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Acute idiopathic thrombocytopenic purpura in an elderly adult: a case study

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Summary The paper is a case study of an 81-year-old female with an acute onset of idiopathic thrombocytopenic purpura (ITP). This diagnosis is rare in adults, and this article discusses the patient's clinical presentation and a review of ITP.

This case is about an 81-year-old female who presented to the office with a five-day history of spontaneous bruising that started on her arms and back and spread to her legs two days before her visit. She denied any trauma or injury, itching, fever, or recent illness. The patient said the only remarkable things in recent history were that she had constipation and used an oral laxative several times within the previous two weeks, and the week before she had respiratory exposure to potent cleaning agents. The day before the bruising began, she ate half of a bag of red licorice and awoke the next morning with red dots she described as tiny bruises on her arms and back, as well as a dark blackish-red spot on her inner left cheek and tongue.

Her past medical history, in addition to asthma and arthritis, included hypertension, hyperlipidemia, gastroesophageal reflux disease, hypothyroidism, and anxiety. Her medications, which were chronic and stable, included atenolol 25 mg daily, fenofibrate 145 mg daily, unithroid 188 mcg daily, esomeprazole 40 mg daily, paroxetine 20 mg daily, temazepam 15 mg at bedtime as needed for insomnia, and propoxyphen with acetaminophen daily as needed for arthritis pain. The patient had no known allergies to medications.

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A review of systems was notable for fatigue, decreased appetite, and dark black stools four two days (but resolved). She had shortness of breath in recent weeks, related to her asthma and not to exposure to the cleaning agents, which improved with correct use of her inhalers. She also had chronic arthritis pain in hands, knees, and lumbar spine, which not changed at the onset of her current complaint. Remaining review of systems was completely normal.

At examination, she was alert and oriented to person, place and time, and in no apparent distress. Her blood pressure was 110/80 mm Hg, pulse 72 beats/min, respirations 12 breaths/min, and she was afebrile. She weighed 174 lbs. Her ears and eyes were normal, but the inside of her mouth was notable for a dark black 3-mm lesion on her left inner buccal mucosa, and the tip of her tongue had two pale 2-mm spots. Her neck was supple without jugular venous distension, bruits, or thyroid masses; her lungs were clear on auscultation; and her heartbeat was regular, with normal heart sounds and a 2/6 systolic murmur at the left sternal border and apex. Her abdomen was soft and nontender, with normal bowel sounds and no masses or organomegaly. Her neurologic, lymphatic, extremity, and musculoskeletal examinations were normal, with the exception of mild arthritic changes of the hands and knees and mild tenderness of the lumbar spine. The patient's skin was remarkable for diffuse nonpalpable petechial lesions on her arms, legs, and trunk.

On the basis of her examination and symptoms, the

initial differential diagnoses included petechiae/purpura rash (with cause to be determined, but medications and food needed to be considered), infection, acute leukemia, and vasculitis. The patient was advised to go to the emergency department for immediate evaluation and possible admission. She did not want to go, but asked if labs could be drawn in the office, and return the next day for follow up. The results were made available the following day when the patient returned to the office. Upon arrival, she said she "felt fine." Complete blood count (CBC) with differential revealed a platelet count of 20,000, but otherwise normal (Table 1). Her blood urea nitrogen/creatinine was 27/1.2, and glomerular filtration fate was 43.1 mL/min. Liver function was normal. Urinalysis revealed orange-colored urine with specific gravity of 1.035, 1+ protein, and 1+ bilirubin; trace leukocyte esterase and ketones were unremarkable. Her lipid levels were elevated (total cholesterol = 210; triglycerides = 237; high-density lipoprotein = 53; low-density lipoprotein = 110). Iron, coagulability (PT/INR), and thyroid studies were normal, and sedimentation rate was normal.

The re-evaluation of the patient did not reveal any major changes. Her blood pressure while seated was elevated at 150/74 mm Hg, and her weight remained at 174 lbs. The examination was unremarkable except for the presence of 4–5 total petechial lesions on her hard palate and inner buccal mucosa. She also had diffuse, worsening petechiae on her arms, legs, and trunk, and ecchymoses on her left upper extremity. There was no evidence of bleeding from her gums or rectum and no complaints of vaginal bleeding. She was neurologically intact and the remainder of her examination was unremarkable. It was again recommended that she go to the emergency department or seek a hematology consult, but she declined again and asked to be treated as an outpatient only.

On the basis of her history, examination, and lab findings, several conditions (e.g., anemia, vasculitis, leukemia, infection) were ruled out. It was determined that her most likely diagnosis was acute idiopathic thrombocytopenic purpura (ITP).

Acute idiopathic thrombocytopenic purpura (ITP) is an immune-mediated disorder that involves the premature destruction of platelets by the reticuloendothelial system. In this disorder, antibodies develop against platelet membrane glycoprotein (Gp IIb/IIIa or Gp Ib/IX) complexes, which are the target antigens. The opsonized platelets become susceptible to phagocytosis, which allows for their destruction.[4,5] In adults, ITP is rare, with an incidence of approximately 66 cases per 1 million people per year.[1] The incidence is usually a ratio of 1.7 females to 1 male, and the peak incidence in people older than 60 years is more than twice that of people younger than 60 years. Only 2% of adults have a spontaneous remission, 43% will have chronic disease, and 64% will have complete recovery.[3] Common physical findings include nonpalpable petechiae, purpura, ecchymoses, gingival bleeding, retinal hemorrhages, signs of gastrointestinal bleeding, hemorrhagic bullae on mucous membranes, spontaneous bleeding (if platelet count $<20{,}000/mm^3$), and evidence of intracranial hemorrhage with possible neurologic symptoms.[1,2] It is unlikely for a patient to have splenomegaly and lymphadenopathy, and if present, other diagnoses should be considered.[5]

Patients may not always present with any symptoms and can be diagnosed incidentally after routine labs.[3] Laboratory findings usually reveal isolated thrombocytopenia without changes to the rest of the CBC. The platelets may appear abnormally large (megathrombocytes) on a peripheral blood

Table 1 Initial laboratory values

CBC with Differential	Result	Reference Interval
WBC	$10.1 \times 10^3/\mu L$	4.0–10.5
RBC	$4.00 \times 10^3/\mu L$	3.80–5.10
Hemoglobin	12.4 g/dL	11.5–15.0
Hematocrit	36.2%	34.0–44.0
MCV	91 fL	80–98
MCH	30.9 pg	27.0–34.0
Platelets	$20 \times 10^3/\mu L$	140–415
Neutrophils (Absolute)	$6.0 \times 10^3/\mu L$	1.8–7.8
Lymphocytes (Absolute)	$3.2 \times 10^3/\mu L$	0.7–4.5
Monocytes (Absolute)	$0.7 \times 10^3/\mu L$	0.1–1.0
Complete Metabolic Panel		
Glucose, serum	90 mg/dL	65–99
BUN	27 mg/dL	5–26
Creatinine, serum	1.2 mg/dL	0.5–1.5
GFR, estimated	43.1 mL/min	60.0–128.0
Sodium, serum	142 mmol/L	135–148
Potassium, serum	3.9 mmol/L	3.5–5.5
Chloride, serum	102 mmol/L	96–109
Carbon Dioxide	28 mmol/L	20–32
Calcium, serum	9.4 mg/dL	8.5–10.6
Albumin, serum	4.1 g/dL	3.5–4.7
Bilirubin, Total	0.4 mg/dL	0.1–1.2
AST	29 IU/L	0–40
ALT	20 IU/L	0–40
Lipid Panel		
Total Cholesterol	210 mg/dL	100–199
Triglycerides	237 mg/dL	0–149
HDL	53 mg/dL	40–59
LDL	110 mg/dL	0–99
Iron and TIBC		
Iron, serum	97 mcg/dL	35–155
TIBC	449 mcg/dL	250–450
Iron Saturation	22%	25–55%
Ferritin	33 ng/mL	10–291
PT and PTT		
INR	1.1	2.0–3.5
Prothrombin Time	11.0 sec	8.7–11.5
APTT	28 sec	22–36
TSH	5.025 uIU/mL	0.350–5.500
Sedimentation Rate - Westergren	17 mm/hr	0–30

Laboratory results and reference intervals obtained from Laboratory Corporation of America, where patient's labs were sent. On CBC differential - few large platelets observed, fibrin strands present.

smear.[5] Coagulation studies are normal. Imaging studies could consist of a computed tomography scan of the head if there is suspicion of intracranial hemorrhage. Diagnosis of ITP is made after excluding other likely causes, such as collagen vascular diseases, therapy with certain drugs, von Willebrand disease type IIB, and HIV infection.[3] In addition, other conditions such as aplastic anemia, acute leukemia, metastatic tumor, viral hepatitis, infectious mononucleosis, toxoplasmosis, and cytomegalovirus need to be considered in the differential diagnosis.[4]

Complications of ITP include severe blood loss, intracranial and other major hemorrhages, side effects from corticosteroids, and pneumococcal infections in patients who have had splenectomy.[1]

Treatment is dependent on symptoms, the patient's condition, and the absolute platelet count. Adults with platelet counts greater than 50,000/mm^3 usually do not require treatment. Those with platelets less than 50,000/mm^3, with significant mucous membrane bleeding, or those with platelets less than 20,000–30,000/mm^3 and risk factors for bleeding should be treated. The goals of therapy are to increase platelet counts to greater than 150,000/mm^3 to demonstrate complete response.[2]

The most common initial therapy is glucocorticoids, which have been shown to increase the platelet count in ITP by inhibiting phagocytosis of antibody-coated platelets and inhibiting autoantibody production, as well as strengthening capillaries. Prednisone at 1–2 mg/kg/day orally, may be used effectively, usually for two weeks to six months. Alternatively, high-dose dexamethasone at 40 mg/day for four days every four weeks may be used.[2] A complete or partial response with steroids may occur in almost two-thirds of patients, often in the first week of treatment.[3]

If severe, life-threatening bleeding occurs, or if a patient is unresponsive to or contraindicated to the use of corticosteroids, intravenous immune globulin (IVIg), 1–2 g/kg intravenously administered over 1–5 days, may be used to rapidly increase the platelet count.[1,2] IVIg may elevate the platelet count in 85% of patients, with 65% of patients developing a normal platelet count.[3] Anti-Rh(D), a plasma-derived immune globulin that inhibits reticuloendothelial phagocytosis of immunoglobulin G–coated platelets, may be used to increase platelets as an alternative to steroids or IVIg, especially in immunocompromised patients who have their spleens and are Rh(D)-positive.[2] The dose is 50 mg/kg per day intravenously and may be 68% effective in increasing platelet levels and delaying or avoiding splenectomy.[3]

Although the scope of this paper focuses on acute ITP, if chronic ITP occurs, thrombopoietic agents, such as eltrombopag and romiplostim, may be used to stimulate bone marrow platelet production, or if warranted, splenectomy may be done.[1] Splenectomy can provide a complete remission in 75% of patients; however, the decision to perform this procedure is individualized and based on the age of the patient, comorbid conditions, duration of the disease (usually after at least 4–6 weeks from diagnosis), and what other treatments (considering efficacy and adverse reactions) have been used.[3] If any of the above treatments fail, other effective treatments for chronic ITP may be used. This includes monoclonal antibodies such as rituximab, alemtuzumab, and danazol; chemotherapy with azathioprine, vinka alkaloids, cyclosporine, or cyclophosphamide; plasma exchange; interferon; colchicines; and dapsone.[2]

For this case, the patient was diagnosed and treated for acute idiopathic purpura. She had no history of anemia or thrombocytopenia. She had been relatively healthy before the sudden onset of symptoms. Her history in the day to weeks before her onset of symptoms provided no clear causation. Her use of oral laxatives could have caused hypokalemia and dehydration, as well as diarrhea, but none of these occurred. The patient added no other medications, and her regular medications were taken as directed. There was not enough exposure to chemical cleaning agents to affect her health, nor have cleaning agents been linked to ITP. Excessive intake of licorice may have contributed to lowering serum potassium and hematocrit, but both levels in the patient were normal. Symptoms of licorice toxicity may consist of fatigue, muscle cramping, weakness, edema, and dyspnea.[6] Other than fatigue, as well as dyspnea likely due to her asthma, the patient exhibited no other symptoms seen with licorice toxicity. With other possible causes for the patient's symptoms and examination and laboratory findings ruled out, it was determined that her most likely diagnosis was ITP.

After discussing with the patient the benefits and risks of outpatient treatment without further workup, she agreed to a relatively conservative approach. She was then begun on treatment with prednisone at 1 mg/kg/day, and was advised to avoid licorice, nonsteroidal anti-inflammatory drugs, and laxatives. The patient's treatment with prednisone began with 80 mg daily for three days, and then it was tapered to 60 mg, 40 mg, 20 mg, and 10 mg, with each dose taken daily for three days and then stepped down. After the last dose, the medicine was discontinued. A complete blood count (CBC) drawn one week after treatment initiation was significant for hemoglobin 11.8, hematocrit 35.9, white blood cell count 19, and platelets corrected to 248,000. The patient said she felt much better and had no further bruising or descriptions of any petechial or purpural rash. Her examination was consistent with her subjective response. There was fading pink bruises and petechiae on her legs, but the examination was otherwise normal. Weekly CBC × four weeks, then monthly CBC × two months was ordered to monitor platelets, white blood cell count, and hemoglobin and hematocrit. Clinically, she improved and labs corrected after prednisone use and remained normal, and the patient had full resolution of her rash and symptoms, by five weeks after initial presentation.

In summary, the patient presented as a rare case of an elderly adult female who developed acute onset of Idiopathic Thrombocytopenic Purpura, with eventual full remission of her symptoms and laboratory findings. She remained clinically stable and healthy, with favorable prognosis even months after treatment with prednisone was completed.

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1.b, 2.c, 3.b, 4.d, 5.a, 6.a, 7.b, 8.c, 9.d, 10.a