

IMMUNE MEDIATED THROMBOCYTOPENIA

Case Report
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Signalment:

“Sadie”

3 year old Great Dane (light faced)

Female Spayed

History:

Sadie presented to VMSG Emergency for evaluation of progressive epistaxis. The epistaxis started the afternoon of presentation. Sadie was seen initially by her primary veterinarian and was given topical epinephrine and cold packing of the nose was recommended if the bleeding returned. The bleeding transiently resolved following the topical epinephrine. No sneezing or nasal rubbing was noticed by the owner. There was no known rat bait exposure or tick exposure.

Clinical Examination:

Left sided epistaxis and ventral abdominal ecchymoses were noted on examination. The remainder of the examination was normal.

Diagnostic Imaging:

The CBC revealed a marked thrombocytopenia of $<50,000$ plt/uL with a normal hematocrit. The Chemistry panel, PT, and PTT were normal. Three view thoracic radiographs, tick panel, ANA titer, and an abdominal ultrasound were performed to determine an underlying cause for Sadie’s thrombocytopenia and were normal.

Diagnosis:

Primary Immune Mediated Thrombocytopenia

Treatment/Management:

Sadie’s initial therapy included prednisone 1 mg/kg q12h, cyclosporine 5 mg/kg q24h, and omeprazole 1 mg/kg q12h. With Sadie’s severe thrombocytopenia, a second immunosuppressive therapy was added to her therapy, azathioprine 2 mg/kg q24h. Sadie was discharged 2 days after presentation following resolution of the epistaxis, platelet count improvement, and continued improvement of the ventral abdominal ecchymoses.

Prognosis:

ITP prognosis is fair to good with continued resolution of thrombocytopenia on medical therapy.

Discussion:

ITP results from the destruction of platelets from an immune mediated reaction by immunoglobulins, complement, or both. With immune mediated thrombocytopenia, there is an increase in platelet antibodies that bind to platelet membranes. The antibody-platelet cause increased destruction of platelets by the mononuclear phagocytic system within the spleen. Ultimately, thrombocytopenia develops when destruction of platelets exceeds platelet production by megakaryocytes in the bone marrow.

The most common clinical signs for ITP include lethargy, weakness, and fever. Other clinical signs include melena, hematemesis, hematuria, hematochezia, and epistaxis. Petechiation or ecchymotic hemorrhages on the mucous membranes or skin are common physical examination findings. Spontaneous hemorrhage does not typically occur unless platelets are less than 50,000. Causes for ITP include primary (idiopathic) or secondary including infection, parasitic (Rocky Mountain Spotted Fever, *Ehrlichia*, *Babesia*), neoplasia, DIC, recent drug therapy, and recent modified live vaccination administration. The diagnosis of ITP is made by the exclusion of the secondary causes of thrombocytopenia.

Therapy initially includes a glucocorticoid at an immunosuppressive dose of 2-4 mg/kg/day. The combination of a glucocorticoid with an immunosuppressive drug is associated with a longer survival and decreased glucocorticoid side effects. Immunosuppressive therapy of choice include: cyclosporine (Neoral) at 5-10 mg/kg divided q12h orally or azathioprine at 2 mg/kg q24h orally. Patients should be monitored for side effects of Imuran including hepatotoxicity and pancytopenia. Primary ITP signs usually resolve within 2-5 days of initiation of therapy. Refractory primary ITP can be treated with Vincristine 0.02 mg/kg IV every 7 days to increase platelets. Once platelet counts have normalized, decrease the glucocorticoid every 2-3 weeks while monitoring the platelet count and to reach a lowest effective dose. Approximately 70% of primary ITP patients respond to glucocorticoid therapy alone or in combination with immunosuppressive therapy. Initial mortality results are from excessive hemorrhage or euthanasia.

References:

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- 2) Ettinger S.J., Feldman E.C. Textbook of Veterinary Internal Medicine, Sixth Edition. Elsevier Saunders. 2005. P. 1918-1929.
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