Case Report

A Case Report on Coumarin Derivatives Induced Coagulopathy

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ABSTRACT

Coumarins are effective for the prevention and treatment of venous and arterial thrombosis but may cause bleeding (especially if overdosed) or thrombosis (especially if under) To minimize the risk of these complications, the prothrombin time (PT) - generally expressed as the international normalized ratio (INR) is used to monitor the degree of coumarin-associated anticoagulation We describe a case of a 50-years male patient with atrial fibrillation with FVR who presented with complaints of abdominal pain at right hypochondrial region radiating to right shoulder since 10 days & also complaint of low grade fever, nausea, decreased appetite. Lab investigations showed increased PT and INR ratio based on that patient diagnosed as Acitrom induced Coagulopathy. Occurrence of Coagulopathy is a known complication of vitamin k antagonist. So providing counselling regarding all aspects of medications as well as lifestyle modifications and by giving patient information leaflets were most important for this condition

Key Words: Acitrom, INR, PT, Coagulopathy.

INTRODUCTION

Coumarin anticoagulants (COAs) are 4-hydroxycoumarin compound; the commercially available Coumarins include warfarin, Acitrom, acenocoumarol and phenprocoumon. Coumarins are effective for the prevention and treatment of venous and arterial thrombosis but may cause bleeding (especially if overdosed) or thrombosis (especially if under) To minimize the risk of these complications, the prothrombin time (PT) - generally expressed as the international normalized ratio (INR) to allow comparability among canters is used to monitor the degree of coumarin-associated anticoagulation. A normal INR is between 0.8 and 1.2. The INR for patients on VKA therapy varies according to the underlying condition but is typically between 2.0 and 3.5. An INR of less than 2.0 is associated with increased risk of thromboembolic events, while an INR of greater than 4.0 is associated with increased risk of bleeding. VKAs are routinely used for the primary and secondary prevention of arterial and venous thromboembolism in patients with prosthetic heart valves, atrial fibrillation, peripheral arterial disease, antiphospholipid syndrome, and recurrent myocardial or cerebral infarction. It works by inhibiting the C1 subunit of the enzyme vitamin K epoxide reductase (VKOR), which is necessary for the activation of the vitamin K-dependent coagulation factors (Factors II, VII, IX, and X) and regulatory proteins
(proteins C, S, and Z). Two treatment strategies are available for vitamin K induced Coagulopathy that is withholding oral anticoagulants and allowing the INR level to return to within the therapeutic range, or withholding oral anticoagulants and administering vitamin K. A third option, withholding oral anticoagulants, administering vitamin K and administering fresh frozen plasma.

**CASE REPORT**

A 50 years old male patient 50 kg was admitted in nephrology department with chief complaints of shortness of breath grade II -III, dry cough, abdominal pain at right hypochondrial region radiating to right shoulder since 10 days & also complaint of low grade fever, nausea, decreased appetite and pedal oedema increasing since 2-3 weeks, melena from 4 to 5 days, his past medical history includes patient was known DM since 20 years, K/H/O HTN for 1year, CAD- S/P CABG (2008), atrial fibrillation with FVR on Tab. Acitrom since 1 year and also H/O hyponatremia, hyperkalemia & anaemia and past medication history shows, the patient was under treatment with Tab. Gemer 1 tablet two times in a day, Tab. Ecosprin 75mg OD, Tab. Telma 20 mg OD, Tab. Acitrom 1mg once daily evening since 1 year Tab. Optineuron 1 tablet OD, Tab. Dytor plus 10mg ½ tablet, and Tab. Pantop 40mg OD. Patient family history was nothing significant. On general examination the patient was conscious and coherent & pallor+ and his physical examination includes PR-99bpm, BP-110/70 mm/Hg, on systemic examination CVS-S1S2+, RR-26CPM, RS- clear. Patient laboratory parameters shows as follows decreased level of Haemoglobin - 9.9gm, serum sodium 117 mEq/L and increased levels of PT >120,INR>5.26, serum potassium -5.3 mEq/L, based on the symptoms and lab investigation the patient was diagnosed with DRUG INDUCED COAGULOPATHY.

The treatment was given as follows Inj. Vitamin K 10mg IV once in a day for 2 days, Tab. Ecosprin 75mg once in day, Tab. Telma 20 mg once in a day, Tab. Optineuron 1tablet once in a day, inj. Pantop 40 mg once in a day, Tab. Aldactone 25mg two times in a day, inj. Lasix 10mg twice in a day, Tab. Gemer-1 tablet two times in a day. Based on the above information here we have suspected that this is an ADR of Tab. Acitrom (acenocoumarol) induced Coagulopathy.

**Causality assessment:**

To evaluate the relationship between the drug and reaction, we have performed causality assessment by using scales like WHO causality assessment scale, Naranjo scale and Karch & Lasagna scale and analysis of observed ADR (Table 1) & (Table 2).

**DISCUSSION**

Oral anticoagulation is an important part of long-term AF management to prevent embolic stroke and other systemic thromboembolic diseases. For decades, warfarin or oral Vitamin K antagonists were the main anticoagulants used. However, with the narrow therapeutic index and multiple drug and food interactions associated with warfarin, an alternative was needed. The coumarin derivative warfarin an anticoagulant works by preventing platelets from sticking from each other to form blood clots. It can cause serious bleeding so proper monitoring is required which is by INR monitoring. In our case, patient had a history of usage of Acitrom and had developed coagulopathy; this is also the reason for hospital admission. After
hospital admission as a clinical pharmacist we have identified adverse drug reactions as follows, the patient was under the medication with Acitrom (acenocoumarin), based upon the literature reviews and based on laboratory investigations we have concluded that this condition is due to the drug Acitrom and performed causality assessment, severity, preventability, predictability. After the identification we have immediately withdrawn the Acitrom and provided appropriate treatment. So, monitoring of INR, PT, is necessary during treatment with Acitrom.

CONCLUSION

Occurrence of Coagulopathy is a known complication of vitamin k antagonist. Coagulopathy must be suspected in patients on vitamin K antagonist presenting with abdominal pain, reduced appetite and melena. Due to this patient required additional monitoring and prolonged hospital stay. Correction of the Coagulopathy is equally vital in the treatment of this condition. Furthermore, this case projects the need for a better communication by means of providing counselling regarding all aspects of medications as well as lifestyle modifications and by giving patient information leaflets.

REFERENCES

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