Psychogenic Purpura (Gardner-Diamond Syndrome)

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ABSTRACT

Psychogenic purpura, also known as Gardner-Diamond syndrome or autoerythrocyte sensitization syndrome, is a rare condition characterized by spontaneous development of painful edematous skin lesions progressing to ecchymosis over the next 24 hours. Severe stress and emotional trauma always precede the skin lesions. The condition is most commonly seen in women, but isolated cases have been reported in adolescents and in males. Psychodermatologic evaluation and dermatology and psychiatry liaison have been successful in the treatment of these patients. This report provides an overview of psychogenic purpura and presents the case of a 15-year-old girl.

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Psychogenic purpura, also known as Gardner-Diamond syndrome or autoerythrocyte sensitization syndrome, is an extremely rare condition typically noted in women with psychiatric comorbidity. Psychogenic purpura is regarded primarily as an autoimmune vasculopathy with sensitization to phosphatidylserine, a component of erythrocyte stroma.[1] Manifestation usually occurs via spontaneous development of painful edematous skin lesions, predominantly in the extremities, which may be isolated or multiple, progressing to ecchymosis over the next 24 hours. Development generally follows severe stress or emotional trauma or is comorbid with certain psychiatric psychopathology. Numerous somatic complaints may be explored in a diagnostic setting without objective findings and with potential history of multiple surgical procedures. While frequency is highest in women, cases in men and adolescents have also been described.[2–5]

HISTORY

The history of psychogenic purpura dates back to the early 20th century. In 1927, the German psychiatrist Rudolf Schindler described 16 patients with skin hemorrhages.[6] He suggested a possible link to hypnosis, corroborated in a few subsequent case reports, in causing or relieving these lesions.[7–9] Shortly thereafter in 1928, 2 other cases were described with associated hysterical personality and delusions, respectively, thus connecting the skin lesions with psychopathology.[10] In the landmark publication in 1955, Gardner and Diamond[11] described in detail the cases of 4 women manifesting abnormal responses to bruises. These lesions were characterized by development of areas of painful ecchymoses at the site of trauma followed by progressive erythema and edema. It was suggested for the first time that the disorder is associated with autosensitization of patients to their own blood. The data were then systemized and formally presented as "Gardner-Diamond syndrome." Since then, about 200 cases of the syndrome have been reported in the literature.[12] The term *psychogenic purpura* was coined by Oscar D. Ratnoff in 1989, who reported a series of 71 cases of autoerythrocyte sensitization syndrome.[13]

ETIOLOGY AND PATHOGENESIS

As mentioned earlier, Gardner and Diamond[11] originally attributed the pathophysiology to autosensitization of erythrocytes and concluded that the causative agent, while not in the blood plasma or associated with hemoglobin, did in fact reside in the erythrocyte stroma. Confirmation was later provided by Groch et al[1] that autosensitization to phosphatidylserine (phosphoglyceride of red blood cell membrane) is of pivotal importance in the pathogenesis of this syndrome. Indirect immunofluorescence studies[14,15] suggested that 50% of erythrocyte phosphatidylserine of patients with Gardner-Diamond syndrome was found on the outer surface of the cell membrane. This finding was reproduced in an experiment after incubation of homologous erythrocytes from a healthy donor with an affected individual's plasma, which was found to contain specific IgE antibodies to cardiolipin and phosphatidylserine.[16] Some authors have suggested associations with a variety of hematologic conditions including thrombocytosis, defective thrombocyte aggregation, increases in activated partial thromboplastin time, idiopathic thrombocytopenic purpura, and circulating fibrinolytic factors.[17–20] Other mechanisms that have been

Clinical Points

- Psychogenic purpura could produce a diagnostic dilemma and thus requires thorough investigation.
- Proper diagnosis of psychogenic purpura could save many unnecessary procedures.
- The role of stress in psychogenic purpura should not be ignored.

suggested include disturbances in tone regulation of venous capillaries due to fluctuation in the kallikrein-kinin system and a disturbance of fibrin synthesis in the endothelium and formation of defective structures of capillary walls.[21]

It may be noted that abnormalities in the coagulation system or blood vessel development are usually not involved with pathogenesis of Gardner-Diamond syndrome, although many of the earlier case reports on the syndrome fail to report exhaustive screening for von Willebrand disease or platelet dysfunction disorders. However, some authors have suggested abnormal platelet responses during platelet aggregation studies.[22] Besides that, a number of immunologic abnormalities have also been reported in patients with Gardner-Diamond syndrome including positive anticardiolipin antibodies,[23] decreased serum complement levels during ecchymoses formation along with their return to normal when lesions heal, angioimmunoblastic lymphadenopathy, vasculitis, lymphoid interstitial pneumonia, and association with multiple glomus tumors.[24–27] Further evidence of the potentially immunologic nature of Gardner-Diamond syndrome is provided by the successful treatment via plasmapheresis.[28] Autoerythrocyte sensitization syndrome may also present as a primary manifestation of systemic lupus erythematosus.[29] Exposure to copper, such as via a copper-containing intrauterine device and direct contact of copper coins to the skin, have also been proposed as possible etiologies.[30]

CLINICAL FEATURES

Initiation of skin lesions generally follows physical trauma or surgery,[31] but can be atraumatic in nature,[32–35] with the common theme being severe emotional disturbance. The prodromal phase always comprises malaise and fatigue. Skin lesions are at first preceded by burning and itching and a stinging sensation or pain, followed by cutaneous induration a few hours later. Subsequently, painful edematous pink or red plaques of variable sizes become visible. The severity of pain and edema can lead to acute immobilization of the afflicted extremity and can be mistaken for acute compartment syndrome[36] or cellulitis.[37] Following a period of up to 48 hours, lesions may evolve into bluish ecchymoses with a yellowish hue. Lesions may become less tender as they regress and eventually disappear completely within approximately a week. While the extremities and trunk are common sites, lesions can occur anywhere on the body.

Associated symptoms may include fever, arthralgia, myalgia, headache, and dizziness. A significant number of patients report different gastrointestinal symptoms such as epigastric pain, hemorrhages, nausea, vomiting, and diarrhea.[27,38] Hematuria, menorrhagia,[31] vaginismus[39] and hemorrhagic blood loss via the ear canals, epistaxis, and subconjunctival hemorrhage,[41,41] as well as gastrointestinal bleeding have also been described in association with Gardner-Diamond syndrome.[31,42] Conversion symptoms, hysterical traits, and dissociative reactions may accompany such hemorrhagic symptoms.[40–43] Exceedingly rare associations include stroke,[11] lymphoid abnormalities,[24,26] glomerulonephritis,[44] and pseudoainhum.[45] Ecchymosis related to copper-containing intrauterine devices appears shortly after insertion and disappears following removal of the device or replacement with noncopper alternatives.[30]

CASE REPORT

Ms A, a 15-year-old white girl, presented with a 1-year history of recurrent bilateral upper extremity and torso bruising. Numerous prior dermatology and internist consults had failed to establish an etiology for these recurrent lesions. Associated symptoms included general malaise, headache, and joint pain. Lesions ranged from skin indurations with a burning and stinging quality progressing to painful edematous plaques, followed by ecchymosis formation, and, finally, disappearance over the course of a few days. No personal or family history of bleeding disorders or use of aspirin or any nonsteroidal anti-inflammatory drugs were reported. A battery of laboratory tests including complete blood count, coagulation profile including bleeding time, prothrombin time, activated partial thromboplastin time, factor VIII, von Willebrand antigen, antinuclear factor, anti-dsDNA antibody, anticardiolipin antibody, lupus anticoagulant, cryoglobulins, and thyroid profile were deemed insufficient in corroborating a diagnosis. At the clinic, detailed history taking correlated temporal changes in mood and functioning and, ultimately, initiation of the lesions with ongoing parental divorce proceedings. A psychiatric interview and the Beck Depression Inventory[46] were then used to confirm a diagnosis of major depressive disorder (*DSM-5* criteria). Coinciding changes included low self-esteem, academic decline, poor interpersonal relationships, and heightened feelings of stress over the past year.

Ms A's own washed red blood cells were then injected intradermally in her right forearm, with the left forearm serving as control with parallel normal saline administration. Tingling and a burning sensation at the injection site on the right forearm were elicited after 3 hours. A new-onset faint bruise was observed, eventually abating over the course of 24 hours. No reaction was observed in the left control arm, thus establishing a Gardner-Diamond syndrome diagnosis. Treatment methods used included intensive supportive therapy along with a daily prescription of escitalopram 10 mg to address underlying symptoms of depression. Follow-up after 3 and 6 months showed complete resolution of symptoms and improvement in mood symptoms.

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DIFFERENTIAL DIAGNOSIS

Patients presenting with a constellation of symptoms resembling psychogenic purpura must have a thorough history recorded as would be done for any other malady. Relevant information regarding bleeding history, family history of bleeding diathesis, onset and recurrence of symptoms, occupational history, and major emotional or stress-inducing events such as physical trauma, abortion, divorce, death of a loved one, unemployment, and even religious pilgrimage can help prevent patient exposure to excessive tests or mutilating surgery. Gardner-Diamond syndrome may be confused with several disorders with dermatologic manifestations including disseminated intravascular coagulation, idiopathic thrombocytopenic purpura, Henoch-Schonlein purpura, von Willebrand disease, Ehlers-Danlos syndrome, dermatitis artifacta, systemic lupus erythematosus, cellulitis, compartment syndrome, factor XIII deficiency, Weber-Christian panniculitis, and polyarteritis nodosa, as well as psychiatric conditions such as Munchausen's syndrome[44] and even malingering. Care must be taken to note a thorough history of medication type, dosage, route of administration, and compliance to rule out a possible bleeding disorder from illicit, nontherapeutic drugs or overdose of medications such as aspirin, nonsteroidal anti-inflammatories, selective serotonin reuptake inhibitors, heparin, or warfarin and steroid-induced purpura.

A tactful history taking and vigilant physical examination can detect old scars, venipuncture sites, and nasopharyngeal necrosis to increase suspicion of use of illicit drugs such as cocaine[48]; 3,4-methylenedioxymethamphetamine (ecstasy); phencyclidine (PCP); khat (*Catha Edulis*) or synthetic cathinones; *Salvia divinorum*; and synthetic cannabinoids and even prescription drug insufflation in patients presenting with abnormal psychiatric features, which require gas chromatography-mass spectrometry analysis and may not be detectable via standard drugs-of-abuse immunoassays.[49] Pediatric cases deserve heightened attention to rule out child abuse, as mere implication can be a traumatic experience for both the patient and family.[50,51] Pediatric cases of Gardner-Diamond syndrome have been associated with or mistaken for a wide variety of etiologies ranging from intramuscular stimulation[52] to conversion anxiety.[41] Clinical diagnosis is made difficult owing to the obscure nature of the disease, absence of standard laboratory markers, and the unreliable nature of many self-described patient histories. Confirmation can therefore involve histories taken from numerous sources including family members, friends, and colleagues, as well as consults involving psychiatry, dermatology, immunology, and hematology among others.

PROGNOSIS

Overall prognosis is quite good. However, in many patients, relapses and remissions[53] are common occurrences and may last for several years. Chronological recurrence around the anniversary mark of major life events is common.[33,40,54] The severity of lesions may considerably fluctuate as well.[40,55] Relief of acute stress and psychological factors is crucial in longer remissions.

HISTOPATHOLOGY

Biopsy has never been conclusive and is considered an unreliable method for the diagnosis of psychogenic purpura. However, biopsies have revealed extravascular erythrocytes in the dermis, edema, and nonspecific lymphohistiocytic infiltration around the blood vessels. Macrophages may also show pigment deposition, predominantly hemosiderin.

LABORATORY EXAMINATION AND DIAGNOSIS

There are no specific laboratory changes observed in Gardner-Diamond syndrome.[56] All hematologic parameters such as hemoglobin, hematocrit, platelets, peripheral smear, erythrocyte sedimentation rate, electrolytes, prothrombin, thrombin, partial thromboplastin time, bleeding time, and coagulation factors are within normal limits. Commercial platelet function assay systems may be used on citrated whole blood samples and are more rapid, sensitive, and specific for platelet abnormalities than the fickle bleeding time test. The most reliable diagnostic test is, in fact, intradermal injection of the patient's own washed erythrocytes or washed autologous lymphocyte or heterologous and autologous DNA.[57] Early methods consisted of assurance to the patient of the potential sensitivity to autologous blood. Three test sites were selected, and only 1 revealed as the patient's own blood, with the other 2 obtained from cross-matched donor blood or saline, even as all 3 aliquots were in fact drawn from the patient. A positive reaction in the form of painful ecchymosis at the revealed site was deemed confirmatory.[58–60] Currently, besides the standard use of intracutaneous 80% suspension washed autoerythrocyte, a variety of modifications include use of homologous donor erythrocytes[61] and autologous leukocytes,[62] as well as autologous[60–63] and heterologous[57] DNA.

Besides intradermal red cell stroma and phosphatidylserine injections, case reports also exist of lesions developing via histamine, histidine, serotonin, old tuberculin, tyramine, platelets, serum, and pregnanetriol, a cortisol precursor.[28] Several authors have levied the autoerythrocyte sensitization hypothesis unviable due to the its low sensitivity—some patients often show inconsistent inflammation to injected RBCs at test sites and even withstand surgery without massive immune reactions.[58,67] Cytotoxic T-cells against autologous erythrocytes or autoantibodies also cannot be detected. While Gardner and Diamond obtained a positive result in all 4 of their patients, Ratnoff[13] called the response "erratic," noting positive findings in only 59% of clinical patients. This finding suggests that a negative test result may, in fact, be unable to rule out a diagnosis.

PSYCHOLOGICAL DISTURBANCES

Association of psychological factors with Gardner-Diamond syndrome is the mainstay for this diagnosis,

and their presence is one of the essential diagnostic components of the syndrome. Nonetheless, there remains marked heterogeneity in the spectrum of psychological findings, and the exact role of such factors in the etiology of psychogenic purpura remains inconclusive. Various associated psychopathologies include depression, anxiety, difficulty in addressing aggression and hostility, emotional instability, adjustment issues, hypochondriasis, abnormal guilt, sexual problems, masochism, and histrionic and borderline personalities, as well as conversion and obsessive disorders.[31,68] How such stress would alter immune reactivity to the point wherein a patient develops pathophysiologic processes leading to lesion formation remains unclear. The stress response hypothesis proposes a psychoendocrine model in which neuropeptide signaling molecules expressed and released by neurons act upon cell surface receptors, ultimately leading to an alteration of hemostatic physiologic processes via catecholamine and glucocorticoid release.[69,70] One such hemostatic equilibrium alteration may be elevated site-specific tPA activity leading to increased local plasmin activity causing fibrin clot degradation and, eventually, bleeding.[71–73]

However, some studies have suggested no apparent predisposing emotional distress, mental illnesses, or traumatic events.[15,23,68] Supporting the true conversion reaction hypothesis is all the more difficult due to the problematic nature of proving a conversion reaction while completely disproving factitial causation. Some distinguishing criteria include a negative red blood cell skin test in an unreachable area, the absence of new ecchymosis under casts, and the regular formation of new lesions at cast sites upon removal in the case of factitial diagnosis.[58]

A careful psychiatric history is often missed and not elicited until all medical tests and examinations and treatments have been exhausted; this becomes the source of frustration for the treating provider, patient, and family alike. Exhaustive testing for skin bleeding episodes beyond prothrombin time, activated partial thromboplastin time, and platelet count and ruling out von Willebrand disease and antiphospholid syndrome is not recommended, especially when laboratory values fall within the normal range and there is no past family history of a bleeding diathesis—this is primarily due to prohibitive costs, time constraints, and elevated patient discomfort. Revealing the psychological nature of the disease can also further complicate the scenario and may increase the level of patient frustration and the family's denial. Ensuing communication concerns can severely affect physician-patient rapport.

In numerous cases, it is very difficult for the patient and the family to accept the psychological nature of the disorder. Under such circumstances, it is advisable to avoid the terms psychogenic purpura due to stigmatization or *autoerythrocyte sensitization syndrome* suggesting an organic disorder.[71]

TREATMENT

There is no specific treatment for Gardner-Diamond syndrome. Symptomatic therapy is provided for severe general symptoms. Several approaches including antihistamines, corticosteroids, antidepressants, hormones, and vitamin use have met with variable success. Medications such as busulfan,[18] promethazine,[5] and numerous others including antidepressants have been used to treat Gardner-Diamond syndrome and alleviate comorbid psychiatric pathology such as depression or mood symptoms.[75] Some authors have suggested the role of medications affecting capillary tone or permeability of vessels in the treatment of Gardner-Diamond syndrome.[21] Extrapolating from the psychological nature of the disease, placebo effect has been used successfully to alter the severity of symptoms.[43] Psychotherapy has also been used in several cases with positive results in reducing stress associated with productions of lesions.[41,76,77] Psychiatric treatment in young age groups such as children and adolescents, as well as a relatively short period of affliction provide the most favorable outcome for rehabilitation.[78]

CONCLUSION

Psychogenic purpura (Gardner-Diamond syndrome) is a rare condition that should be considered in the differential diagnoses of conditions in which unexplained echymosis or purpuric lesions are noticed and no apparent identifiable cause is known. The role of psychological association should always be considered in these cases. Complete psychodermatologic evaluation and the liaison between dermatology, primary care, and psychiatry are of vital importance for the management of these conditions. The role of primary care physicians should not be underestimated in treating these patients.

Drug names: escitalopram (Lexapro and others), warfarin (Coumadin, Jantoven, and others).

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