Vancomycin-induced linear Immunoglobulin A bullous dermatosis

Htay Phyu, Takaaki Kobayashi, Prerna Rastogi, Christine Cho

DESCRIPTION
A 72-year-old woman with a history of left hip prosthetic joint infection (PJI) on parenteral antibiotics presented with rash. Prior to this presentation, she was diagnosed with PJI of her left hip, for which she underwent hardware explant. Intraoperative cultures were negative, and she was discharged on an empiric 6-week course of vancomycin and ceftriaxone. Nine days into therapy, she developed pruritic and painful rash on her right arm, which progressed to the rest of her body. On admission, vital signs were unremarkable and physical examination showed symmetrical erythematous plaques on the bilateral arms, axillae, abdomen, groins, thighs and back with overlying pustules and bullae (figure 1A,B). Skin biopsy revealed a subepidermal split with numerous underlying neutrophils and eosinophils and underlying perivascular mixed inflammation in the superficial dermis (figure 2A). Direct immunofluorescence microscopy demonstrated linear marking for IgA and C3 along the dermal–epidermal junction (figure 2B). No IgM or IgG was identified. The patient was diagnosed with vancomycin-induced linear IgA bullous dermatitis. Vancomycin was discontinued and topical steroid was started for her symptomatic rash. About 9 days after discontinuation of vancomycin, her rash improved significantly. She completed the remainder of the 6-week course of antibiotics for PJI with daptomycin and ceftriaxone without complication.

Linear IgA bullous dermatosis (LABD), also known as linear IgA disease, is a rare autoimmune blistering disease. LABD is characterised by the linear deposition of IgA at the dermoepidermal junction. Reports of disease incidence range from less than 0.5 to 2.3 cases per million individuals per year. Aetiology of LABD can be drug-induced; autoimmune, such as ulcerative colitis; infectious (upper respiratory tract, gynaecological infections, typhoid, brucellosis, varicella zoster and tetanus); malignant; or idiopathic. Drug-induced LABD cases represent approximately 37.5% of all LABD in adults. Vancomycin is the most frequently reported pharmacological agent as a potential inciting factor. New lesions can develop from within 24 hours up to 15 days after the last dose of vancomycin. Other drugs that are associated with LABD include penicillin, amoxicillin, ampicillin–sulbactam, piperacillin–tazobactam, ceftriaxone, moxifloxacin, trimethoprim–sulfamethoxazole, metronidazole, rifampin, nonsteroidal anti-inflammatory agents, lithium, captoril, amiodarone, phenytoin, cyclosporine, furosemide, interferon alfa and somatostatin. Lesions vary, including tense serous/hemorrhagic bulla (blister), string of pearl ‘herpetiform’ configurations or targetoid/erythema multiforme-like eruptions. Vancomycin-induced LABD commonly involves extremities, palms and soles. Macular involvement is known to be relatively rare. Direct immunofluorescence is generally needed to confirm the diagnosis of LABD and to help exclude other conditions. Linear IgA deposition at the dermal–epidermal junction of the basement membrane zone is pathognomonic of LABD. Drug-induced LABD typically resolves with withdrawal of the offending agent. In severe or persistent cases, treatments including dapsone, sulfonamides, colchicine, topical and oral corticosteroids or intravenous immunoglobulins may be required. Early diagnosis is important for its management, because discontinuing the causative drug is most crucial.

Learning points
► Linear IgA bullous dermatosis (LABD) is an autoimmune vesiculobullous disease and can be caused by medications, autoimmune disorder, infections or malignancy.
► Vancomycin is the most common drug associated with LABD.
► Early diagnosis is important for its management, because discontinuing the causative drug is most crucial.
immunoglobulins have been effective, although limited data are available.

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ORCID ID

Takaaki Kobayashi http://orcid.org/0000-0003-4643-4798

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