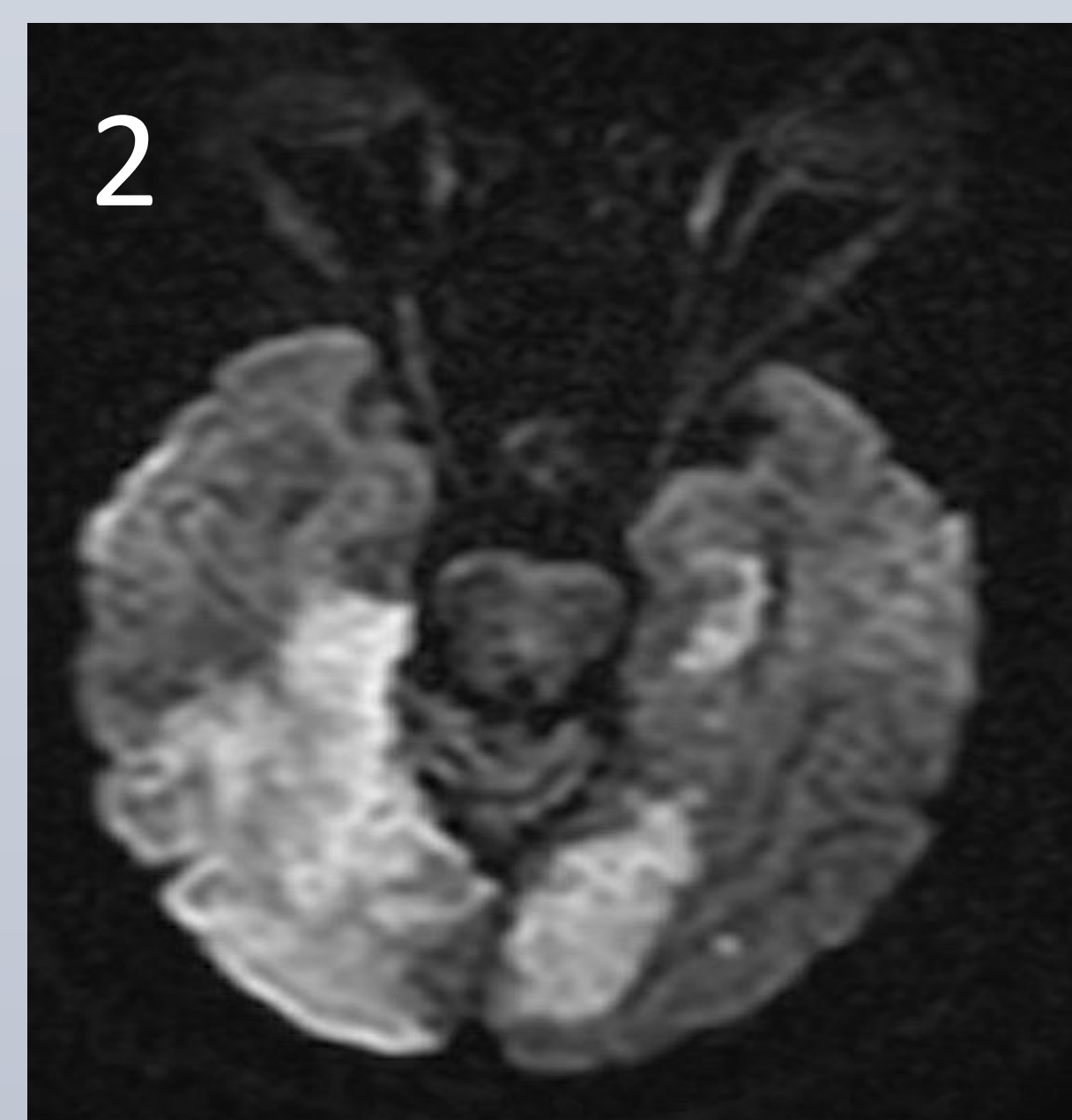
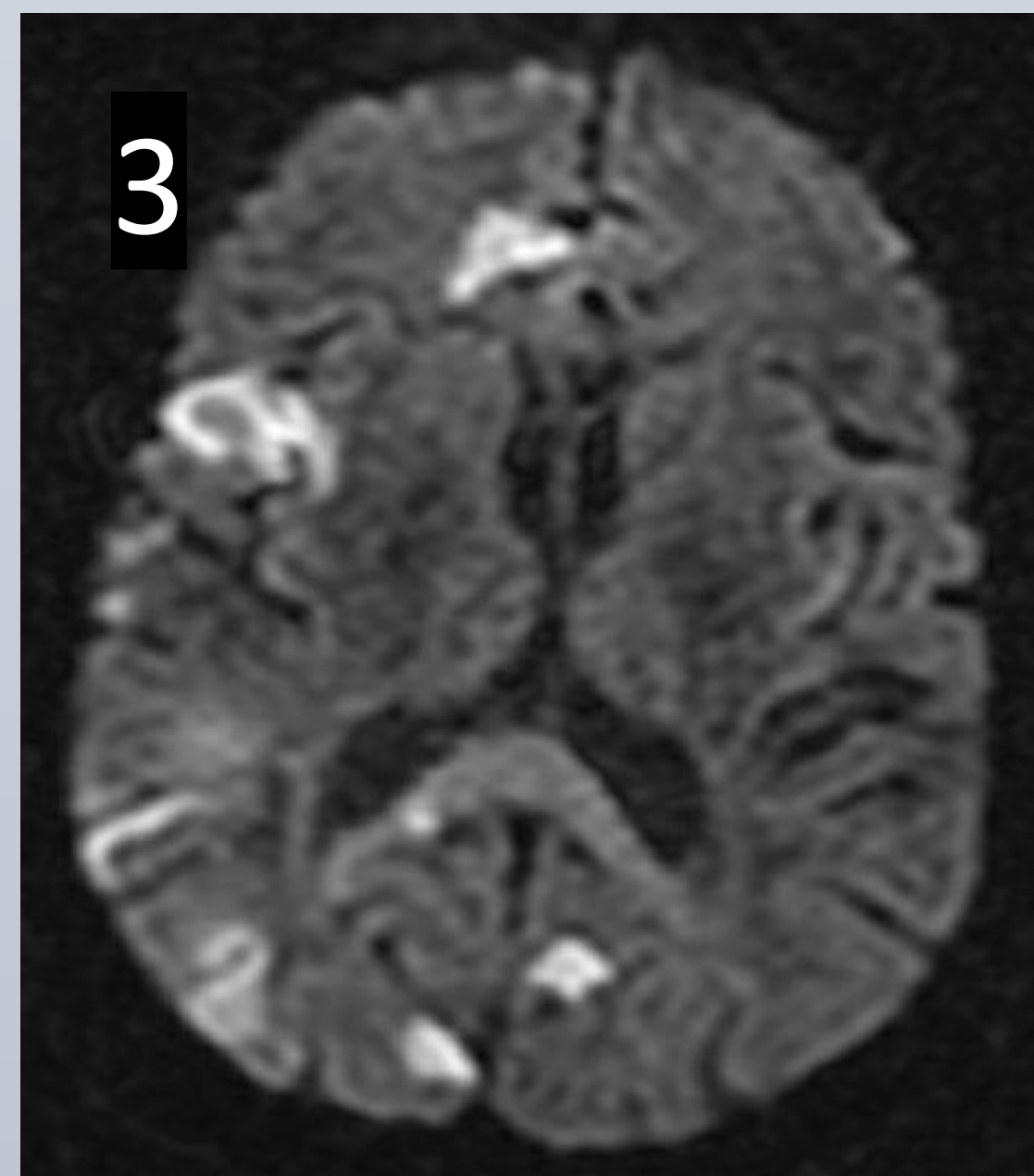


Acquired Coagulopathy in a patient with MDS treated with Lenalidomide

INTRODUCTION

The myelodysplastic syndromes (MDS) comprise a group of hematologic malignancies characterized by clonal hematopoiesis, one or more cytopenias (ie, anemia, neutropenia, and/or thrombocytopenia), and abnormal cellular maturation. MDS shares clinical and pathologic features with acute myeloid leukemia (AML), but MDS has a lower percentage of blasts in peripheral blood and bone marrow (by definition, <20 percent). Patients with MDS are at risk for symptomatic anemia, infection, bleeding, and transformation to AML, the incidence of which varies widely across MDS subtypes. [1] We describe a case of acquired coagulopathy leading to splenic and cerebral venous thromboembolism (VTE) in a patient with a recent use of Lenalidomide as a treatment for MDS.

IMAGING



- Figure 1: CT abdomen pelvis without contrast showing wedge shaped area of hypodensity (the arrow) indicating splenic infarct.
- Figures 2,3: MRI brain with and without contrast consistent with extensive acute/subacute ischemic change in the cerebral hemispheres bilaterally right greater than left. No midline shift, evidence of blood products, or abnormal enhancement was appreciated.

CASE REPORT

- A 69-year-old gentleman with a past medical history of myelodysplastic syndrome (MDS) treated with Lenalidomide 3 weeks ago presented to the emergency department with left-sided abdominal pain gradually worsening over the past 24 hours. He denied any chest pain, shortness of breath, nausea, vomiting or diarrhea. In the emergency department vital signs were stable. Physical examination revealed left upper quadrant abdominal tenderness. Complete blood count significant for thrombocytopenia with platelets of 21,000, hemoglobin of 8.2, hematocrit of 24.7. Complete metabolic profile was unremarkable. Lipid panel was within normal limits, urinalysis was unremarkable. Coagulation profile was within normal limits. Troponin was 0.47. EKG showed normal sinus rhythm with no ST or T wave changes.
- Imaging including CT scan of the head revealed no acute intracranial pathology, CT angiography of the chest no evidence of pulmonary embolism. CT scan of the abdomen and pelvis showed a wedge shaped area of hypodensity consistent with a splenic infarction. Patient was admitted for splenic infarction. General surgery was consulted and recommended conservative management with pain control. In the setting of severe thrombocytopenia and risk of bleeding, we did not start heparin. Troponin was trending up, however patient denied any chest pain, Troponin peaked at 2.49 then trending down.
- On the next day patient denied any chest pain however he became disoriented, vital signs were stable, labs significant for hemoglobin of 7.3, hematocrit of 23.4, platelets of 24,000, complete metabolic profile was unremarkable, repeated brain CT scan showed new hypoattenuating foci in the right frontotemporal lobe and left occipital lobe. Given the distribution, embolic and watershed infarction would be favored. Transesophageal echocardiogram was unremarkable, MRI of the brain showed findings consistent with extensive acute ischemic change cerebral hemisphere bilaterally right greater than left. In the setting of severe thrombocytopenia and stroke we have a discussion with the neurology team, hematology team and cardiology team, the widespread infarction etiology is likely secondary to MDS versus Lenalidomide. We have discussed antiplatelet and anticoagulation in the setting of thrombocytopenia, MDS and risk of bleeding, we came to the conclusion that risk of antiplatelet and anticoagulation outweighed the benefit.

DISCUSSION

MDS patients may present with symptoms associated with cytopenias (i.e. fatigue, bruising, infection): while others remain asymptomatic and seek medical attention due to abnormal routine blood testing.

In our case we describe a patient with known MDS who received lenalidomide therapy 3 weeks prior to hospital admission and presented with severe thrombocytopenia as well as coagulopathy leading to multiorgan VTE.

Patients with MDS may benefit from treatment with immunomodulators such as lenalidomide by reducing transfusion requirements and improving cytologic/cytogenetic abnormalities. Studies have shown an increased risk of VTE with lenalidomide therapy, but the exact risk remains unclear. [2, 3, 4]

Hematologic malignancies and their treatments pose increased VTE risk but bleeding risks are also increased largely due to thrombocytopenia, as exemplified in this report. Many other acquired risk factors exist for VTE as well (i.e. immobility, hospitalization, infection). [5]

VTE risk stratification and prevention strategies remain challenging in patients with MDS given the frequency of associated thrombocytopenia. General VTE prophylaxis guidelines exist but clinical application is limited for MDS patients in the setting of severe thrombocytopenia.

This case highlights the importance of careful consideration for VTE prophylaxis in patients receiving lenalidomide-based therapy and calls for further research on treatment strategies in patients with cytopenias secondary to hematologic malignancies such as MDS who are also faced with increased VTE risk.

CONCLUSION

Lenalidomide treatment in a patient with myelodysplastic syndrome was associated with widespread thrombosis including splenic infarction and stroke.

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