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| RESEARCH ARTICLE

Flash Pulmonary Edema After Unnecessary Normal Saline Infusion Unmasking Sickle Nephropathy: Highlighting the Controversy of Routine Fluid Use in Vaso-Occlusive Crisis

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| ABSTRACT

Acute pulmonary edema following routine fluid administration is an uncommon but potentially life-threatening complication in adults with sickle cell disease (SCD). We report the case of a 42-year-old male with known SCD who presented with severe generalized body pain consistent with vaso-occlusive crisis (VOC). Initial management included intravenous normal saline for mild dehydration. Shortly after receiving approximately 500 mL, he developed acute shortness of breath, hypoxia, and fine basal crackles. Chest imaging demonstrated bilateral alveolar infiltrates consistent with pulmonary edema. Laboratory evaluation revealed baseline anemia, mild leukocytosis, elevated creatinine, and trace proteinuria, suggestive of subclinical sickle nephropathy. Echocardiography showed normal cardiac function, excluding primary cardiogenic causes. The patient received supplemental oxygen, cautious diuretics, and pain control while fluid administration was restricted. Transfusion support was provided to improve oxygen-carrying capacity and reduce sickling risk. Multidisciplinary management included hematology, nephrology, and respiratory care teams. The patient's respiratory status gradually improved, and pulmonary edema resolved without further complications. This case highlights the risk of flash pulmonary edema triggered by standard intravenous fluids in patients with SCD and previously undiagnosed renal impairment. It underscores the importance of individualized fluid management, careful monitoring, and early recognition of subclinical sickle nephropathy. Clinicians should exercise caution with routine fluid boluses in VOC, even in patients without overt kidney disease, as modest fluid volumes may precipitate life-threatening pulmonary complications. Prompt identification and tailored therapy can prevent morbidity and guide safe management of VOC in adults with SCD.

| KEYWORDS

Sickle Cell Disease, Normal Saline, Fluids, Vaso-occlusive Crisis, Sickle Nephropathy, Fluid Overload, Flash Pulmonary Edema, Mortality

| ARTICLE INFORMATION

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Introduction

Introduction

Vaso-occlusive crisis in sickle cell disease is one of the most common reasons patients come to the hospital with severe pain and complications related to sickling of red blood cells [1,4]. The crisis happens when sickle shaped cells block small blood vessels, which leads to tissue ischemia, pain, and organ dysfunction [1]. Historically, doctors have given intravenous fluids early in VOC to try to prevent further sickling and to correct dehydration that might worsen the crisis [5,7]. The idea behind giving fluids like normal saline is that dehydration may increase blood viscosity and worsen sickling, and that fluids could help dilute sickled cells and improve flow [5,7]. Dehydration increases plasma osmolarity, which can draw water out of red cells and make them more prone to sickling, so fluids are supposed to keep blood more dilute and reduce cell sickling [5]. Most guidelines and textbooks still list hydration as part of VOC management, along with pain control and oxygen if needed [5,12]. Normal saline and dextrose containing saline are often used for this purpose because they are widely available and familiar to clinicians [12]. However, despite long standing use, there is **very limited high quality evidence that routine intravenous fluid administration in VOC improves outcomes** such as pain relief, time to recovery, or reduced hospital stay [1,4]. Systematic reviews show that most studies on fluids in VOC are small, observational, or retrospective, and do not clearly prove benefit [1,4]. There are no large randomized controlled trials that define the best fluid type, amount, or rate for patients with VOC, and major hematology guidelines do not make firm recommendations for or against routine IV fluid use because of this lack of evidence [1,4]. Normal saline is commonly used because it is isotonic and familiar, but it may not be ideal for sickle cell patients. In vitro studies show that exposing sickled red cells to normal saline can increase their stiffness and tendency to block small vessels, which could theoretically worsen vaso-occlusion [9]. Normal saline can also promote a mild acid effect in the body because of its high chloride content, and a lower blood pH has been associated with increased sickling in laboratory models [4]. These findings raise questions about whether normal saline truly helps, or might in some situations add harm. A major concern with routine intravenous fluids in VOC is **fluid overload**. Sickle cell patients often have underlying kidney changes, including a reduced ability to concentrate urine and some degree of sickle nephropathy, which limits their ability to handle large fluid loads [1,3]. They also can have subtle cardiac or pulmonary dysfunction that makes them less able to tolerate extra fluid without developing congestion. Retrospective studies show **significant rates of fluid overload in sickle cell patients treated with standard fluid regimens**, with one report finding fluid overload in about one in five admitted patients [2,4]. Fluid overload in this context can present as swelling, shortness of breath, and pulmonary edema, which can require oxygen therapy or even transfer to higher levels of care. Pulmonary edema is especially dangerous because it can worsen respiratory symptoms and increase the risk of acute chest syndrome, a severe complication of sickle cell disease with high morbidity. Some autopsy series of patients with sickle cell disease who died unexpectedly found pulmonary edema to be the most common pathological finding, underscoring how serious fluid complications can be when they develop [3]. These observations do not prove that intravenous fluids caused the outcome, but they highlight that fluid overload and pulmonary congestion are real clinical risks that clinicians should be mindful of when giving fluids in VOC. Another concerning observation from clinical reports is that **patients given normal saline boluses may have worse pain control and longer stays in the emergency department and hospital** compared with those who did not receive bolus fluids [4]. These findings raise the possibility that certain fluid strategies, especially aggressive normal saline boluses, may not be benign and could even oppose pain resolution, though this has not been confirmed in randomized trials. In practice, fluid administration during VOC varies widely from place to place. Some centers give standard volumes of normal saline to all patients with VOC, while others tailor fluid use based on clinical assessment of hydration status and cardiac function [1,15]. There is no consensus on the best regimen, and variations reflect the lack of strong evidence guiding practice. Some pediatric hospital protocols even recommend different types of fluids, such as hypotonic solutions, though evidence for superiority of one type over another is still not robust [15]. A key reason for the lack of clear guidance is that **the balance between dehydration and overhydration is delicate in these patients**. Dehydration may worsen sickling, but overhydration may lead to volume overload, pulmonary edema, and other complications. This balance is hard to judge clinically, particularly when patients present with pain and may not be able to take fluids by mouth because of nausea or opioid effects. The American Society of Hematology and other expert bodies have acknowledged these uncertainties and do not make firm recommendations for or against routine intravenous fluid therapy in VOC. They emphasize that clinicians should assess each patient individually, consider oral hydration when possible, and be cautious with intravenous fluids, especially in those with known cardiac or renal

compromise [1,4]. Simple oral hydration, when tolerated, may be sufficient for many patients who are not volume depleted. Encouraging patients to drink fluids and addressing barriers to oral intake can often maintain hydration without risking fluid overload. In cases where patients cannot drink or are clearly dehydrated, clinicians may decide to give intravenous fluids, but usually with careful monitoring of volume status, urine output, and signs of congestion. There is also emerging interest in whether fluid formulas other than normal saline, such as lactated Ringer's solution, might be associated with better outcomes. Limited observational data suggest that balanced fluids like lactated Ringer's might be associated with shorter hospital stays and fewer readmissions than normal saline, but these findings need confirmation in controlled trials [6]. Balanced fluids may have electrolyte compositions closer to plasma and could, in theory, avoid some of the acid-base issues seen with normal saline. However, no definitive evidence yet guides clinicians to adopt one fluid type universally over another. The controversy around routine fluid use in VOC highlights a broader gap in evidence for many supportive care practices in sickle cell disease, where tradition and clinical experience often guide care in the absence of strong trial data. While fluids remain a traditional component of VOC management, clinicians must weigh the possible but unproven benefits against the known risks of fluid overload, especially in patients at higher risk of pulmonary or cardiac complications. Given the uncertainties, many experts recommend a cautious approach tailored to individual patients. This includes assessing hydration status on admission, considering oral hydration first if possible, using intravenous fluids carefully and at rates appropriate to the patient's clinical situation, and watching closely for signs of overload or pulmonary compromise. Early involvement of hematology specialists and use of standardized care pathways may help reduce variation in practice and improve patient safety. In summary, intravenous fluid therapy has been a long standing part of managing vaso-occlusive crisis in sickle cell disease, with the goal of preventing dehydration and reducing sickling. However, there is limited evidence to support routine use of normal saline or any specific fluid regimen, and concerns about fluid overload, pulmonary edema, and even worse clinical outcomes have been raised in observational studies. Clinicians should use fluids judiciously, monitor patients closely, and prioritize individualized care while recognizing the gaps in evidence and the need for well designed clinical trials to guide future practice [1,2,4,7,15].

Case Presentation

Patient's history and Physical Examination

This case involves a 42-year-old male with known sickle cell disease who presented to the emergency department with severe generalized body pain, predominantly in the lower back and legs, for the past two days. He described the pain as constant, throbbing, and rated it at nine out of ten, with minimal relief from over-the-counter analgesics. The pain began gradually and had worsened despite increased oral fluids and home pain medications. He denied recent trauma, infections, or unusual physical activity. He reported that similar episodes had occurred intermittently throughout his life, usually requiring hospital visits once or twice a year, but this episode was more severe than previous ones. The patient had limited follow-up for sickle cell disease in recent years. He denied prior strokes, acute chest syndrome, or pulmonary hypertension. His medications included folic acid daily and occasional painkillers. He had no known drug allergies. He did not smoke, drink alcohol, or use illicit drugs. Family history was significant for sickle cell disease in a sibling but no known kidney or heart disease. On examination in the emergency department, the patient appeared uncomfortable, lying still on the stretcher. He was alert and oriented. Vital signs showed heart rate 110 beats per minute, blood pressure 148/88 millimeters of mercury, respiratory rate 20 breaths per minute, and oxygen saturation 95 percent on room air. He appeared mildly dehydrated with dry oral mucosa. General examination revealed mild pallor of the conjunctiva and slight icterus of the sclera. There was no generalized lymphadenopathy. Cardiovascular examination showed normal heart sounds with no murmurs. Peripheral pulses were palpable, and capillary refill was slightly delayed. Respiratory examination revealed clear breath sounds bilaterally at presentation, and the patient was not in respiratory distress. Abdominal examination revealed a soft, non-tender abdomen. The liver edge was palpable about one to two centimeters below the right costal margin and felt firm. The spleen was palpable approximately two centimeters below the left costal margin. Skin examination showed scattered hyperpigmented areas over the forearms and lower legs, consistent with previous sickle cell–related changes. There were no open wounds or acute lesions. There was no peripheral edema. The patient was started on intravenous normal saline for mild dehydration at the standard maintenance rate. After receiving approximately 500 milliliters, he developed acute shortness of breath and oxygen desaturation to 85 percent on room air. Physical examination revealed increased work of breathing and fine basal crackles on auscultation. A chest X-ray performed immediately demonstrated bilateral alveolar infiltrates consistent with **acute pulmonary edema** (Image 1).

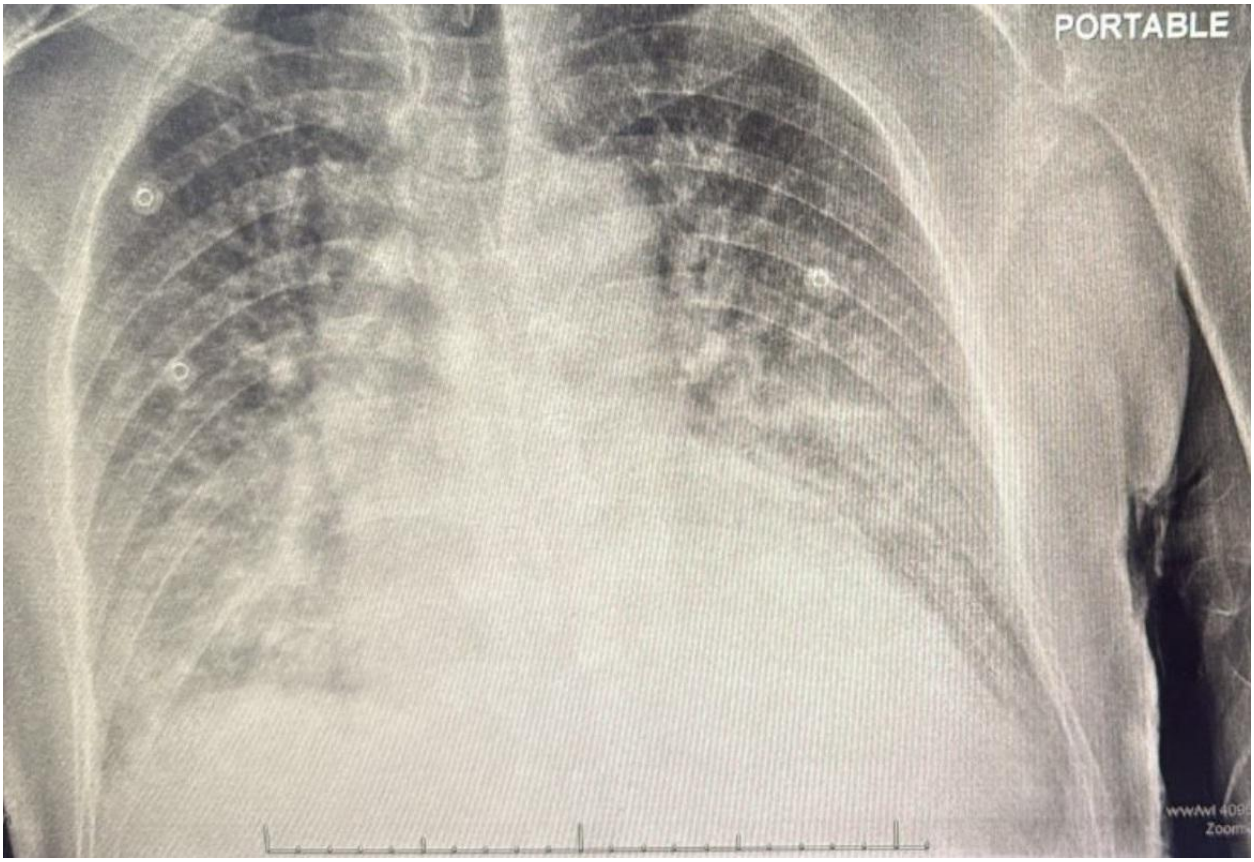

Image 1: Chest X-ray showing bilateral alveolar infiltrates consistent with flash pulmonary edema.

The acute onset of pulmonary edema following a relatively small volume of normal saline raised concern for fluid intolerance, potentially related to underlying cardiac or renal compromise. Differential diagnoses considered at this stage included acute chest syndrome, cardiogenic pulmonary edema, volume overload from intravenous fluids, and atypical infections. Given the timing immediately after fluid administration, fluid-induced pulmonary edema was considered the most likely cause. Although the patient had no prior known kidney disease, the rapid development of pulmonary edema after standard fluid infusion raised suspicion for **underlying sickle nephropathy**, which can impair the kidney's ability to handle even modest fluid loads. Sickle nephropathy is characterized by glomerular and tubular dysfunction due to chronic microvascular occlusion and ischemia in the kidneys. Patients may have subclinical renal impairment, which is often undiagnosed until stressors like intravenous fluid administration reveal limited renal reserve.

Diagnostic Reasoning:

Following the development of acute shortness of breath and hypoxia after intravenous normal saline, the patient underwent urgent evaluation in the hospital. The sudden onset of pulmonary edema following a relatively small fluid infusion raised immediate concern for fluid intolerance, prompting careful laboratory and imaging assessment. Baseline investigations were obtained to evaluate organ function, hematologic status, and possible contributors to pulmonary edema. Initial complete blood count showed a hemoglobin level of 9.2 grams per deciliter, consistent with baseline anemia in sickle cell disease. White blood cell count was 14,500 per microliter, mildly elevated, likely reflecting stress and inflammation rather than infection. Platelet count was 210,000 per microliter, within normal limits. Peripheral blood smear showed sickle-shaped red cells and occasional target cells, but no blasts or abnormal immature cells were seen. These findings were consistent with chronic hemolytic anemia of sickle cell disease and excluded an acute hematologic malignancy. Renal function tests revealed serum creatinine of 1.6 milligrams per deciliter and blood urea nitrogen of 38 milligrams per deciliter, mildly elevated from normal laboratory ranges and above his known baseline. Electrolytes were within normal limits. These results indicated impaired renal function, suggesting the patient had **subclinical or previously undiagnosed chronic kidney disease**, likely related to sickle nephropathy. Liver function tests showed mild elevation of transaminases and normal bilirubin, thought to be secondary to chronic hemolysis rather than acute hepatic injury. Inflammatory markers were mildly elevated, with C-reactive protein of 22 milligrams per liter and erythrocyte sedimentation rate of 38 millimeters per hour. Blood cultures were drawn to rule out infection, but ultimately remained negative. Urinalysis demonstrated trace proteinuria, supporting the possibility of renal dysfunction, although there was no hematuria or

active sediment. Following the onset of hypoxia, a chest X-ray was obtained, showing bilateral alveolar infiltrates consistent with **acute pulmonary edema**. This confirmed the clinical suspicion of fluid overload. There was no evidence of consolidation or focal pneumonia, and the heart size appeared normal, suggesting that the edema was more likely related to impaired renal fluid handling rather than primary cardiac failure. Given the acute nature of the pulmonary edema after a modest 500-milliliter infusion of normal saline, the differential diagnosis included acute chest syndrome, cardiogenic pulmonary edema, volume overload due to renal impairment, and atypical infections. Acute chest syndrome was considered; however, the patient had no fever, new pulmonary infiltrates typical of infection, cough, or chest pain, and the rapid onset after fluid administration favored a **non-infectious cause**. Cardiogenic pulmonary edema was less likely as he had no known cardiac disease, normal heart size on X-ray, and no murmurs or clinical evidence of heart failure. Infection was considered, but blood cultures and inflammatory markers did not suggest sepsis. The most plausible explanation was **fluid-induced pulmonary edema secondary to impaired renal function from sickle nephropathy**. Sickle nephropathy causes chronic glomerular and tubular injury due to repeated microvascular occlusion, ischemia, and infarction in the kidneys. This can reduce the kidney's ability to handle fluid loads, even small volumes, leading to pulmonary congestion. The timing of the edema immediately after the normal saline infusion strongly supported this mechanism. Additional laboratory tests were obtained to further assess renal function and fluid balance. Serum albumin was mildly low at 3.2 grams per deciliter, which may have contributed to reduced plasma oncotic pressure and predisposition to pulmonary edema. Serum bicarbonate was slightly decreased, consistent with mild metabolic acidosis. Urine protein to creatinine ratio was elevated, indicating chronic proteinuria, further supporting underlying sickle nephropathy. Echocardiography was performed and revealed normal left ventricular systolic function, no significant valvular disease, and no evidence of pulmonary hypertension. This ruled out a primary cardiac cause of pulmonary edema. The overall diagnostic reasoning integrated both **clinical events and laboratory findings**. The patient's acute pulmonary edema occurred immediately after a small fluid bolus, which was disproportionate to the volume given. Laboratory data demonstrated mild renal impairment with proteinuria and reduced bicarbonate, consistent with chronic sickle nephropathy. Cardiac evaluation was normal, excluding primary heart failure. Infection and acute chest syndrome were unlikely due to the absence of fever, consolidation, or systemic infection markers. Taken together, the most likely diagnosis was **flash pulmonary edema triggered by routine normal saline infusion in a patient with previously undiagnosed sickle nephropathy**. The event highlighted the kidney's limited ability to manage fluid loads in the context of chronic sickle cell–related renal injury. This explained why the patient developed acute respiratory distress after a standard fluid volume, which would normally be tolerated in patients with normal renal function. This diagnostic process illustrates the importance of integrating clinical presentation, lab results, and imaging. The sequence of events, combined with renal dysfunction markers, allowed the team to distinguish fluid-induced pulmonary edema from other potential causes such as infection or cardiac failure. Recognition of sickle nephropathy as a predisposing factor was crucial for guiding further fluid management and avoiding recurrence of pulmonary edema. The case also underscores the need for careful fluid administration in adult patients with sickle cell disease, particularly those with subclinical renal impairment, which may not be apparent without baseline lab testing.

Management course

Management focused on stabilizing the patient, addressing acute complications, and preventing further fluid-related events while arranging ongoing care for sickle cell disease and underlying renal impairment. Upon hospital admission, the patient was placed under close monitoring in a high-dependency unit, with continuous assessment of oxygen saturation, respiratory status, blood pressure, heart rate, and urine output. Given the development of acute pulmonary edema after fluid infusion, careful attention was paid to fluid balance and respiratory support. Oxygen therapy was initiated via nasal cannula to maintain saturations above 94 percent. The intravenous normal saline infusion was immediately stopped. Diuretics were administered cautiously to relieve pulmonary congestion, while monitoring for electrolyte shifts and hemodynamic stability. The patient was positioned upright to improve lung expansion and comfort. Continuous clinical assessment ensured early detection of worsening hypoxia or signs of respiratory failure. Pain management for vaso-occlusive crisis was a priority. Analgesia was escalated using intravenous opioids, titrated to effect, with careful monitoring for sedation and respiratory depression given his compromised oxygenation. Non-pharmacologic measures, including warm compresses and gentle movement, were employed to reduce musculoskeletal pain. Laboratory monitoring was continued, with repeat renal function tests, electrolytes, and blood counts to guide ongoing care. Given evidence of subclinical sickle nephropathy, fluid administration was carefully restricted and guided by clinical status, urine output, and weight changes. Maintenance fluids were given at minimal rates, avoiding boluses unless critically needed. Transfusion support was considered for worsening anemia and hypoxia. A single packed red blood cell transfusion was administered to improve oxygen-carrying capacity and reduce sickling risk. Blood pressure and oxygenation were monitored closely during and after transfusion. Multidisciplinary care was coordinated, involving hematology, nephrology, and respiratory teams. Sickle cell management focused on preventing further vaso-occlusive episodes, monitoring for acute chest syndrome, and planning long-term follow-up for renal function. The patient was counseled regarding fluid management, signs of pulmonary complications, and the importance of regular follow-up for sickle nephropathy. Over the next 48 hours, the

patient's oxygenation improved, respiratory rate normalized, and pulmonary edema resolved on repeat chest imaging. Pain gradually decreased with ongoing analgesia, and he remained hemodynamically stable. He was discharged on careful outpatient follow-up, including hematology and nephrology review. Prognosis was considered favorable with avoidance of further fluid overload, vigilant monitoring, and ongoing management of sickle nephropathy, though he remained at risk for future complications of both VOC and renal impairment.

Discussion

This case highlights several important clinical challenges in the management of vaso-occlusive crisis in sickle cell disease and the controversial role of routine fluid administration. Vaso-occlusive crisis remains the most common cause of hospitalization among adults with SCD and contributes substantially to morbidity, mortality, and health care utilization worldwide [1,2]. Because dehydration is thought to worsen sickling and microvascular obstruction, clinicians have traditionally used intravenous fluids, often normal saline, alongside analgesia in VOC management [1,3]. However, the practice of routine intravenous fluid administration, especially using normal saline, is based largely on historical practice rather than strong evidence, and emerging studies raise concerns about potential harm [1,4]. The clinical reasoning in this case underscores the need to weigh expected benefits against possible risks, particularly fluid overload and pulmonary complications. The theoretical rationale for fluid administration in VOC rests on the premise that dehydration increases plasma osmolarity, encourages hemoglobin S polymerization, and thereby worsens red cell sickling and microvascular occlusion [3]. Based on this physiologic model, expanding intravascular volume should theoretically improve microvascular flow and reduce sickling [3]. Despite this belief, there are no large randomized controlled trials that definitively demonstrate benefit from routine intravenous fluid therapy during VOC [1,4]. Systematic reviews incorporating retrospective data reveal limited evidence supporting current fluid practices and highlight significant variation in type, rate, and volume of fluids used [1,4]. Normal saline is the most frequently used intravenous fluid in VOC, yet it may not be the ideal choice. Studies show that sickle red blood cells exposed to normal saline have increased stiffness and transit times in microfluidic models, suggesting that normal saline may paradoxically worsen microvascular occlusion rather than improve it [5]. Normal saline's slightly higher osmolarity and potential to cause hyperchloremic acidosis may further promote hemoglobin S polymerization, intensifying sickling in vulnerable patients [1,5]. These in vitro findings, while not definitive clinical proof, lend biological plausibility to concerns about routine normal saline use. Clinical data from retrospective analyses suggest that the practice of routine normal saline boluses may correlate with worse pain control, higher admission rates, and longer emergency department stays [1,4]. In one study of 400 VOC patients, those who received a normal saline bolus had less improvement in pain scores and a higher rate of admission compared with those who did not receive a bolus [1,4]. The reasons for this are not fully clear, but the association raises concern that fluid choices may influence pain trajectories and hospital resource use. More importantly, evidence shows that volume overload is a common and significant complication of intravenous fluid therapy in VOC. In a retrospective cohort of adults treated with standard fluid regimens, 21 percent developed fluid overload defined by pulmonary edema or signs of fluid excess [4,6]. Patients with fluid overload had longer hospital stays with a median of 6 days compared with 4 days for those without overload and required interventions such as diuretics [4,6]. Despite a protocol aimed at 3 liters per 24 hours, many patients developed overload, and fluid regimens were often not adjusted even in those with prior overload history [4,6]. In other series, greater total fluid volume administered in the first 24 hours was associated with a higher risk of adverse events including new oxygen requirements, acute chest syndrome, acute kidney injury, and ICU transfer [7]. In one analysis, each liter of intravenous fluid given in the first 24 hours was associated with increased odds of adverse events with an odds ratio of 1.899 and 95 percent confidence interval 1.319 to 2.733 [7]. These associations do not prove causation, but they raise concern that excessive or unselected fluid administration in VOC may worsen clinical outcomes and contribute to complications traditionally attributed to disease progression alone. The risk of pulmonary complications is particularly relevant given that acute chest syndrome is a leading cause of mortality in SCD, often evolving after VOC hospitalization [8]. While studies have not definitively linked intravenous fluid use to acute chest syndrome, the potential for fluid overload to produce pulmonary edema which can mimic or trigger acute chest syndrome supports caution [1,7]. In autopsy series of adults with SCD who died unexpectedly, pulmonary edema was the most common pathological finding observed in nearly 50 percent of cases [4]. These data emphasize that pulmonary fluid balance in SCD is fragile and may be easily tipped by routine fluid administration. Another important issue is the risk of kidney injury. Although volume expansion is expected to reduce acute kidney injury by improving renal perfusion, excessive intravenous fluids may instead cause intrarenal venous congestion and contribute to kidney injury particularly in patients with underlying sickle nephropathy [7,9]. Observational data demonstrate that acute kidney injury occurs in about 17 percent of VOC admissions, and while hypotension and NSAIDs contribute, fluid overload likely plays a part in hemodynamic derangements at the renal level [7,9]. Balanced fluid choices like lactated Ringer's have been associated with better short-term outcomes including shorter hospital stays and lower readmission rates in extensive database analyses compared with normal saline [5]. These findings suggest that fluid type not just volume may influence clinical outcomes. Despite these concerns, intravenous fluids remain a central part of many VOC protocols. Emergency and inpatient guidelines often recommend intravenous hydration to correct deficits and replace ongoing losses, especially when severe pain, vomiting, or poor oral intake limit drinking [3]. Yet, even guideline bodies such as the American Society of

Hematology have not issued strong recommendations for or against specific fluid strategies reflecting the underlying evidence gaps [1,4]. This ambiguity underscores a critical need for personalized fluid management based on individual volume status, renal function, and cardiopulmonary reserve rather than routine fixed-dose fluid boluses for all VOC patients. Emerging questions that merit further research include whether oral hydration might suffice for many patients, how to define meaningful dehydration in VOC, and whether balanced crystalloids should replace normal saline in standard practice [4,5]. Research also needs to identify clinical metrics that help tailor fluid therapy such as bioimpedance to assess volume status or biomarkers of fluid tolerance to reduce the risk of overload. Without such tools, clinicians risk overshooting the therapeutic target of euvolemia and inadvertently causing harm. In summary, the management of VOC in SCD involves balancing the theoretical benefit of hydration against real risks of fluid overload, pulmonary edema, acute chest syndrome, and kidney injury. Retrospective studies involving hundreds of patients show that a substantial proportion develop fluid overload with associated longer hospital stays and that routine normal saline boluses may worsen pain control without clear benefit [1,4,6,7]. While intravenous fluids may be necessary in some patients with true dehydration or poor oral intake, the evidence supports a more judicious individualized approach to fluid therapy and further high-quality research is urgently needed.

Conclusion

This case teaches clinicians that routine intravenous fluid administration in vaso-occlusive crisis can be more harmful than beneficial when applied indiscriminately. Evidence from retrospective studies shows that routine normal saline boluses are associated with worse pain control, increased rates of fluid overload in about one in five patients, longer hospital stays, and higher risk of pulmonary and renal complications [1,4,6,7]. Fluid therapy should be individualized based on the patient's actual hydration status, renal function, and cardiopulmonary reserve rather than given empirically to all VOC patients. Balanced crystalloids may offer advantages over normal saline but high-quality randomized data are lacking [5]. Careful monitoring for signs of fluid intolerance, avoidance of excessive volumes, and prompt adjustment of therapy are essential to reduce morbidity. Thoughtful fluid management is a key lesson from this case.

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