

Case Report

Spontaneous Epidural Hemorrhage in Sickle Cell Disease, Are They All the Same? A Case Report and Comprehensive Review of the Literature

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Trauma to the skull causing injury to the middle meningeal artery, middle meningeal vein, or dural venous sinuses is responsible for most cases of epidural hemorrhage (EDH). Spontaneous EDH is a rare entity in clinical practice. Common causes include sinusitis, coagulation abnormalities, dural metastasis, and Langerhans cell histiocytosis. Isolated nontraumatic EDH is an exceedingly rare complication of sickle cell disease (SCD). We report a case of spontaneous EDH in a patient with SCD and review the world literature regarding this rare entity. A 20-year-old African American female with sickle cell disease presented with vaso-occlusive crisis. About 24 hours after hospital admission, the patient had sudden deterioration of her mental status. An emergent CT scan of the head revealed a large right-sided frontoparietal epidural hematoma with midline shift, subfalcine, and uncal herniation. The patient underwent emergent hematoma evacuation but died 24 hours after surgery.

1. Introduction

Sickle cell disease (SCD) is caused by a single amino acid substitution on the beta globin chain resulting in the propensity of the hemoglobin molecule to polymerize in deoxygenated state. The abnormal polymerization is responsible for subsequent RBC injury, hemolysis, microvascular injury, and classical acute and chronic manifestations of the disease. Neurologic manifestations secondary to SCD are common and affect about 35% of patients [1]. Although reports vary [2], existing data suggest that 54% of these patients suffer from ischemic strokes and 34% from intracranial bleeding [1]. Intracerebral, subarachnoid, intraventricular, subdural, and epidural bleeds have been reported, with subarachnoid hemorrhage being the most common, especially in young adults [1–3]. Isolated nontraumatic spontaneous epidural hematoma (EDH) is an exceedingly rare complication of SCD. Other reported etiologies of spontaneous EDH are infectious [4–6],

coagulation abnormalities associated with end stage renal disease and hemodialysis [7–9], dural metastasis [10–13], and Langerhans cell histiocytosis [14–16]. We present a case of spontaneous EDH in a sickle cell patient suffering from vaso-occlusive crisis and review the world literature regarding the rare entity.

2. Case Presentation

A 20-year-old African American female with a history of sickle cell disease (HbSS) and multiple previous admissions for vaso-occlusive crisis (VOC) presented to the hospital with severe generalized pain throughout her body. The patient was in severe distress. Her blood pressure was 155/101 mmHg, pulse 117 beats per minute, temperature 37.6 C, respiratory rate 25 breaths per minute, and oxygen saturation 98% on room air. Physical examination revealed poor bilateral air entry on lung auscultation due to splinting and an ejection systolic murmur over the aortic area. Mild-to-

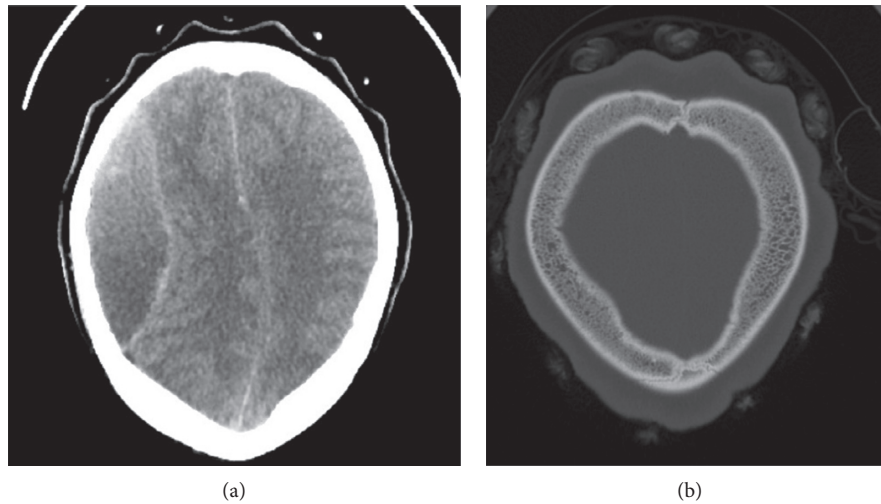


FIGURE 1: (a) Mixed density right frontoparietal epidural hematoma with midline shift. (b) Bony window showing massive expansion of the diploic bone from extramedullary hematopoiesis.

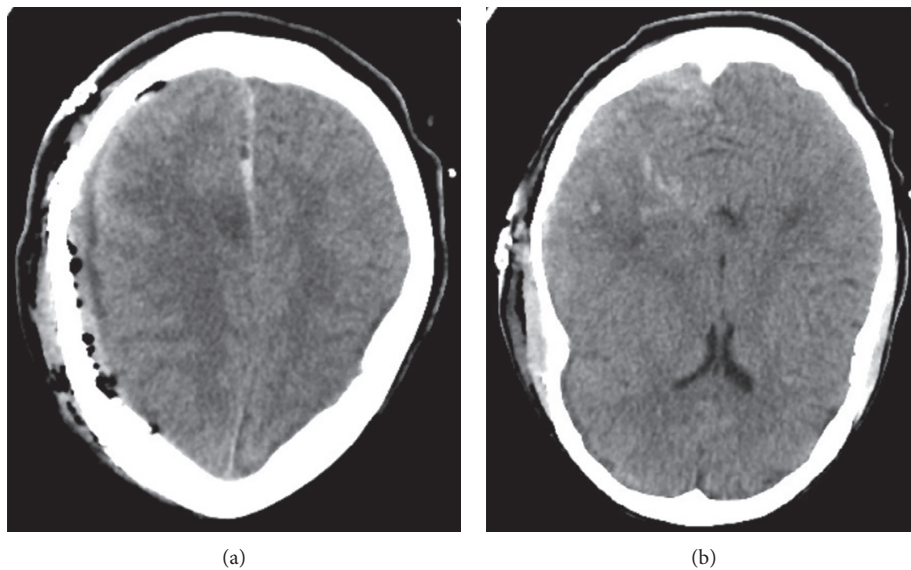


FIGURE 2: (a) Postoperative changes following hematoma evacuation with a small collection in the subdural space and significant improvement of midline shift (b) Punctate hemorrhage in the right temporal lobe postop.

moderate tenderness was present over the extremities on palpation. Neurological examination was normal. Blood work showed leukocytosis, 12,800 with 53% neutrophil, 32% lymphocyte, and 1% band, hemoglobin 7.5 gm/dL, hematocrit 22.3%, platelet 181,000/dL, reticulocyte count 13%, lactate dehydrogenase 1144 IU/L, normal blood urea nitrogen, creatinine, and serum electrolyte studies. Liver function tests were normal except a total bilirubin level of 16.2 mg/dL. Chest X-ray was normal. The patient was started on IV hydration; analgesia was achieved by IV narcotics.

About 24 hours after hospital admission, the patient suddenly became unresponsive. Naloxone failed to improve her mental status. An emergent CT scan of the head revealed a large right-sided frontoparietal epidural hematoma with midline shift, subfalcine, and uncal herniation. No subgaleal

or subperiosteal collection was noted. There was no noticeable bone infarction overlying the hematoma (Figure 1).

Laboratory data at this time demonstrated a platelet of 45,000/dL, prothrombin time 19.7 Seconds, INR 1.7, activated partial thromboplastin time 43 seconds, and a fibrinogen level of 96 mg/dL, consistent with a diagnosis of disseminated intravascular coagulation (DIC). The patient was emergently taken to the operating room for hematoma evacuation (Figure 2). Intraoperatively, no obvious bony abnormality was noted. Exchange transfusion was performed. The patient, however deteriorated, became hypotensive requiring multiple vasopressors and eventually died about 24 hour after surgical intervention from DIC.

Total 31 cases. R, right; L, left; N/S, not specified; B/L, bilateral; VOC, vaso-occlusive disease; FP, frontoparietal;

TABLE 1

Case report	Year	Age (years)	Sex	Haplotype	Presentation	Identification of EDH after hospitalization	Location of EDH	Subgaleal hemorrhage	Subperiosteal collection	Skull infarction	DIC present	Outcome
Current case	2019	19	Female	HbSS	VOC	24 hours	R FP	No	No	No (CT/IO)	Yes	Died
Komarila et al. [17]	2018	18	Female	Unspecified	VOC	24 hours	R parietal	No	No	Yes (MRI)	N/S	Survived
Banerjee et al. [18]	2018	17	Male	Unspecified	Headache	POA	Bifrontal	Yes	Yes	Yes (MRI)	N/S	Survived
Moyen et al. [19]	2018	Teenage	Male	HbSS	VOC	6 hours	L frontal	No	No	No (MRI)	Yes	Died
Mishra et al. [20]	2018	13	Male	HbSS	Seizure	Imaging not performed till day 8	R FP	No	N/S	No (MRI)	No	Died
Gajjar and Gupta [21]	2017	18	Male	HbSS	VOC	120 hours	R parietal	Yes	No	Yes (IO)	No	Survived
Hettige et al. [22]	2015	20	Male	Unspecified	Headache	POA	L PT	No	No	No (CT)	No	Survived
Yogarajah et al. [23]	2015	7	Female	HbSS	Coma	POA	B/L parietal	No	No	No (CT/IO)	Yes	Died
N'dri Oka et al. [24]	2015	19	Male	HbSC	VOC	24 hours	R PT	No	No	No (CT/IO)	No	Survived
Ilhan et al. [25]	2015	19	Male	HbSC	Headache	POA	Occipital	Yes	No	No (CT)	No	Survived
Serarslan et al. [26]	2014	15	Male	HbSS	Headache	POA	Right frontal	Yes	No	Yes (MRI)	No	Survived
Page et al. [27]	2014	19	Female	HbSS	Headache	POA	L FP	No	No	No (CT/IO)	No	Survived
Babatola et al. [28]	2014	20	Male	HbSS	VOC	48 hours	L frontal	Yes	No	Yes (MRI)	No	Survived
Bolke and Scherer [29]	2014	7	Female	HbSS	Coma	POA	R temporal	No	No	Yes (MRI)	No	Survived
Patra et al. [30]	2012	18	Male	HbSS	Headache	POA	R Frontal	No	No	No (CT/IO)	No	Survived
Arends et al. [31]	2012	19	Male	Unspecified	VOC	72 hours	L frontal	No	No	No (CT/IO)	No	Died
Sangle et al. [32]	2012	13	Male	Unspecified	Headache	POA	B/L parietal	No	No	No (CT)	No	Survived
Azhar [33]	2011	19	Male	HbSC	Headache	POA	R parietal	No	No	Yes (MRI)	No	Survived
Dahdaleh et al. [34]	2011	15	Male	Unspecified	Headache	12 hours	Bifrontal	No	No	No (MRI)	No	Died
Kotb et al. [35]	2010	12	Male	HbSD	VOC	24 hours	L frontal	No	No	N/S (CT)	N/S	Survived
	2009	18	Male	Unspecified	VOC	12 hours	B/L FP	Yes	No	No (CT/IO)	No	Survived
	2006	10	Male	Unspecified	Headache	N/S	Bifrontal	Yes	No	Yes (MRI)	N/S	Survived

TABLE 1: Continued.

Case report	Year	Age (years)	Sex	Haplotype	Presentation	Identification of EDH after hospitalization	Location of EDH	Subgaleal hemorrhage	Subperiosteal collection	Skull infarction	DIC present	Outcome
Kalala Okito et al. [36]	2004	2	Male	HbSS	Coma	POA	R FT	No	No	N/S (IO)	N/S	Died
Ganesh et al. [37]	2001	11	Male	HbSS	Proptosis	POA	L parietal	No	No	Yes (X-ray)	No	Survived
Naran and Fontana [38]	2001	16	Male	Unspecified	Headache	? POA	Bifrontal	Yes	No	Yes (BS)	N/S	Survived
Cabon et al. [39]	1997	14	Female	HbSS	Unknown	Unknown	R Frontal	No	No	Yes (MRI/BS)	No	Survived
Resar et al. [40]	1996	14	Male	HbSS	VOC	48 hours	Bifrontal	Unknown	Unknown	Yes	Unknown	Unknown
Tony et al. [41]	1995	35	Male	Sickle-thalassemia	Proptosis	POA	Left parietal and B/L frontal	Yes	Yes	Yes (MRI/BS)	No	Survived
Karacostas et al. [42]	1991	19	Male	HbSS	VOC	48 hours	Left frontal	No	No	Yes (BS)	No	Survived
Mallouh et al. [43]	1987	13	Male	HbSS	Eye swelling	POA	Left frontal	No	No	No (CT)	No	Survived
							Bifrontal	No	No	Yes	N/S	Survived

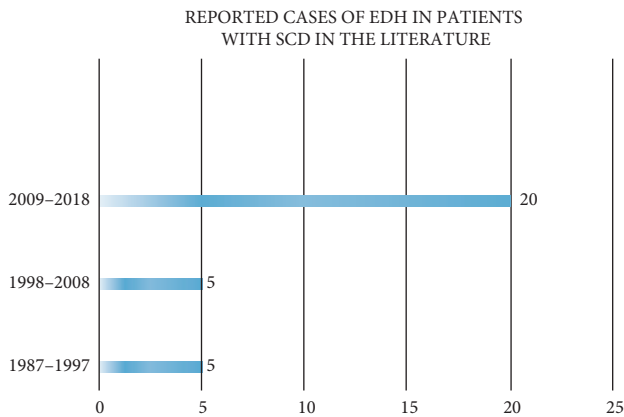


FIGURE 3: Reported cases of EDH in patients with SCD in the literature.

PT, parietotemporal; DIC, disseminated intravascular coagulation; POA, present on admission; BS, bone scan; IO, intraoperatively.

3. Discussion

We have described a patient with SCD suffering from spontaneous EDH in the setting of VOC. Including the present case, we have identified 31 cases described in the literature, which are shown individually in Table 1. Since first reported about 3 decades ago, the reporting incidence has increased precipitously in the last decade possibly due to increased awareness, radiologic advancement, and more opportunities for publication rather than actual increase in incidence (Figure 3).

Most reported cases comprised males 25/31 (80%). The age ranged from 2–35 years (median 16.5) and 7–19 years (median 16), respectively, for males and females. 26/31 (84%) had SCD (17 patients with reported homozygosity and 9 known cases without specified haplotype). Other cases included 3 patients with HbSC disease, 1 with HbSD, and sickle thalassemia each.

Headache was present on admission in 37% (11/30), whereas symptoms consistent with VOC, primarily pain crisis, were the initial complaint in 40% (12/30). Other presenting symptoms were eye swelling, proptosis, seizure, and coma. In patients who presented with a headache, EDH was present on admission in 82% of the time. Patients who presented with VOC, progressed to have EDH within 6–120 hours, with a median of 24 hours after hospital admission.

Unilateral EDH was more common 20/31 (65%). Subgaleal hemorrhage was concurrently present in 9/30 (30%) of patients and associated with bone infarction in (7/9) 78% of cases. 48% of patients (15/31) with EDH showed evidence of overlying bony infarction. The overall mortality among the reported cases was 23% (7/30). Interestingly, patients with evidence of overlying bone infarction had a survival of 100% and patients with DIC had a mortality of 100%.

The most common cause of EDH is trauma causing injury to the middle meningeal artery, middle meningeal vein, the diploic vein, or the dural venous sinuses [44].

Spontaneous nontraumatic EDH is a rare manifestation of SCD and variant sickle cell syndromes. The pathophysiology of this rare occurrence is not completely understood.

Most reported cases in the literature are males. Whether any gender specific etiology is responsible for this discrepancy is not known. Low steady-state hemoglobin and high leukocyte count are known risk factors for hemorrhagic stroke in patients with SCD [1]. Whether this is also true for EDH is not clear. In the landmark study by Ohene-Frempong et al., hemorrhagic strokes were more prevalent in the adult SCD patients in the age range of 20–30 years [1]. However, EDH appears to be more prevalent in the adolescent age group.

Three pathophysiologic explanations have been proposed over the years for the causation of this rare entity: (1) Vaso-occlusion of the haematopoietically active calvarial diploic bone resulting in bone infarction and subsequent leaking of blood and proteinaceous material in the subperiosteal, epidural, or subgaleal space [38–41, 43, 45]. (2) Acute rapid expansion of hematopoiesis with resultant microfracture of already thinned inner cortex and extravasation of blood and hematopoietic tissue [24, 28, 36]. (3) Sludging of sickle cells in the diploic veins hampering venous drainage and oozing of blood due to vascular injury and elevated back pressure. A combination of different mechanisms could also be responsible. Presence of coagulopathy or platelet dysfunction worsens hematoma expansion and portends a dire clinical outcome.

Patients admitted with VOC developed EDH, did so with a median of 24 hours, suggesting early deterioration is expected in this patient population. Patients who suffered from concurrent bony infarction or subgaleal hemorrhage had a survival rate of 100%. This may indicate that vaso-occlusive etiology of the EDH carries better prognosis. This would also explain presence of EDH with contralateral subgaleal hemorrhage as skull bone infarction could be diffused [40]. The identification of bone infarction can be challenging. MRI appears to be the most sensitive tool. CT scan has low yield especially in the acute phase of the disease [18]. Infarcted bone can appear normal intraoperatively although thinning of the inner cortex is frequently seen. In 3 out of 7 patients who succumbed to their disease, an MRI ruled out any bony infarction. In the other 4 cases, no bone damage was noted by CT scan or on intraoperative inspection. We propose that patients without any obvious bony disruption and infarction, most likely suffer from microfracture of the inner table and extrusion of blood and hematopoietic cells in the epidural space due to rapid expansion of hematopoiesis. Presence of coagulopathy as in DIC makes the bleeding significantly worse. Patients who suffered from spontaneous EDH and suffered from DIC had a 100% mortality.

4. Conclusion

The understanding regarding this clinical entity is rapidly evolving in the era of advanced technology and improved awareness among healthcare providers. We believe this review will shed more light on the clinical as well as the pathophysiological aspect of the disease process and help in

rapid identification of patients at risk of deterioration and formulate management plan accordingly.

Conflicts of Interest

The authors declare that they have no conflicts of interest.

References

- [1] K. Ohene-Frempong, S. J. Weiner, L. A. Sleeper et al., "Cerebrovascular accidents in sickle cell disease: rates and risk factors," *Blood*, vol. 91, no. 1, pp. 288–294, 1998.
- [2] A. Gueguen, M. Mahevas, R. Nzouakou et al., "Sickle-cell disease stroke throughout life: a retrospective study in an adult referral center," *American Journal of Hematology*, vol. 89, no. 3, pp. 267–272, 2014.
- [3] J. J. Strouse, M. L. Hulbert, M. R. DeBaun, L. C. Jordan, and J. F. Casella, "Primary hemorrhagic stroke in children with sickle cell disease is associated with recent transfusion and use of corticosteroids," *Pediatrics*, vol. 118, no. 5, pp. 1916–1924, 2006.
- [4] M. C. Papadopoulos, A. Dyer, and C. Hardwidge, "Spontaneous extradural haematoma with sinusitis," *Journal of the Royal Society of Medicine*, vol. 94, no. 11, pp. 588–589, 2001.
- [5] G. Moonis, A. Granados, and S. L. Simon, "Epidural hematoma as a complication of sphenoid sinusitis and epidural abscess," *Clinical Imaging*, vol. 26, no. 6, pp. 382–385, 2002.
- [6] S. Chaiyasate, S. Halewyck, K. Van Rompaey, and P. Clement, "Spontaneous extradural hematoma as a presentation of sinusitis: case report and literature review," *International Journal of Pediatric Otorhinolaryngology*, vol. 71, no. 5, pp. 827–830, 2007.
- [7] K. Shahlaie, A. Fox, L. Butani, and J. E. Boggan, "Spontaneous epidural hemorrhage in chronic renal failure. a case report and review," *Pediatric Nephrology*, vol. 19, no. 10, pp. 1168–1172, 2004.
- [8] S. Shimokawa, T. Hayashi, S. Anegawa et al., "Spontaneous epidural hematoma in a patient undergoing hemodialysis: a case report," *No to Shinkei = Brain and Nerve*, vol. 55, no. 2, pp. 163–166, 2003.
- [9] F. Zheng and Y. Chao, "Spontaneous intracranial extradural hematoma: case report and literature review," *Neurology India*, vol. 57, no. 3, pp. 324–326, 2009.
- [10] S. Anegawa, S. Hirohata, T. Tokutomi, and S. Kuramoto, "Spontaneous epidural hematoma secondary to dural metastasis from an ovarian carcinoma," *Neurologia Medico-Chirurgica*, vol. 29, no. 9, pp. 854–856, 1989.
- [11] J. I. McIver, B. W. Scheithauer, C. H. Rydberg, and J. L. D. Atkinson, "Metastatic hepatocellular carcinoma presenting as epidural hematoma: case report," *Neurosurgery*, vol. 49, no. 2, pp. 447–449, 2001.
- [12] K. Hayashi, T. Matsuo, M. Kurihara, M. Daikoku, G. Kitange, and S. Shibata, "Skull metastasis of hepatocellular carcinoma associated with acute epidural hematoma: a case report," *Surgical Neurology*, vol. 53, no. 4, pp. 379–382, 2000.
- [13] M. S. Shamim, M. E. Bari, and S. A. Enam, "Dural metastases presenting as an extradural hematoma: a rare presentation," *JPMA Journal of Pakistan Medical Association*, vol. 55, no. 11, pp. 509–510, 2005.
- [14] D.-Y. Cho, W.-R. Liau, and I.-P. Chiang, "Eosinophilic granuloma with acute epidural hematoma," *Pediatric Neurosurgery*, vol. 35, no. 5, pp. 266–269, 2001.
- [15] H. C. Chen, W. C. Shen, D. Y. Chou, and I. P. Chiang, "Langerhans cell histiocytosis of the skull complicated with an epidural hematoma," *AJNR American Journal of Neuroradiology*, vol. 23, no. 3, pp. 493–495, 2002.
- [16] J. F. Martinez-Lage, M. Bermudez, E. Martinez-Barba, J. L. Fuster, and M. Poza, "Epidural hematoma from a cranial eosinophilic granuloma," *Child's Nervous System: ChNS: Official Journal of the International Society for Pediatric Neurosurgery*, vol. 18, no. 1-2, pp. 74–76, 2002.
- [17] R. Komarla, B. P. Soares, J. J. Chern, and S. S. Milla, "Spontaneous epidural hematoma secondary to bone infarction in sickle cell anemia: case report," *Journal of Neurosurgery: Pediatrics PED*, vol. 22, no. 1, pp. 18–21, 2018.
- [18] C. Banerjee, J. Yowtak, D. Fridlyand, and C. Alleyne Jr., "Acute spontaneous intracranial epidural haematoma and disseminated intravascular coagulation in a paediatric sickle cell patient," *BMJ Case Reports*, vol. 2018, 2018.
- [19] E. Moyen, J. Kambourou, H. B. Ekouelé-Mbaki et al., "Spontaneous epidural hematoma in a child with sickle cell disease," *Internal Medicine Review*, vol. 4, no. 1, pp. 1–7, 2018.
- [20] S. S. Mishra, S. B. Senapati, A. K. Gouda, S. K. Behera, and A. Patnaik, "Spontaneous extradural and subgaleal hematoma: a rare neurosurgical crisis of sickle cell disease," *Asian Journal of Neurosurgery*, vol. 12, no. 1, pp. 47–50, 2017.
- [21] R. Gajjar and P. Gupta, "Spontaneous extradural hematoma: a rare neurological crisis in sickle cell disease," *IOSR Journal of Dental and Medical Sciences*, vol. 14, no. 12, pp. 94–96, 2015.
- [22] S. Hettige, A. Sofela, S. Bassi, and C. Chandler, "A review of spontaneous intracranial extradural hematoma in sickle-cell disease," *Acta Neurochirurgica*, vol. 157, no. 11, pp. 2025–2029, 2015.
- [23] M. Yogarajah, C. C. Agu, B. Sivasambu, and M. A. Mittler, "HbSC disease and spontaneous epidural hematoma with kernohan's notch phenomena," *Case Reports in Hematology*, vol. 2015, Article ID 470873, 3 pages, 2015.
- [24] D. N'Dri Oka, A. Tokpa, A. Bah, and L. Derou, "Spontaneous intracranial extradural hematoma in sickle cell disease," *Journal of Neurological Surgery Reports*, vol. 76, no. 1, pp. e97–e99, 2015.
- [25] N. Ilhan, C. Acipayam, F. Aydogan et al., "Orbital compression syndrome complicated by epidural hematoma and wide cephalohematoma in a patient with sickle cell disease," *Journal of American Association for Pediatric Ophthalmology and Strabismus*, vol. 18, no. 2, pp. 189–191, 2014.
- [26] Y. Serarslan, M. Aras, M. Altaş, H. Kaya, and B. Urfah, "Non-traumatic spontaneous acute epidural hematoma in a patient with sickle cell disease," *Neurocirugia*, vol. 25, no. 3, pp. 128–131, 2014.
- [27] C. Page, K. Gardner, S. Height, D. C. Rees, T. Hampton, and S. L. Thein, "Nontraumatic extradural hematoma in sickle cell anemia: a rare neurological complication not to be missed," *American Journal of Hematology*, vol. 89, no. 2, pp. 225–227, 2014.
- [28] B. O. Babatola, Y. A. Salman, A. M. Abiola, K. O. Okezie, and A. S. Oladele, "Spontaneous epidural haematoma in sickle cell anaemia: case report and literature review," *Journal of Surgical Technique and Case Report*, vol. 4, no. 2, pp. 135–137, 2012.
- [29] E. Bolke and A. Scherer, "Sickle cell disease," *Canadian Medical Association Journal*, vol. 184, no. 3, p. E201, 2012.
- [30] S. Patra, S. Mishra, and S. Das, "A rare case of spontaneous bilateral extradural hematoma in a sickle cell disease child," *Journal of Pediatric Neurosciences*, vol. 7, no. 1, pp. 77–78, 2012.
- [31] S. Arends, J. A. Coebergh, J. L. Kerkhoffs, A. van Gils, and H. Koppen, "Severe unilateral headache caused by skull bone

- infarction with epidural haematoma in a patient with sickle cell disease,” *Cephalalgia*, vol. 31, no. 12, pp. 1325–1328, 2011.
- [32] S. Sangle, R. Lohiya, S. Karne, and K. Chugh, “Spontaneous epidural hematoma: a rare complication of sickle cell anemia,” *Neurology India*, vol. 59, no. 2, pp. 301–302, 2011.
- [33] M. J. Azhar, “Extradural hemorrhage: a rare complication and manifestation of stroke in sickle cell disease,” *Oman Medical Journal*, vol. 25, no. 4, p. e017, 2010.
- [34] N. S. Dahdaleh, T. E. Lindley, P. A. Kirby, H. Oya, and M. A. Howard 3rd, “A “neurosurgical crisis” of sickle cell disease,” *Journal of Neurosurgery: Pediatrics*, vol. 4, no. 6, pp. 532–535, 2009.
- [35] M. M. Kotb, W. H. Tantawi, A. A. Elsayed, G. A. Damanhoury, and H. M. Malibary, “Brain MRI and CT findings in sickle cell disease patients from Western Saudi Arabia,” *Neurosciences (Riyadh, Saudi Arabia)*, vol. 11, no. 1, pp. 28–36, 2006.
- [36] J. P. Kalala Okito, O. Van Damme, and L. Calliauw, “Are spontaneous epidural haematoma in sickle cell disease a rare complication? A report of two new cases,” *Acta Neurochirurgica*, vol. 146, no. 4, pp. 407–410, 2004.
- [37] A. Ganesh, R. R. William, S. Mitra et al., “Orbital involvement in sickle cell disease: a report of five cases and review literature,” *Eye*, vol. 15, no. 6, pp. 774–780, 2001.
- [38] A. D. Naran and L. Fontana, “Sickle cell disease with orbital infarction and epidural hematoma,” *Pediatric Radiology*, vol. 31, no. 4, pp. 257–259, 2001.
- [39] I. Cabon, J. P. Hladky, A. Lambilliotte, A. Cotten, and P. Dhellemmes, “Uncommon etiology of extradural hematoma,” *Neurochirurgie*, vol. 43, no. 3, pp. 173–176, 1997.
- [40] L. M. S. Resar, M. M. Oliva, and J. F. Casella, “Skull infarction and epidural hematomas in a patient with sickle cell anemia,” *Journal of Pediatric Hematology/Oncology*, vol. 18, no. 4, pp. 413–415, 1996.
- [41] J. Tony, G. Subramanya, K. G. Kallur, A. V. Chalapathy, S. Sheshadri, and B. Lakhkar, “Proptosis, skull infarction and epidural haematoma in sickle thalassemia,” *Postgraduate Medical Journal*, vol. 71, no. 837, p. 445, 1995.
- [42] D. Karacostas, N. Artemis, M. Papadopoulou, and J. Christakis, “Case report: epidural and bilateral retroorbital hematomas complicating sickle cell anemia,” *American Journal of the Medical Sciences*, vol. 302, no. 2, pp. 107–109, 1991.
- [43] A. A. Mallouh, M. Young, J. Hamdan, and M. M. Salamah, “Proptosis, skull infarction, and retro-orbital and epidural hematomas in a child with sickle cell disease,” *Clinical Pediatrics*, vol. 26, no. 10, pp. 536–538, 1987.
- [44] M. R. Bullock, R. Chesnut, J. Ghajar et al., “Surgical management of acute epidural hematomas,” *Neurosurgery*, vol. 58, no. 3, pp. S7–15, 2006.
- [45] M. Watanabe, N. Saito, R. N. Nadgir et al., “Craniofacial bone infarcts in sickle cell disease,” *Journal of Computer Assisted Tomography*, vol. 37, no. 1, pp. 91–97, 2013.