

1 **Type of Article:** Case Report

2 **Title:** Survival Following a Fentanyl-Related Mass Casualty Event: A Case Report

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Clinical Question Box

What should clinicians recognize when a patient presents after suspected cocaine use with altered mental status or prolonged unconsciousness?

Clinicians should consider polysubstance toxicity rather than attributing the presentation solely to cocaine. Mixed stimulant-opioid exposure may produce overlapping or conflicting toxidromes combining opioid-related central nervous system depression and immobilization with stimulant-associated hypertension, vasospasm, and stroke risk. Patients should be promptly evaluated for multisystem complications including rhabdomyolysis, acute kidney injury, compartment syndrome, aspiration, and cerebrovascular injury.

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23 **Abstract**

24 **Opioid** contamination of recreational substances has become a significant contributor to overdose-
25 related morbidity and mortality in the United States. **The co-use of opioids and stimulants** produces
26 a mixed toxidrome combining opioid-induced central nervous system depression and
27 immobilization with stimulant-associated vasospastic and prothrombotic effects. We report a
28 survivor of **an overdose event** in which multiple individuals unknowingly **used** fentanyl-
29 contaminated cocaine at a social gathering. Two individuals were found deceased at the scene,
30 while a 53-year-old man was found unresponsive but survived, likely due to positional airway
31 protection. On presentation, he was hypertensive and confused, with significant right-sided soft
32 tissue swelling. Urine analysis was positive for cocaine and fentanyl. Laboratory evaluation
33 revealed markedly elevated creatine kinase levels, and imaging demonstrated an acute small vessel
34 infarct in the left corona radiata. The patient's course was complicated by severe rhabdomyolysis
35 and acute kidney injury requiring emergent dialysis, with concern for compartment syndrome. This
36 case highlights **the diagnostic complexity of mixed stimulant-opioid exposure** and demonstrates
37 how prolonged immobilization, positional airway protection, and delayed recognition of mixed
38 toxidromes can influence survival outcomes **following polysubstance overdose**.

39 **Keywords:** Fentanyl contamination, cocaine, mixed toxidrome, **stimulant-opioid co-use**, case
40 report

41 Introduction

42 Fentanyl is a synthetic opioid used in the setting of anesthesia and severe, chronic pain
43 relief. Its illicit use has increased significantly in the last two decades, along with the number of
44 drug overdose deaths seen in the United States.^{1,2} Illicit fentanyl analogs, historically referred to
45 as “China White,” first emerged in U.S. heroin markets during the 1980s, with recorded fatalities
46 occurring shortly after.³ Although not all of these substances originated from China, the term
47 became associated with illicit fentanyl products distributed outside of medical settings. Since 2013,
48 overdose deaths involving illicitly manufactured fentanyl have risen sharply as fentanyl and
49 fentanyl analogs increasingly entered the U.S. illicit drug supply through international trafficking
50 networks.⁴ This trend became apparent in California in 2016, when Sacramento County
51 experienced a marked increase in fentanyl-related overdoses associated with counterfeit opioid
52 tablets, including tablets sold as Norco and oxycodone; UC Davis Medical Center reported
53 multiple severe overdose cases during the regional outbreak.⁵ Because fentanyl is highly lipophilic,
54 it is rapidly absorbed in the body but slowly excreted, allowing it to act quickly and remain active
55 for long periods of time.⁶

56 Cocaine is a stimulant that is a major source of abuse in the US and worldwide. It works
57 by inhibiting the reuptake of the monoamines dopamine, norepinephrine, and serotonin, causing
58 euphoria and high addictive potential.⁷ Common routes of use include inhalation, intranasal use,
59 and intravenous injection, with effects that can occur rapidly depending on the route of
60 administration. The main concern with cocaine intoxication and overdose is its potent
61 sympathomimetic effects, including severe tachyarrhythmias, hypertension, and increased risk for
62 stroke.⁸ Fentanyl contamination of non-opioid recreational substances has become increasingly
63 reported, particularly in stimulant supplies like cocaine. Such contamination leads to unintentional

64 overdoses, particularly in users who lack tolerance to opioids.⁹ Although unintentional fentanyl
65 exposure is more commonly associated with counterfeit opioid tablets, increasing reports have
66 demonstrated fentanyl contamination in stimulant supplies, including cocaine, placing recreational
67 and opioid-naïve users at increased risk for fatal overdose.¹⁰

68 In this context, patients presenting with unintentional or purposeful **co-use** of fentanyl and
69 cocaine prompt urgent care, as this combination is a major driver of stimulant-related overdoses
70 and subsequent mortalities in the U.S.¹¹ This combination produces a mixed toxidrome in which
71 stimulant-induced vasospastic and prothrombotic effects may occur alongside opioid-induced
72 central nervous system depression and prolonged immobilization. Together, these overlapping
73 mechanisms increase the risk for multisystem complications and obscure diagnosis. The present
74 account describes a survivor of **an overdose event** involving suspected fentanyl-contaminated
75 cocaine use at a social event, resulting in **two** fatalities.

76 **Case Report**

77 A 53-year-old man was found unresponsive for an unknown period of time by Emergency Medical
78 Services (EMS) after **an overdose event involving multiple individuals**. He was found in a home
79 with two other deceased individuals and subsequently brought to the Intensive Care Unit. Past
80 medical history was notable for hypertension, bipolar disorder, hyperlipidemia, obesity, and
81 chronic knee pain controlled with Norco, Tylenol, and ibuprofen. After being discovered by EMS,
82 the patient admitted to recreational use of cocaine during the previous night's social gathering,
83 where multiple individuals reportedly purchased and **used** what they believed to be cocaine from
84 a street vendor. The patient denied any intention to use fentanyl or additional pain medications,
85 suggesting a possible unintentional opioid exposure through **an adulterated stimulant supply**. He
86 also denied any suicidal or homicidal ideation. On admission, however, the patient had difficulty
87 providing a clear history. He could not recall how long he was incapacitated, but reported right
88 neck and shoulder pain and difficulty hearing.

89 Upon presentation, the patient was afebrile (36.7°C) with a pulse of 89 beats/minute, blood
90 pressure of 183/122 mmHg, respiratory rate of 15 breaths/minute, and an oxygen saturation of
91 100% on room air. Physical exam revealed an ill-appearing, confused man with chemosis and a
92 right-sided induration of the neck. His right shoulder was swollen and tense, with scattered areas
93 of patchy erythema and tenderness to minimal palpation (Fig. 1). Neurological exam demonstrated
94 no gross motor or sensory deficits bilaterally, and both radial pulses remained palpable. No
95 cardiovascular or pulmonary abnormalities were noted.

96 Laboratory studies on admission were notable for a potassium level of 6.9 mmol/L, blood urea
97 nitrogen of 39 mg/dL, creatinine of 5.22 mg/dL, and a markedly elevated creatine kinase (CK) of

98 153,500 U/L (Table 1, Fig 2). Liver enzymes were also elevated, with an alanine aminotransferase
99 of 513 U/L and aspartate aminotransferase of 876 U/L. The white blood cell count was $11.6 \times$
100 $10^3/\mu\text{L}$, hemoglobin was 18 g/dL, and platelet count was $292 \times 10^3/\mu\text{L}$. Serum acetaminophen,
101 ethanol, and salicylate levels were negative. A standard urine drug screen returned positive for
102 cocaine, fentanyl, and tetrahydrocannabinol.

103 Initial computed tomography (CT) of the head was nonacute; CT of the neck was without C-spine
104 fracture or subluxation. Right upper extremity x-rays were negative for acute bone injury. CT of
105 the chest, abdomen, and pelvis demonstrated subcutaneous and intramuscular edema or myositis
106 involving the patient's right shoulder and right upper back musculature (Fig. 3). Micronodular
107 opacities were found in the upper lobe of the right lung and nonspecific, mild subcutaneous edema
108 was seen adjacent to the right buttock. Additionally, the patient was found to have mild urinary
109 bladder thickening and a hyperattenuating material within the lumen of the stomach. Magnetic
110 resonance imaging (MRI) of the brain without contrast revealed an acute small-vessel infarct in
111 the left corona radiata, with follow-up CT performed (Fig 4). No suspicious white matter signals
112 or abnormalities were noted.

113 Based on these investigations, the initial assessment and plan were to address the massively
114 elevated CK levels and acute tubular necrosis secondary to rhabdomyolysis. The patient was sent
115 for emergent dialysis and referred to orthopedics to rule out compartment syndrome, due to a
116 history of prolonged unconsciousness post-overdose. With the patient's prolonged lateral
117 positioning while unresponsive, the injury was suspected to be secondary to pressure-induced
118 rhabdomyolysis and immobilization. Other concerns, including hypertensive emergency,
119 abnormal liver function tests, lung abnormalities concerning for aspiration or community-acquired

120 pneumonia, and stomach opacity were likely secondary to rhabdomyolysis and cocaine overdose,
121 and continued to be monitored.

122 After ruling out compartment syndrome and determining the stroke was likely secondary to drug
123 overdose, the patient continued dialysis and empiric treatment for pneumonia in the hospital. By
124 Day 3 of dialysis, the patient was downgraded to telemetry. By Day 5, the patient had completed
125 a 5-day course of treatment for pneumonia. After 9 days, consultation with nephrology determined
126 the patient's CK levels were continuing to drop and that kidney function had returned to adequate
127 ranges for discharge back to his home (Fig 2). The patient was discharged after a 9-day hospital
128 course. Post-discharge, the patient was set up to follow up with nephrology in 7 days.

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129 **Discussion**

130 Mixed stimulant-opioid overdose is an increasingly prevalent clinical presentation associated with
131 significant multisystem complications.¹² Opioid contamination of cocaine introduces an often
132 unrecognized risk, particularly among opioid-naïve individuals, in whom unexpected opioid
133 exposure may worsen clinical outcomes.¹³ This case is notable because multiple individuals
134 reportedly used what they believed to be cocaine at a social gathering, resulting in two fatalities
135 and one survivor. More importantly, this case highlights the diagnostic complexity of
136 polysubstance exposure involving both stimulant and opioid toxicity, particularly when patients
137 present with overlapping or conflicting toxidromes.

138 Recent reports have similarly described overdoses occurring in party or social settings in which
139 individuals unknowingly use fentanyl-contaminated stimulants, particularly cocaine.¹⁴⁻¹⁶ Such
140 scenarios may delay recognition of opioid toxicity, especially among opioid-naïve individuals who
141 do not anticipate fentanyl exposure after recreational stimulant use.¹⁷ Clinically, these
142 presentations can be misleading because patients may exhibit overlapping or atypical findings that
143 do not fit a single expected toxidrome. A reported history of cocaine use should therefore not
144 exclude concurrent opioid exposure, particularly given the increasing recognition of opioid
145 contamination in stimulant supplies. Additionally, patient-reported substance histories may be
146 incomplete or inaccurate because of impaired recall, stigma, uncertainty regarding the composition
147 of illicit substances, or reluctance to disclose opioid use. Clinicians should therefore maintain a
148 broad differential diagnosis and avoid anchoring on a single reported exposure. A normal
149 respiratory rate or oxygen saturation at presentation does not rule out prior opioid toxicity,
150 especially in patients who have partially recovered before medical evaluation.

151 An important limitation of this case is that confirmatory toxicologic testing beyond standard
152 hospital urine drug screening was not available. Although fentanyl and cocaine were detected, the
153 possibility of additional adulterants, including fentanyl analogs, nitazenes, synthetic opioids, or
154 other contaminants, cannot be excluded.¹⁸ Contemporary illicit stimulant supplies may contain
155 multiple substances not routinely identified on standard toxicology screening panels.
156 Consequently, the patient's presentation may reflect broader polysubstance exposure rather than
157 fentanyl contamination alone.

158 The combined effects of cocaine and opioids may produce parallel mechanisms of injury. Cocaine
159 is a potent sympathomimetic that promotes hypertension, vasospasm, endothelial injury, platelet
160 activation, and a prothrombotic state, thereby increasing the risk of cerebrovascular events.^{7,19,20}
161 In contrast, fentanyl produces central nervous system and respiratory depression, which can result
162 in loss of consciousness, prolonged unresponsiveness, and immobilization.²¹ Together, these
163 effects create a dual-pathway mechanism in which opioid-induced immobilization and stimulant-
164 induced vascular pathology may act simultaneously.

165 One of the most significant complications observed in this patient was severe rhabdomyolysis with
166 subsequent acute kidney injury requiring dialysis. Prolonged immobilization is a well-established
167 cause of muscle ischemia and necrosis, particularly in patients found unconscious for an unknown
168 duration.²² Sustained pressure on dependent muscle groups leads to myocyte breakdown, release
169 of intracellular contents such as creatine kinase and myoglobin, and subsequent renal tubular
170 injury.²³ Additionally, stimulant exposure itself may worsen muscle injury through increased
171 muscular activity, vasoconstriction, hyperthermia, and metabolic stress. In this patient, the
172 combined effects of prolonged immobilization and stimulant-associated muscle toxicity likely
173 contributed to the severity of rhabdomyolysis.²⁴ His prolonged lateral positioning while

174 **unconscious may have paradoxically contributed to both survival and injury.** While lateral
175 positioning may have provided partial airway protection and reduced the likelihood of fatal
176 respiratory compromise, prolonged pressure on the dependent extremity likely contributed to
177 severe unilateral soft tissue injury, rhabdomyolysis, and renal failure. These effects may be further
178 exacerbated by dehydration, vasoconstriction, and direct myotoxicity in the setting of unknown
179 **polysubstance** use, emphasizing the importance of early recognition and prompt management.²⁴

180 Neurologic complications may also be overlooked in overdose presentations. In this case, MRI
181 revealed an acute small-vessel infarct, which may be explained by cocaine-associated ischemic
182 stroke. Because altered mental status in overdose patients is often attributed solely to opioid-
183 induced central nervous system depression, focal neurologic pathology may be missed or
184 recognized late.²⁵ This underscores the importance of comprehensive neurologic evaluation in
185 patients with suspected cocaine exposure, unexplained deficits, or atypical recovery patterns.

186 In addition to rhabdomyolysis, clinicians should maintain a high index of suspicion for
187 compartment syndrome in patients with prolonged immobilization. In this case, unilateral
188 swelling, tension, and tenderness raised concern for evolving compartment syndrome. Early
189 orthopedic consultation is critical when suspicion arises, as delayed diagnosis can result in
190 irreversible neuromuscular damage or limb loss.

191 This case highlights the importance of avoiding anchoring bias in patients with suspected
192 recreational drug exposure. It would have been easy to attribute this patient's presentation solely
193 to stimulant intoxication, thereby missing the contribution of opioid-induced immobilization and
194 its associated complications. More broadly, the case reflects an evolving public health concern in
195 which fentanyl contamination of non-opioid substances places individuals at risk for unexpected
196 and severe outcomes. Increased awareness of this phenomenon is essential to support harm-

197 reduction strategies, including expanded naloxone access, education for stimulant users, and
198 broader recognition of fentanyl exposure risk in social or recreational settings.

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199 **Conclusion**

200 This case highlights the severe and often underrecognized consequences of mixed stimulant–
201 opioid exposure from fentanyl-contaminated cocaine. **Occurring during an overdose event**
202 **involving multiple unsuspecting individuals and several fatalities**, it illustrates how patients may
203 present with overlapping or atypical clinical features rather than a single predictable toxidrome.
204 Clinicians should maintain a high index of suspicion for polysubstance exposure, avoid anchoring
205 on a single agent, and promptly evaluate for multisystem complications. This case also underscores
206 the potential impact of positioning during overdose-related unconsciousness: prolonged
207 immobilization may contribute to rhabdomyolysis and pressure injury, whereas lateral positioning
208 may help preserve airway patency. More broadly, the case reflects the evolving public health threat
209 of fentanyl contamination in non-opioid substances and reinforces the importance of early
210 intervention, careful clinical assessment, and harm-reduction strategies.

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216 G.C. served as project coordinator and contributed to manuscript revision, proofreading, and
217 overall supervision of the project. All authors have read and approved the final manuscript and
218 agree with its content and data.

219 **Data Availability Statement:** The datasets used in the current study are available from the
220 corresponding author upon reasonable request.

221 **Generative AI Declaration:** During the preparation of this manuscript, the authors used OpenAI
222 to assist with proofreading and image editing to blur identifying body markings for patient
223 confidentiality. All content was subsequently reviewed and edited by the authors, who assume full
224 responsibility for the accuracy and integrity of the published work.

225 **Ethical Statement:** The patient provided written informed consent for the publication of this case
226 report. Identifying information has been omitted to protect patient confidentiality.

227 **Conflict of Interest:** The authors report no conflicts of interest in this work.

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304 **Table 1.** Initial laboratory findings on admission.

Laboratory Test	Result	Reference Range
WBC count	11.6 ×10 ³ /μL	4.0–11.0 ×10 ³ /μL
Creatine Kinase	153,500 U/L	39–308 U/L
Potassium	6.9 mmol/L	3.5–5.0 mmol/L
Creatinine	5.22 mg/dL	0.7–1.3 mg/dL
Blood Urea Nitrogen	39 mg/dL	7–20 mg/dL
Aspartate aminotransferase	876 U/L	10–40 U/L
Alanine aminotransferase	513 U/L	7–56 U/L
Cocaine	Positive	Negative
Fentanyl	Positive	Negative
Tetrahydrocannabinol	Positive	Negative

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307 **Figure and legends**

308

309 **Figure 1.** Pressure-related skin changes after suspected fentanyl-contaminated cocaine overdose

310 A. Right shoulder and neck swelling with patchy erythema; B. Back pressure-related erythema and
311 skin changes

312 **Figure 2.** Clinical timeline showing changes in creatine kinase and creatinine levels during
313 hospitalization

314 CK: Creatine kinase; Cr: Creatinine; ICU: Intensive care unit

315 **Figure 3.** CT findings of pressure-related soft tissue injury

316 A. Right shoulder soft tissue and intramuscular edema; B. Upper back soft tissue and intramuscular
317 edema

318 **Figure 4.** MRI and CT findings of acute small vessel infarct

319 A: Magnetic resonance imaging shows a small acute infarct; B: Computed tomography shows
320 subtle corresponding hypodensity.

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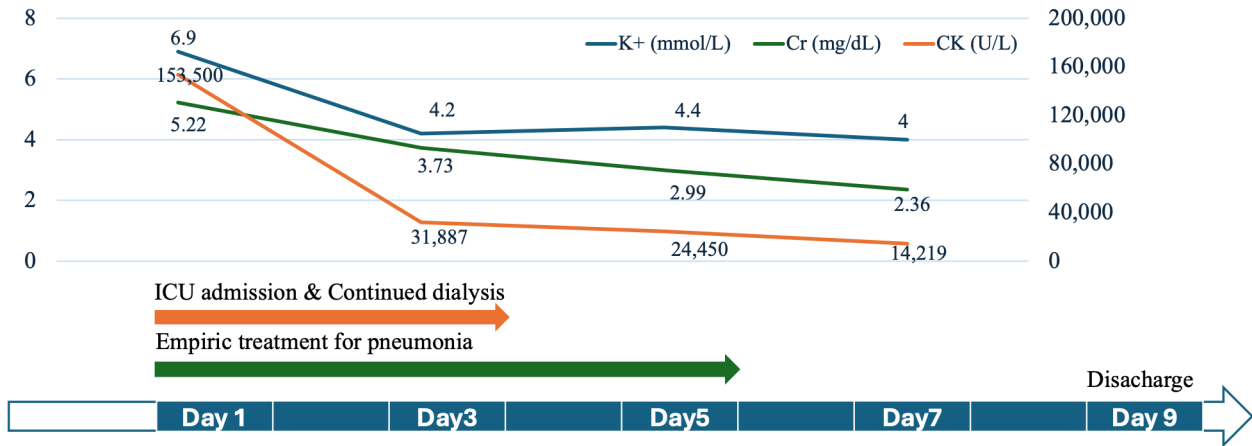
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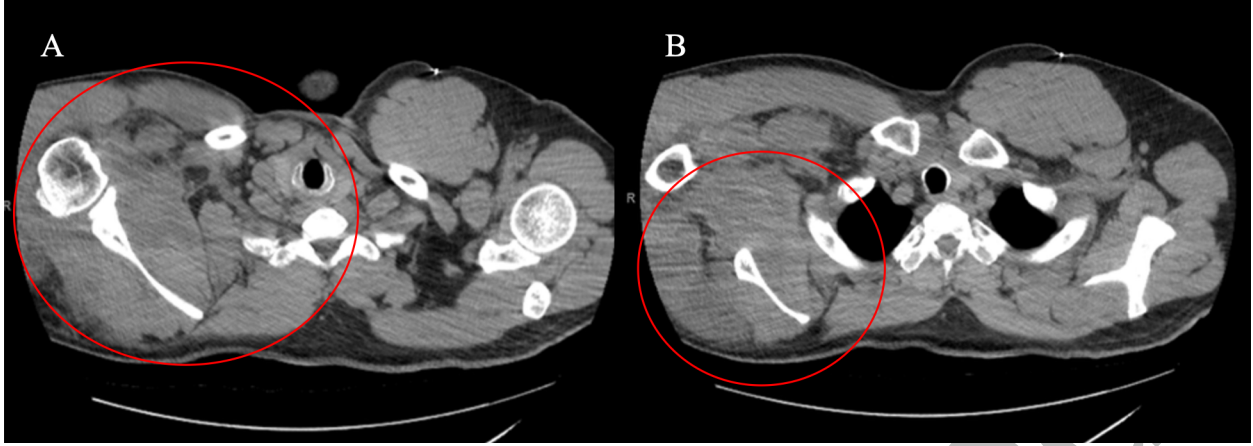
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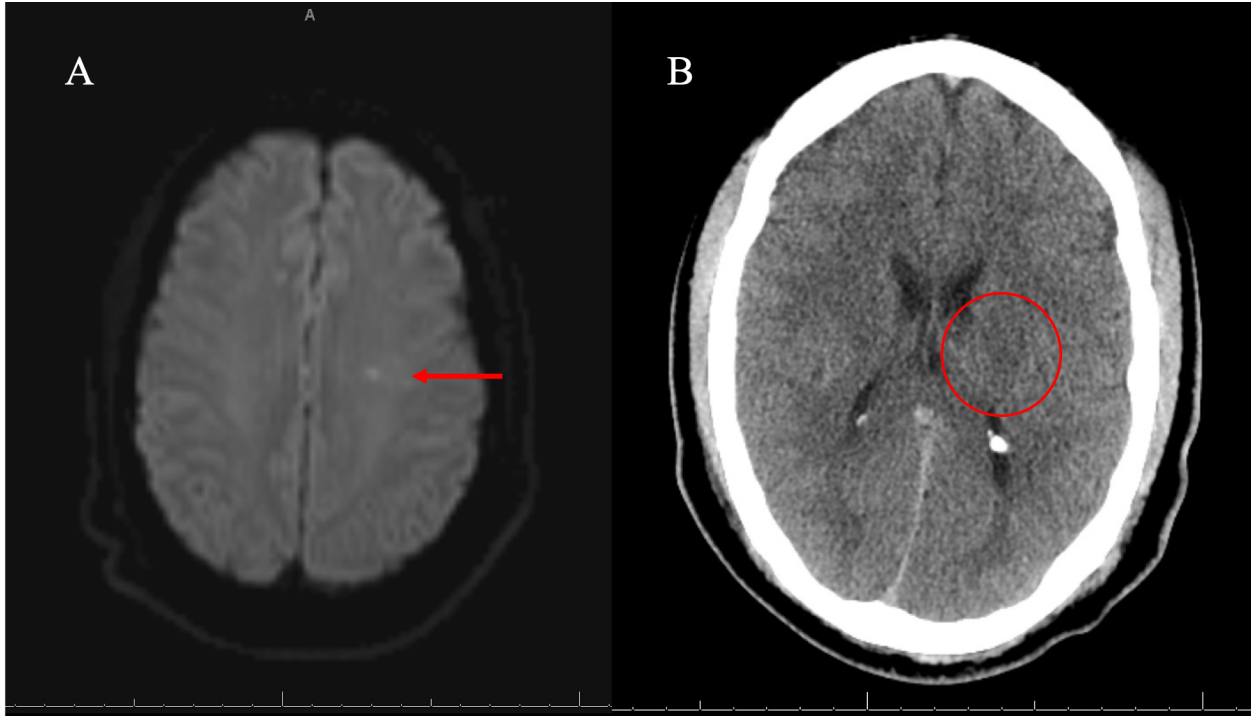
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