH (Fig. 3D). At discharge, platelets had declined to $69 \times 10^9/L$.

Key baseline data (third hospitalization): Child-Pugh score B (7 points), Model for End-Stage Liver Disease-Sodium (MELD-Na) 12, HIV viral load 12,300 copies/mL, CD4 count 186 cells/ μL , HCV RNA positive (genotype 3a) (Table 2).

Table 1

Timeline of key events, platelet counts, and interventions during the third hospitalization (focused on the "repair window" period)

Time point	Platelets ($\times 10^9/L$)	Surgery / Event	rhTPO (300 U/kg SC daily)	HBOT (2.0 ATA, 60 min daily)	Atorvastatin (20 mg nightly)	CT finding
3rd admission, Day 2	43	Left burr hole	No	No	Continued (poor pre-adherence)	New L-sided CSDH, mass effect (Fig. 3A)
Post-op day 3	51	Clinical improvement	No	No	Continued	Drain in place, resolving (Fig. 3B)
Post-op day 7	48	Neurological deterioration	No	No	Continued	Recurrence with mass effect (Fig. 3C)
Day 0 (re-do surgery)	43	Emergency re-do burr hole (L)	Started	Not yet	Continued	Post-evacuation
Day 1	51	Clinical improvement	Continued		Continued	Stable
Day 3	89	-	Continued	Continued	Continued	Improving
Day 5	201	Remove evacuation	Continued	Continued	Continued	Markedly improved
Day 7	178	-	Stopped (7 days total)	Continued	Continued	No new collection
Day 14	132	-	Finished	Continued	Continued	Residual thin SDH
Day 28	110	-	Finished	Continued (20th session, stopped)	Continued	Near-complete resolution
Day 40	69	Discharged	Finished	Finished	Continued (advised)	Near-complete resolution (Fig. 3D)

SC: subcutaneous; HBOT: hyperbaric oxygen therapy; ATA: atmospheres absolute.

Table 2
Baseline clinical characteristics at third hospitalization (most complete data)

Parameter	Value	Interpretation
Liver function		
Child-Pugh score	7 (Grade B)	Moderate hepatic impairment
MELD-Na score	12	6-month mortality ~ 5–10%
Bilirubin (total)	28 µmol/L	Elevated
Albumin	30 g/L	Low
INR	1.4	Prolonged
HIV status		
HIV viral load	12,300 copies/mL	Detectable, poor adherence
CD4 count	186 cells/µL	Immunocompromised (CDC category 2)
HCV status		
HCV RNA	Positive (genotype 3a)	Active replication
Hematology		
Platelet nadir	43×10 ⁹ /L	Severe thrombocytopenia
Fibrinogen	1.8 g/L	Mild hypofibrinogenemia
Leukocytes	3.3×10 ⁹ /L	Mild leukopenia (splenic sequestration)

2.3 Long-term clinical follow-up

After discharge, the patient was enrolled in a structured follow-up program. His condition remained stable. A head CT one month post-discharge showed no recurrence. At two months, platelets were 45×10⁹/L, but the patient declined further intervention. Over the following year, outpatient and telephone follow-ups indicated no recurrent headaches or focal neurological deficits. Although the patient repeatedly declined further CT scans, clinical assessment suggested a low likelihood of recurrence.

2.4 Patient perspective

The patient provided informed consent. He expressed that the prolonged illness and multiple surgeries were highly distressing. After the third surgery and starting the new combined treatment (rhTPO injections, oxygen chamber, and statin), he reported: “I felt different. For the first time, my headaches didn’t just temporarily go away – they gradually faded and stayed away. The dizziness stopped, and my arm strength came back steadily. I finally believed the doctors were treating the real reason my blood wouldn’t clot, not just the bleed in my brain.” He remains engaged in regular follow-up for his systemic conditions.

3. Discussion

This case exemplifies the management complexity of CSDH in patients with concurrent coagulation and immune dysfunction due to HIV/HCV-related cirrhosis. Our experience leads us to propose a hypothesis that the early postoperative period may represent a critical “repair window” for membrane stabilization and vascular remodeling. However, this remains an unproven hypothesis derived from a single, heavily confounded observation.

Interpretation and alternative explanations

The patient’s favorable outcome after the third hospitalization (no recurrence for 12 months) coincided with the introduction of the combined regimen (rhTPO + atorvastatin + HBOT) following the re-do surgery. Nevertheless, multiple alternative explanations exist: (1) natural history modified by the two prior surgeries on the same side; (2) prolonged hospitalization (40 days) allowing more healing time; (3) improved adherence to antiretrovirals and hepatoprotectives during this admission; (4) possible contribution from earlier platelet transfusions or IVIG. Therefore, no causal link can be asserted.

Why this hypothesis-generating observation may still be valuable

Despite severe limitations, the case highlights a potential time-targeted strategy for a specific high-risk phenotype (Child-Pugh B cirrhosis, HIV/HCV co-infection, severe thrombocytopenia, and early recurrence after surgery). In such patients, preoperative hemostatic correction is often inadequate or transient. Using rhTPO to actively stimulate thrombopoiesis during the immediate postoperative days (rather than relying on transfusions) achieved a rapid and substantial platelet increase ($43 \rightarrow 201 \times 10^9/L$ within 5 days). This temporally coincided with resolution of the recurrence. While causation is unproven, this observation generates the hypothesis that achieving hemostatic competence during the first postoperative week may be critical for breaking the re-bleeding cycle.

Comparison with alternative treatments

Glucocorticoids were avoided due to meta-analyses showing no long-term functional benefit and increased infection/metabolic risks—particularly concerning in an immunocompromised patient with liver disease^[8, 9]. Middle meningeal artery embolization (MMAE) is promising, but recent evidence suggests its recurrence reduction benefit may be less pronounced in surgical cohorts, and underlying coagulopathy is a known risk factor for MMAE failure^[10, 11]. Our patient’s severe cirrhosis-associated coagulopathy warranted a primary focus on systemic hemostatic correction rather than adjunctive embolization.

Reflecting on management shortcomings and a preliminary framework

Based on this case, we identified several shortcomings that may inform future practice and research:

- 1) Delayed etiologic workup – early screening for HIV, HCV, and liver disease in atypical/recurrent CSDH is imperative.
- 2) Late MDT integration – proactive admission-triggered collaboration among neurosurgery, hematology, hepatology, and infectious diseases is essential.
- 3) Suboptimal hemostatic strategy – a shift from reactive transfusion to proactive stimulation of thrombopoiesis (e.g., with TPO-RAs) is supported by evidence for durable hemostasis in cirrhosis [12, 13].

4) Insufficient dynamic monitoring – serial viscoelastic testing could guide coagulation management.

- 5) Missed adjuvant therapy timing – earlier initiation of evidence-supported adjuncts (atorvastatin, HBOT) within the hypothesized repair window may enhance efficacy, though more CSDH-specific RCT data for HBOT are needed [14].

These observations lead to a preliminary, hypothesis-generating management framework (not a validated protocol): (i) prospective screening for systemic comorbidities upon admission; (ii) immediate MDT co-management; (iii) active perioperative hemostatic strategy using TPO-RAs to normalize platelets; (iv) consolidated repair-phase therapy with atorvastatin ± HBOT. This model is intended only to stimulate hypothesis-driven research.

Limitations

This study has severe limitations inherent to a single case report. First and foremost, no causal relationship can be established between the multimodal “repair window” therapy and the eventual favorable outcome. The clinical course was confounded by multiple surgeries (three right-sided, two left-sided), variable medication adherence, and concurrent treatments (IVIG, platelet transfusions, urokinase, antiretrovirals). Second, while each component therapy (rhTPO, atorvastatin, HBOT) is individually known, their coordinated application timed to a hypothesized “repair window” is novel but entirely hypothesis-generating. Third, key baseline data (e.g., detailed coagulation factor assays, pre-HBOT pulmonary function tests) were incomplete due to the retrospective nature. Fourth, lack of long-term radiological follow-up beyond one month (patient declined further CT scans) limits assessment of late recurrence. Fifth, the patient’s non-adherence further confounds interpretation. Finally, this is a single case from a single center, with no generalizability. Therefore, our proposed framework should be viewed as a clinical hypothesis and educational model, not a validated standard of care.

4. Conclusions

This single case observation proposes an early postoperative “repair window” concept for CSDH patients complicated by HIV/HCV cirrhosis and severe thrombocytopenia. Multimodal perioperative intervention

combining thrombopoietic stimulation, statin treatment and hyperbaric oxygen may help interrupt repeated rebleeding cycles during this critical time window. Nevertheless, definite therapeutic causality cannot be confirmed due to abundant confounding factors. The proposed time-based multimodal strategy remains purely hypothesis-generating and requires verification via large-scale controlled clinical trials before routine clinical application.

Declarations

Ethical Approval

This case report was conducted in accordance with the Declaration of Helsinki. The Ethics Committee of Tongren People's Hospital confirmed that formal ethical approval was waived for this retrospective single-patient case report.

Informed Consent Statement

Written informed consent for the publication of anonymized clinical data and all cranial CT images was fully obtained from the patient. The original signed paper consent form can be submitted to the editorial office upon official request.

Author Contributions

Li Cai: Data curation, Writing – original draft.

Heng Zhao: Conceptualization, Methodology, Supervision.

Hui Ran, Kunming Liu: Investigation, Writing – review & editing.

Yongming Zhang: Conceptualization, Supervision, Writing – review & editing. All authors have read and approved the final submitted manuscript.

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Data Availability Statement

All clinical data supporting the findings are fully presented within this article, including laboratory indicators, imaging descriptions and treatment records. Raw original laboratory test sheets and unprocessed CT imaging files cannot be publicly released to protect the patient's personal privacy.

Conflicts of Interest

The authors declare no conflicts of interest related to this work.

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Figures



Figure 1

Serial non-contrast cranial CT scans during the first admission, demonstrating the evolution of the right-sided subdural hematoma.

(A) Scan upon admission reveals an acute/subacute subdural hematoma in the right frontotemporoparietooccipital region, accompanied by mild compression of the right lateral ventricle and a slight midline shift to the left.

(B) Scan on day 3 of admission shows significant progression of the hematoma, with increased mass effect.

(C) Scan 14 days post-operatively following right-sided burr hole drainage, indicates post-surgical changes with reduced hematoma density and volume, though mild compression and midline shift persist.

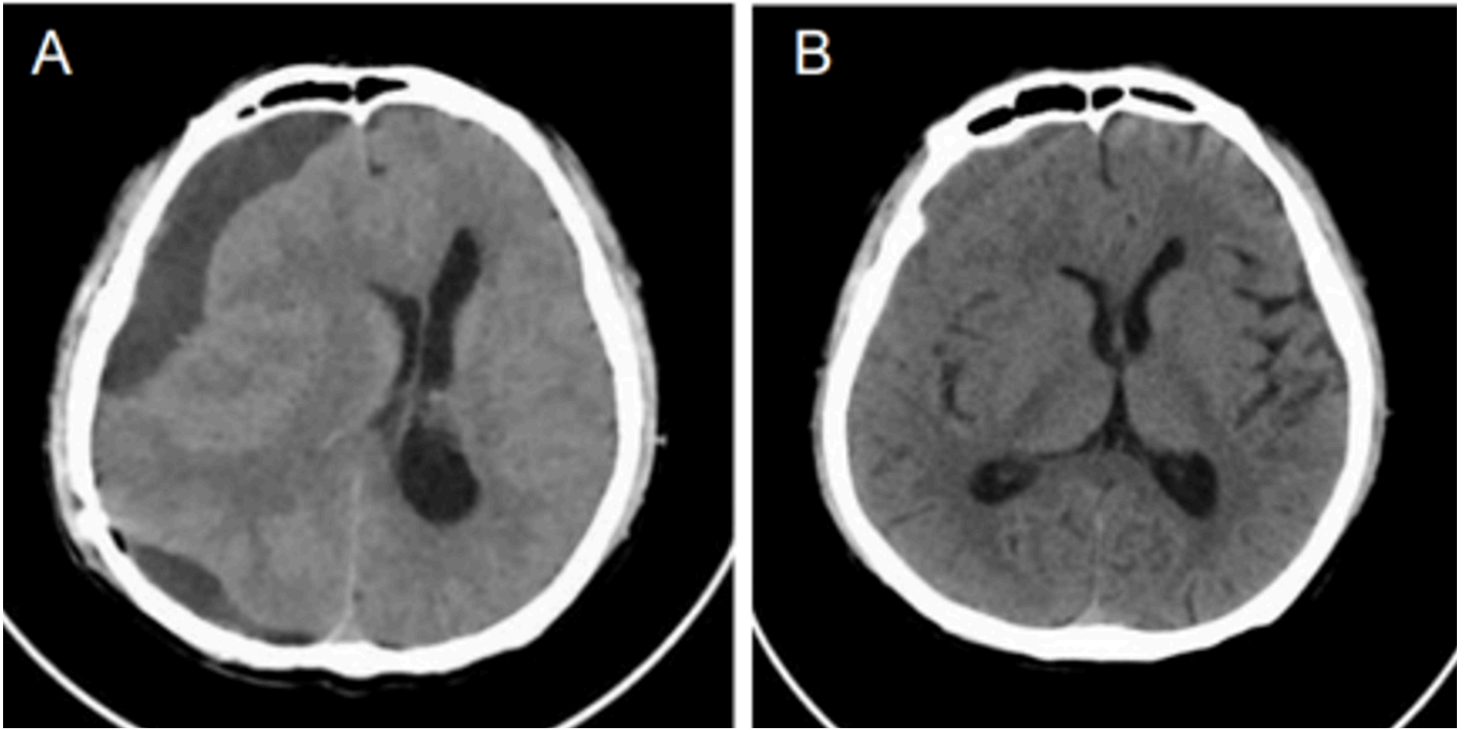


Figure 2

Neuroimaging evolution during the second hospitalization, demonstrating hematoma recurrence and subsequent resolution with conservative management.

(A) Non-contrast cranial CT upon readmission reveals recurrence of the right-sided chronic subdural hematoma, accompanied by significant mass effect, including marked midline shift and ventricular compression.

(B) Follow-up non-contrast cranial CT after 20 days of conservative therapy shows marked reduction in the size of the hematoma, with corresponding clinical improvement.

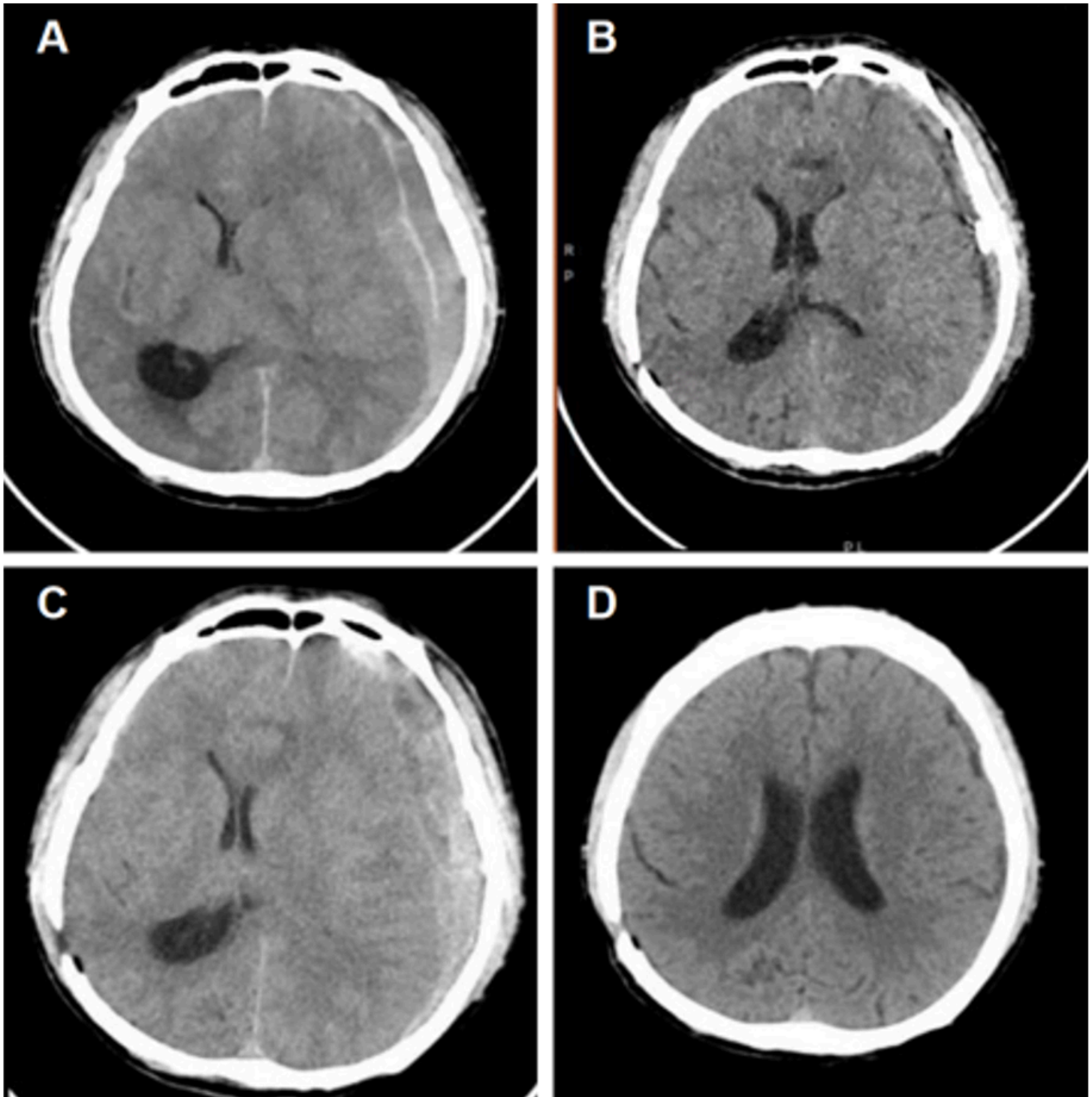


Figure 3

Radiological course during the third hospitalization, illustrating a new contralateral hematoma with postoperative recurrence and eventual resolution.

(A) Non-contrast cranial CT upon admission reveals a new left-sided frontotemporoparietal chronic subdural hematoma with significant mass effect.

(B) Postoperative CT scan following left-sided burr hole drainage demonstrates satisfactory evacuation of the hematoma and a notable reduction in the midline shift.

(C) On postoperative day 7, A repeat CT scan indicated recurrence and enlargement of the left-sided hematoma (Figure 3C)

(D) A pre-discharge CT scan showed near-complete resolution of the left fronto-temporo-parieto-occipital subdural hematoma (Figure 3D).