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Case Study

A Case Report On Acitrom Induced Coagulopathy With Seizure Disorder

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ABSTRACT

The purpose of this case report is to provide insight into the complex relationship between Acitrom-induced coagulopathy and seizure disorder in a patient with a history of cerebral venous thrombosis and coronary artery disease. Acitrom, an anticoagulant, requires vigilant monitoring to prevent bleeding complications. The patient's symptoms, including melena and bleeding from the oral cavity and gums, highlights the need for prompt diagnosis and treatment of coagulation issues. Phenytoin and Levetiracetam are two antiepileptic medications that are helpful in treating both the underlying cause and abnormal coagulation linked to seizure disorder. The case emphasizes the significance of individualised interventions, ongoing observation, and interdisciplinary cooperation to obtain the best possible patient results.

INTRODUCTION

Acitrom belongs to a particular class of coumarin derivatives and is known by its generic name, acenocoumarol. This specific derivative is used as an anticoagulant with the main goal of preventing the formation of blood clots. [1]. Its mechanism of action involves inhibiting the function of vitamin K reductase, which leads to the impaired carboxylation of vitamin K- dependent clotting factors (II, VII, IX, and X). This interference with the coagulation process necessitates regular monitoring of hematocrit, hemoglobin, international normalized ratio, and liver panel in patients. Furthermore, individuals taking acenocoumarol are not allowed to donate blood. It serves as a preventive measure against thromboembolic diseases occurring in cases of infarction and transient ischemic attacks [2]. Additionally, it is utilized for the management of deep vein thrombosis and myocardial infarction.

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Frequently, an excessive anticoagulant effect is observed with the use of coumarins. Although, coumarins are effective in preventing and treating venous and arterial thrombosis, they may also lead to bleeding (particularly if overdosed) or thrombosis (especially if under dosed). To reduce the risk of these complications, the prothrombin time (PT) – typically expressed as the international normalized ratio (INR) for standardized comparison across different centers and it is employed to monitor the level of anticoagulation associated with coumarin therapy. The INR range considered normal is 0.8 to 1.2. However, for patients on Vitamin K Antagonists (VKA) therapy, the desired INR target depends on the specific medical condition and usually ranges from 2.0 to 3.5 [3]. INR values below 2.0 are associated with an elevated risk of thromboembolic events, whereas INR values above 4.0 are linked to a higher risk of bleeding. VKAs are commonly used for both primary and secondary prevention of arterial and venous thromboembolism in patients with prosthetic heart valves, atrial fibrillation, peripheral arterial disease, antiphospholipid syndrome, as well as those who have experienced recurrent myocardial or cerebral infarction [4].

POTENTIAL COMPLICATIONS OF ACITROM-INDUCED COAGULOPATHY

Intracerebral haemorrhage has become increasingly common in patients with Acitrom-associated coagulopathy, posing a life-threatening scenario that requires prompt management [5]. Acitrom and other VKAs, whether used accidentally, on purpose, or as a result of an overdose of oral anticoagulant medication, deplete vitamin K-dependent proteins, resulting in extended prothrombin time and partial thromboplastin time, as well as obvious clinical bleeding signs [6]. Dysfunctional hemostasis can result from a variety of physiological disturbances, both congenital and acquired. Coagulopathy is a word that refers to any hemostasis deviation that results in excessive bleeding or clotting, with poor clot formation being the most prevalent manifestation. Abnormalities in coagulation tests, even in the absence of obvious clinical bleeding, might suggest the existence of coagulopathy in some circumstances. Intracerebral hemorrhage (ICH) is characterized by bleeding within the brain tissue, potentially affecting the ventricles and subarachnoid space as well. It constitutes up to 15% of all acute stroke cases and represents the deadliest manifestation of this condition. Additionally, if hematoma enlargement occurs within the first 24-48 hours of admission, it is strongly linked to unfavorable outcomes for these patients. In critically ill patients, coagulopathy can lead to a significant complication known as major bleeding, which includes fatal hemorrhage, hemodynamic instability, transfusion requirements, or intracranial hematomas. Managing coagulopathy in the ICU can be complex, particularly when it coexists with a thrombotic condition. Coagulopathy associated with intracerebral hemorrhage (ICH) directly impacts the neurological prognosis and functional outcome. However, there is a lack of high-quality evidence concerning the management of coagulopathies in neurocritical care [7]. Currently, there is a consensus that instances of life-threatening bleeding, such as intracerebral hemorrhage (ICH), necessitate prompt acitrom reversal to effectively address acitrom-associated coagulopathy as expeditiously as possible. Patients diagnosed with intracerebral hemorrhage (ICH) face a significant risk of hematoma enlargement within 24 hours after admission, especially if acitrom reversal is not completely achieved. Coagulation abnormalities are linked to neonatal encephalopathy (NE) [8]. Such coagulopathy can manifest as one of the facets of multi-organ dysfunction that arises after a perinatal hypoxic-ischemic insult. Patients with cerebral venous thrombosis, a condition involving blood clot

formation in the venous system of the brain, often experience frequent epileptic seizures.

COAGULOPATHY ASSOCIAED SEIZURE

Seizure-related coagulopathy is defined as the presence of abnormal blood clotting factors or impaired coagulation mechanisms in people who have seizures. Seizures have the ability to change the body's clotting mechanisms, resulting in coagulopathy [9]. This disease may raise the risk of bleeding or clot formation during or after a seizure. Epileptic seizures are regularly observed as a result of stroke, but there is little understanding of the numerous risk factors and how different stroke types, such as sinus thrombosis and bleedings, affect the likelihood of developing such seizures [10]. With an ageing population and advances in stroke treatment, it is critical to investigate seizure prevention strategies which have a substantial social and psychological impact on patients [11]. Given the aging population and advancements in stroke treatment, it becomes increasingly important to explore preventive measures for seizures, as they significantly affect patients both socially and psychologically. However, there is limited available data regarding the occurrence of seizures following other cerebrovascular events, such as transient ischemic attack (TIA), various types of bleeding, and intracranial venous thrombosis [12]. The occurrence of post-stroke seizures was not found to be linked to conventional vascular risk factors [13]. However, exploring the risk of seizures after specific types of cerebrovascular events could aid in the early identification of patients who may benefit from anticonvulsive treatment [14]. Proper evaluation and management of seizure-related coagulopathy is essential to minimize potential complications and ensure appropriate treatment for affected individuals. Medical professionals closely monitor coagulation parameters in patients with seizures to promptly identify and address any coagulation abnormalities that may arise [15]. Further investigation is needed to determine if these patients could potentially gain advantages from pre- emptive anticonvulsant therapy in the future.

TREATMENT FOR ACITROM INDUCED COAGULOPATHY

The primary objective of treating acitrom-induced coagulopathy is to manage the medication's excessive anticoagulation while assuring patient safety and preventing complications associated with bleeding. Timely recognition and assessment of coagulopathy are crucial. Patients receiving acitrom therapy should undergo close monitoring for indications of bleeding, bruising, or other abnormal bleeding manifestations [16]. Regular evaluation of prothrombin time (PT) and international normalized ratio (INR) is imperative to assess the degree of anticoagulation and identify any deviations from the desired therapeutic range. As a part of the management strategy for acitrom-induced coagulopathy, temporary discontinuation of the medication is advised upon suspicion or confirmation of the condition to prevent worsening of anticoagulation [17]. In situations where significant bleeding or severely elevated INR levels are observed, the administration of vitamin K serves as an effective method to reverse the effects of acitrom. Vitamin K can be given intravenously or subcutaneously, depending on the severity of the coagulopathy. For severe cases of acitrom-induced coagulopathy, when immediate reversal is necessary or when vitamin K is not recommended, medical practitioners may consider using either Prothrombin Complex Concentrates (PCC) or Fresh Frozen Plasma (FFP). These products contain essential clotting factors that aid in rapidly restoring normal coagulation function [18, 19]. Furthermore, an individualized treatment plan is essential for managing acitrom-induced coagulopathy, taking into account each patient's specific clinical circumstances. Factors such as age, existing medical conditions, and the severity

of the coagulopathy should be carefully considered when devising the appropriate treatment approach. This personalized strategy ensures optimal management and better outcomes for affected patients. After commencing the suitable treatment, it is crucial to conduct diligent monitoring of coagulation parameters, bleeding symptoms, and the patient's response to therapy. Regular assessments of INR levels and overall progress will be indispensable in evaluating the effectiveness of the treatment. Based on the patient's status and INR levels, adjustments to the treatment plan may be required to ensure optimal management of acitrom-induced coagulopathy [20]. Seeking guidance from a healthcare professional well-versed in managing anticoagulant therapy and coagulopathy is essential to develop a tailored and efficient treatment plan. Regular communication with the patient, along with comprehensive education about potential risks, the significance of medication adherence, and the importance of monitoring, are crucial elements in ensuring successful management of the condition.

MANAGEMENT OF SEIZURE-RELATED COAGULOPATHY

Treatment guidelines for seizure-related coagulopathy involve addressing both the underlying cause of seizures and managing the coagulation abnormalities.

Seizure Management:

The main objective is to successfully control and manage seizures. The core of seizure management is antiepileptic medications (AEDs) [21]. The type of AED used will be determined by the type of seizure and the patient's unique circumstances. AEDs that are often used include phenytoin, carbamazepine, valproic acid, and levetiracetam.

Coagulation Assessment:

Close monitoring of coagulation parameters is crucial in patients with seizures, especially if they are on anticoagulant therapy or have known bleeding disorders. Regular evaluation of prothrombin time (PT), activated partial thromboplastin time (aPTT), and platelet count helps detect coagulation abnormalities promptly [22].

Vitamin K Supplementation:

When coagulopathy is caused by a lack of vitamin K, vitamin K supplementation is required to restore normal clotting function. Depending on the severity of the deficit, this can be given orally or intravenously.

Specific Coagulation Factor Replacement:

In severe coagulopathy, such as hemophilia or other clotting factor deficiencies, specific coagulation factor replacement therapy may be required. This involves infusing the deficient clotting factor to correct the abnormality.

Tranexamic Acid:

Tranexamic acid may be considered in patients with ongoing bleeding or increased bleeding risk to reduce bleeding episodes and stabilize clot formation.

Individualized Treatment Plan:

Treatment should be implemented to each patient's specific clinical condition. When developing a treatment strategy, examine the actual cause of the seizures, the presence of co morbidities, and the level of coagulation abnormalities. Following the initiation of treatment, it is necessary to regularly and attentively assess both seizure control and coagulation markers. The treatment strategy may need to be changed based on the patient's particular response and progress during the follow-up period [23].

CASE HISTORY

A 55-year-old female was admitted to the general medicine department on March 15, 2023 with the complaints of headache, vomiting 2- episodes, burning sensation of both lower limbs, and heart burn. The patient was again admitted to the same department on June 24, 2023 with complaints of bleeding from the gums and oral cavity for 2 days,

about 5 to 6 episodes, black coloured stool for 1 day. She also has a history of seizures (GTC type) for 1 episode of around 15 minutes involving loss of consciousness, a day before her first admission, and a history of melena for 3 episodes, a day before her second admission. Her past medical history was Cerebral Venous Thrombosis and Coronary Artery Disease for 7 years, seizures for 5 years, hypothyroidism, and B/L Mild Renal dysfunction, Grade-II on regular treatment. Her past medication history includes T. Nicoumalone (Acitrom)- 2 mg OD, T. Thyroxin- 100 mcg OD, T. Atorvastatin- 10 mg HS, T. Aspirin- 150 mg OD, T. Aldactone- 25 mg OD, T. Enalapril- 2.5 mg BD, T. Ranitidine- 150 mg BD, and T. Calcium lactate- 300 mg OD. Then she was diagnosed as Acitrom-induced coagulopathy/ Seizure/ CAD/ Hypothyroidism/ Sinusitis and Peripheral neuropathy. On Physical examination, she appears to be conscious and oriented. Her body temperature was normal. Her heart rate was 88 beats per minute. Her blood pressure seemed to be normal and her oxygen saturation in room air was 99 %. Her laboratory investigation is given in the table 1. The diagnosis was made after a thorough clinical assessment of the patient's medical history, symptoms, and use of Acitrom as an anticoagulant medication. Laboratory tests were also conducted to evaluate coagulation parameters, such as prothrombin time (PT) and international normalised ratio (INR), to identify any abnormalities indicating a coagulopathy. The MRI report (Brain) of this patient showed the impression of bilateral prominent perioptic CSF spaces. A small vessel ischemic change was noted according to the Fazeka scale- Grade 1. T1, T2 and Flair hyper intensity was noted in the right transverse sinus, C/O bilateral maxillary and ethmoid sinus was noted. Magnetic Resonance Angiography (MRA) was normal and Magnetic Resonance Venography (MRV) was found to be irregular and defects in bilateral transverse, sigmoid and especially, superior sagittal sinus appeared to be thin which is an indicator of partial thrombosis with recanalization and there was no evidence of infarct or haemorrhage. ECHO report showed that Left ventricular internal diameter end diastole and end systole [LVid (d) and (s)] was found to be 5 and 3.5 cm respectively, which was an indicator of LV diastolic dysfunction With normal LV systolic function, the Left Ventricular Ejection Fraction (LVEF) was 60% indicated mitral regurgitation (MR-Grade I) and there was no clot or thrombus.

The ECG report showed that there was evidence of normal sinus rhythm on the first admission and AV Block- I, Possible Inferior Infarction on second admission. The best treatment for coagulopathy induced by Acitrom in association with seizures entails discontinuing Acitrom usage and addressing the underlying coagulation disorder. This may involve the administration of suitable doses of vitamin K and/or fresh frozen plasma to reverse the anticoagulant effects. Seizure management might necessitate the use of antiepileptic medications, along with supportive care to address any potential complications. Receiving prompt medical attention is essential to ensuring a safe and effective treatment strategy. This patient was managed with the medications that were noted in the table 2. On her first admission, she received acitrom as her main therapy (From day 1 to day 6). On her second admission, she complained of bleeding from her gums and oral cavity. Then, she received the therapy of Fresh frozen Plasma (FFP) for the first 4 days and got it when it was needed. After that, the patient’s complaints were reduced, and she was discharged. At the time of discharge, she was advised to continue the following tablets: 2 mg of Atorvastatin once a day, 25 mg of Metoprolol half a dose twice a day, 5 mg of Levetiracetam twice a day, 100 mg of Phenytoin twice a day, 150 mg of Thyroxin once a day, 500 mg of Paracetamol

thrice a day, 25 mg of Amitriptylline once a day, 20 mg of Sodium Valporate once a day and 25 mg of Acetazolamide once a day for 30 days. She was advised to receive a follow up care after 30 days.

INVESTIGATION

Table 1. The laboratory investigation data of the patient

Lab Parameters	Observed Value during 1st admission	Observed Value during 2nd admission	Reference Value
Total count	13,900	11000	4,500 to 11,000 cells/cu.mm
Differential count			
Polymorphs	82	77	40 to 70 %
Lymphocytes	14	16	20 to 40 %
Monocytes	5	7	2 to 6 %
Hemoglobin	14.5	11.6	11to15 g/Dl
Platelets	4.2	4.21	1.5 to 4.5 lakhs/cu.mm
Packed Cell Volume	52.3	52.1	39 to 49 %
Red blood cell	6.7	5.7	4.6 to 5.9 million/cu.mm
Random blood sugar	81	85	80 to 120 mg/dL
Liver function			
Total bilirubin	0.7	0.6	0.1-1.2 mg/dl
Direct Bilirubin	0.5	0.3	0.0-0.3 mg/dl
Indirect Bilirubin	0.2	0.3	0.2-0.8 mg/dl
SGOT	28	28	5-40 IU/L
SGPT	34	22	7-96 IU/L
Alkaline Phosphatase	90	98	44-147 U/L
Total Protein	5.2	5.9	60-80 g/L
Renal function			
Urea	58	35	8 to 40 mg/dL
Creatinine	1.2	1.0	0.6 to 1.2 mg/dL
Serum electrolytes			
Sodium	122	132	135 to 145 mEq/L
Potassium	6.8	4.0	3.5 to 5 mEq/L
Chloride	98	107	96 to 106 mEq/L
Prothrombin time	21.5	22.4	10-13 secs
INR	1.7	1.80	< 1.1

DRUG CHART

Table 2. Medication chart during 1st admission

Drugs	Dose	Route	Frequency	Start	Stop
Tab. Thyroxin	100 mcg	Oral	OD	Day 1	Day 6
Tab. Atorvastatin	10 mg	Oral	HS	Day 1	Day 6
Tab. B-complex	30.5 mg	Oral	OD	Day 1	Day 6
Tab. Metoprolol	25 mg	Oral	1/2 -0-0	Day 1	Day 6
Tab. levetiracetam	10 mg	Oral	BD	Day 1	Day 6
Tab. Acitrom	2 mg	Oral	0-0-1-0	Day 1	Day 6
Tab. Calcium	300 mg	Oral	TDS	Day 1	Day 6
Inj. Phenytoin	100 mg	IV	TDS	Day 1	Day 6
Inj. Ondansetron	2 mg/ml	IV	BD	Day 1	Day 1

Inj. Ranitidine	50mg/ ml	IV	BD	Day 1	Day 1
Tab. Aspirin	150 mg	Oral	0-1/2-0	Day 2	Day 6
Tab. Amitriptalline	25 mg	Oral	OD	Day 2	Day 6
Tab.Sodium icarbonate	325 mg	Oral	TDS	Day 2	Day 6
Tab. Ferrous sulphate	333.5 mg	Oral	OD	Day 2	Day 6
Inj. Calcium gluconate	10%	Slow IV for 10 min			
Cap. Omeprazole	20 mg	Oral	BD	Day 3	Day 6

Table 3. Medication chart during 2nd admission

Drugs	Dose	Route	Frequency	Start
Inj. Vitamin K	10 mg	IM	OD	Day 1
Inj. Streptochrome	1.2 g	IV	OD	Day 1
Inj.Tranxemic acid	500 mg	IV	OD	Day 1
Inj. Ranitidine	50 mg/2ml	IV	BD	Day 1
Tab. Clopidogrel	75 mg	Oral	OD	Day 1
Tab. Atorvastatin	10 mg	Oral	HS	Day 1
Tab. Metoprolol	25 mg	Oral	1/2-0-1/2	Day 1
Tab. Levitriacetam	100 mg	Oral	BD	Day 1
Tab. Phenytoin	100 mg	Oral	BD	Day 1
Tab. Amitriptalline	25 mg	Oral	OD	Day 1
Tab. Sodium bicarbonate	325 mg	Oral	TDS	Day 1
Tab.Calcium carbonate	300 mg	Oral	TDS	Day 1
Tab. Paracetamol	500 mg	Oral	TDS	Day 3

DISCUSSION

In this case, the recommended medications are prescribed for the subsequent ailments: In the initial admission of the patient, the seizure disorder was treated by means of administering the injection Phenytoin and Levetiracetam. The blood pressure was managed through the use of the tablet Metoprolol. The hypothyroidism was regulated by the tablet Thyroxin. The migraine (headache) was relieved by the tablet Amitriptylline. The coagulopathy condition of the patient was treated by prescribing the tablets Atorvastatin and aspirin. For heartburn and vomiting, single-day therapy was provided in the form of injections of Ranitidine and Ondansetron, respectively. Subsequently, the injection of Ranitidine was replaced with an Omeprazole capsule. For the treatment of the CVT condition, Acitrom tablets were recommended. Furthermore, the symptom of burning in both lower limbs (hypocalcaemia) could be treated by administering Calcium gluconate injections. During the patient's second admission, various complaints were addressed through the administration of certain medications. Bleeding from the gums and oral cavity was treated with injections of Vitamin-K, Streptochrome and Tranxemic acid. Seizure episodes were controlled with the use of Phenytoin and Levetiracetam tablets. To regulate the patient's blood pressure levels, Metoprolol tablets were given. Additionally, Atorvastatin and aspirin tablets were prescribed to treat the patient's coagulopathy condition, which was the same as that of the first admission. Symptomatic malena, characterised by the passage of black-coloured stool, was treated with Ranitidine injection and paracetamol tablets. In accordance with the guidelines pertaining to seizures linked to

coagulopathy, it is recommended that the treatment protocol be promptly initiated with the administration of seizure medications (AEDs), followed by anti-coagulant therapy, such as Acitrom. Subsequently, the coagulopathic condition can be treated with Vitamin-K supplementation, Streptochrome and Tranxemic acid injections. Furthermore, the patient received additional therapy in the form of Calcium, BCT, Sodium bicarbonate, and Ferrous sulphate tablets.

RECOMMENDATION

As per the guidelines, the additional therapy recommended is special factor replacement therapy. This therapy involves administering specific clotting factors to individuals with coagulopathies caused by deficiencies in those particular factors. The treatment process begins with identifying the deficient clotting factor through laboratory tests. Once the specific factor is identified, it can be obtained from purified human plasma or through recombinant technology. The replacement factor is then infused into the patient's bloodstream, effectively restoring their blood's ability to clot normally. The treatment process begins with identifying the deficient clotting factor through laboratory tests. Once the specific factor is identified, it can be obtained from purified human plasma or through recombinant technology. The replacement factor is then infused into the patient's bloodstream, effectively restoring their blood's ability to clot normally. This targeted therapy is customised to the individual's specific coagulation deficiency, minimising the risk of excessive bleeding episodes and improving overall quality of life. Regular monitoring of clotting factor levels, along with close collaboration between healthcare providers and patients, ensures that treatment is adjusted as needed to maintain optimal clotting function and prevent complications associated with coagulopathy.

CONCLUSION

The current case report highlights the intricate link between Acitrom-induced coagulopathy and seizure disorder in a patient with a history of cerebral venous thrombosis and coronary artery disease, in addition to the complex interactions between these two medical conditions. Acitrom, a popular anticoagulant, requires careful monitoring to avoid complications involving bleeding. The patient's symptoms of melena, bleeding from the oral cavity, and bleeding from the gums highlight the importance of promptly diagnosing and treating coagulation problems. Acitrom- induced coagulopathy must be treated by stopping the medicine, administering vitamin K, and using supportive techniques such fresh frozen plasma. Antiepileptic drugs like Phenytoin and Levetiracetam were used to treat the patient's seizure disease since they addressed both the underlying cause and abnormal coagulation. Only prevention of acitrom-induced coagulopathy is possible, thus careful medication administration, regular Precautionary the administration of drugs, regular INR level monitoring, and medication modifications as considered appropriate are all required to prevent the coagulopathy caused by Acitrom. Patients should be informed about the symptoms of bleeding as well as the need of taking their medications as directed. Antiepileptic drugs must be used as effectively as possible in cases of seizure-related coagulopathy. Monitoring coagulation parameters is crucial, and any irregularities must be dealt with quickly. A multidisciplinary strategy combining critical care, haematology, and neurology experts is advised to guarantee the most effective management. It is crucial to educate patients about seizure triggers, medication adherence, and the ability to recognise bleeding symptoms. In future cases, early intervention, individualized treatment plans, and close monitoring of both coagulation parameters and seizure control will facilitate the improvement of patient outcomes. This instance demonstrates

the importance of healthcare practitioners continuing to pay close attention to the subtle interplay between coagulopathy and underlying illnesses.

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CONFLICT OF INTREST

Authors have no conflict of interest to declare.

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