Drug- induced lupus erythematosus case report

Drug-induced lupus (DIL) is an autoimmune phenomenon in which drug exposure leads to the development of systemic lupus erythematosus (SLE) such as clinical characteristics. DIL is a clear example of an environmental trigger that leads to the development of lupus in a genetically vulnerable individual. Hydralazine was the first active ingredient associated with the development of lupus-like symptoms in 1954[1]. Since then, more than 100 drugs have been identified as the cause of drug-induced lupus, with the list expanding each year with the development of newer biological agents. DIL tends to be less severe than SLE, with symptoms usually resolved after dissolution of the offensive agent. Hundreds of drugs have been reported to cause DIL. [2] [3] While some drugs have good evidence of a link with DIL, there are case reports that imply several other drugs as a possible cause of DIL. Several herbal medications have also been reported to cause a lupus-like syndrome. Clinical and immunological characteristics may vary for any agent responsible for the development of DIL. Procainamide and hydralazine have the highest incidence of DIL, with procainamide risks of up to 30% and 5% to 10% in hydralazine. All anti-TNF agents have been associated with DIL, with a higher risk of etanercept and infliximab. [5] [5] Other drugs with clear association with DIL include interferon-alpha, minocycline, isoniazid, rifampin, phenytoin, penicillamin, quinidine, phenytoin, methyldopa, chlorpromazine, carbamazepine, ethosuximide, propylthiouracil and sulfasalazine. Several other drugs are thought to be reported to possible causes of DIL with case reports, including statins, ace inhibitors, proton pump inhibitors, gold salts, non-steroidal anti-inflammatory agents (NSAIDs), oral contraceptives, etc. Herbal medicines such as alfalfa sprouts, echinacea, and melatonin were also dropped more lupus torches. Drug-induced lupus accounts for 6% to 12% of all lupus cases, with an annual incidence of 15,000 to 30,000 new cases per year in the United States. [7] The epidemiology of DIL reflects the population taking the drug responsible for DIL. Minocycline-induced lupus is more common in younger women, while procainamide or hydralazine induced lupus is more common in the older population. The pathophysiology of DIL is unclear and various mechanisms are responsible for the induction of autoimmunity by various lupus-inducing drugs. There are some genetic risk factors such as HLA-DR4, HLA-DR0301 and complement C4 zero alleles that vary between different active ingredients. Slow acetylators with genetic deficiency of N-acetyltransferase have a higher dil risk, especially of procainamide Hydralazine. Inhibition of DNA methylation is thought to contribute to the development of DIL from many active ingredients including procainamide and hydralazine. Demethylation of CD4+ T cells makes them authoreactively authoreactive by overexpression of the LFA-1 Molecule. These autoreactive T cells can then overstimulate autoantibody production by interacting with self-class II MHC molecules on B cells and inducing apoptosis of macrophages by interacting with self-class II MHC molecules on macrophages that release the highly antigenic apoptotic chromatin from the dying macrophages. This autoantibody production and release of the antigenmacrophage chromatin is said to contribute to the development of lupus-like autoimmunity. Skin rash is one of the most common clinical presentations of drug-induced lupus. The pathological examination of the dermatology from the rash in patients with DIL is similar to that in patients with SLE. Subacute skin-close lupus erythematosus (SCLE) is one of the most common rashes that can detect vacuolar boundary dermatitis with lymphotoytic infiltration of the upper dermis in a perivascular pattern, epidermal atrophy, dead keratinocytes and dermo-epidermal degeneration. Drug-induced lupus can develop a few weeks to several months after the drug is started, which can make diagnosis difficult. In addition, it is not possible to distinguish DIL from SLE solely on the basis of clinical characteristics, although DIL tends to be milder and renal or CNS involvement, vasculitis, leukopenia and pericarditis are rare. Arthralgia is common and often the first symptom and is present in up to 90% of patients. Constitutional symptoms such as myalgia, fever and weight loss are also common. The Kutane involvement is common and may include light sensitivity, purpura, erythema nodosum, malar rash and subacute skin lupus erythematosus (SCLE) skin rash. Scarring alopecia, discoid lesions, and mucosal ulcers are less common in DIL than in SLE. Serositis especially pleurisy is also commonly seen. Although pericarditis has rarely been reported with DIL secondary to some medications, large pericardial or cardiac tamponade is rare in DIL.Hydralazine-induced lupus is often manifested by arthralgia, myalgia, fever, rash (malar rash is common), hepatosplenomegaly, lymphadenopathy, and pleuriss. Rare cases of glomerulonephritis, neuropsychiatric manifestations and pericarditis have been reported. While arthralgia, myalgia, fever, and pleuritis are commonly induced in procainamide, rash and lymphadenopathy are less common, and glomerulonephritis or CNS involvement is rare. Minocycline induced lupus is usually characterized by fever, arthralgia, arthritis, rash and rarely pneumonitis and cutaneous vasculitis. Anti-TNF agents, which are now the mainstay of the treatment of rheumatoid arthritis, have often been reported with positive autoantibodies as well as drug-induced lupus. While there is a high incidence of positive autoantibodies, ANA and anti-dsDNA in patients treated with anti-TNF drugs (up to 50%), only a few of these patients develop DIL (less than 1%).[8] Skin rash, thrombocytopenia, leukopenia, hypolonemia and rarely hemolytic anemia are manifestations of manifestations secondary to anti-TNF agents. In particular, the development of DIL from an anti-TNF agent may not always lead to DIL from another anti-TNF agent. [9] The symptoms of DIL dissolve within a few weeks to months after discontinuation of the anti-TNF agent, but the autoantibodies can remain positive for several years. Both interferon-alpha and interferon-beta have been associated with the development of DIL. As with anti-TNF agents, the development of autoantibodies including ANA and anti-dsDNA is common, while DIL develops in less than 2% cases. [10] Arthralgia, arthritis and leukopenia are the usual manifestations. Subacute pure skin lupus erythematosus (SCLE) can be idiopathic, but is drug-induced in about a third of cases. Hydrochlorothiazide is the classic drug associated with SCLE. Other drugs that cause sCLE include terbinalin, anti-TNF agents, antiepileptics, proton pump inhibitors, NSAIDs, calcium channel blockers and ace inhibitors. [11] [12] The rash appears in a photo distribution. Morphologically, SCLE is a prudish psoriasis form to an outbreak of braidoids, but can also have a scaly annular morphology with central clearing. SCLE is associated with anti-Ro/SSA positivity greater than 3/4 of the time, speckled ANA, and 30% positivity associated with anti-La/SSB. [13] [3] Lupus-like symptoms to the exclusion of other autoimmune diseases and the dissolution of symptoms with drug withdrawal indicate the diagnosis of DIL. [14] [15] Laboratory evaluation is crucial, but cannot always be able to distinguish DIL from SLE. Cytopenias are rarer and, if present, mild. Methyldopa has been associated with hemolytic anemia, while a positive Coombs test with methyldopa, procainamide and carbamazepine has been reported. The autoantibody evaluation shows a positive ANA, usually in a homogeneous pattern, although the speckled pattern has been reported. Anti-histon antibodies are present in 75% of cases of DIL, although their benefit in distinguishing DIL from SLE is limited due to their positivity in up to 75% of cases of SLE as well. Positive antihistone antibodies are in DIL secondary to several drugs including hydralazine, procainamide, hydrochlorothiazide, chlorpromazine, isoniazid, quinidine, penicillamin, anti-TNF agents, although they are less commonly seen in DIL secondary to minocycline or propylthiouracil. Anti-dsDNA antibodies can be seen in more than 50% of cases of SLE and less than 5% of DIL cases, in particular secondary to anti-TNF agents and interferon-alpha. Rare cases of positive anti-dsDNA have been reported in DIL secondary to minocycline and isoniazid. Anti-Ro/SSA antibodies can be positive in SCLE, either idiopathic or secondary to hydrochlorothiazide. Antiphospholipid antibodies including and lupus anticoagulant were reported in DIL secondary to chlorpromazine, procainamide, quinidine and interferon-alpha. These are rarely associated with thrombotic events. Antineutrophil cytoplasm antineutrophilcytoplasm (ANCA) in particular P-ANCA or atypical ANCA have been reported in DIL secondary to minocycline, hydralazine, propylthiouracil, methimazole and anti-TNF agents. In particular, up to 20% of patients with SLE may also have a positive ANCA. Other autoantibodies against antigens such as blacksmith, ribonuclear protein (RNP), SCL70, centromer, Jo-1 are rare in DIL and can help distinguish DIL from other autoimmune diseases. The laboratory evaluation also includes the evaluation of complements (C3 and C4), renal function including urine for the evaluation of proteinuria, liver function. Skin biopsy for DIL is indistinguishable from SLE, although it should be followed in case of suspicious skin lesions in order to exclude other etiologies. The mainstay of treatment is the recognition and discontinuation of the offensive drug. The development of positive ANA alone after receiving a drug is not a reason for discontinuation of the drug, although these patients should be closely monitored for the development of DIL. Symptoms of DIL usually dissolve within a few weeks of discontinuation of the drug, though rarely, they can last several months. NSAIDs or low-dose corticosteroids can be considered for milder manifestations, with high-dose corticosteroids reserved for more serious manifestations such as symptomatic pericardial effusion. As already mentioned, although the symptoms of DIL dissolve within a few weeks of discontinuation of the drug, the autoantibodies can remain positive for several months to years, and their presence alone must be a reason to use anti-inflammatory or immunosuppressive therapy. Idiopathic systemic lupus erythematosus The main difference diagnosis of DIL is idiopathic SLE. There is significant overlap in clinical characteristics and autoantibodies in both diseases. As mentioned above, severe manifestations such as renal or neurological involvement, vasculitis and severe haematological involvement are rare in DIL, and if present, will increase a high distrust for SLE. Idiopathic subacute cutaneous lupus erythematosus Idiopathic SCLE is in differential diagnosis of drug-induced SCLE. The widespread distribution of the rash and the dissolution of the rash after discontinuation of the drug are consistent with the drug-induced SCLE and not with the idiopathic SCLE. In patients with anti-TNF pathogens with fever, rash and arthralgia, infections due to the immunocompromised condition of these patients should be excluded at first. Drug-induced lupus carries a favorable prognosis with less morbidity and mortality compared to SLE. In most cases, DIL dissolves within a few weeks of discontinuation of the drug, with rare patients having to be treated for several months. Life-threatening are rare. [16] However, early detection is essential to prevent longer hospital stays or multiple outpatient visits due to uncertainty about the diagnosis. In most patients, there are no serious complications of drug-induced lupus. Rare cases of glomerulonephritis may include corticosteroids with however, the development of permanent kidney damage is rare. Many drugs can induce lupus and often these patients will present to the nurse, primary care provider, internists and rheumatologists. However, it is important to get an elaborate drug history because the diagnosis may not be straight out. In most cases, once the offending drug is discontinued, the symptoms gradually subside. However, if there is an organ dysfunction, then the patient must be monitored until the laboratory parameters are normal. The pharmacist should always keep an overview of patient medications, as it may be the first indication that a drug is involved in the pathology of the patient. [17] [18] An interprofessional team approach to the evaluation and treatment of drug-induced lupus will provide the best result. [Level V] Continuing Education / Review Questions1.REINHARDT DJ, WALDRON JM. Lupus erythematosus-like syndrome makes hydralazine (apresoline) therapy difficult. J Am Med Assoc. 1954 Aug 21;155(17):1491-2. [PubMed: 13183772] 2.Bojinca VC, Bojinca M, Gheorghe M, Birceanu A, Iosif CI, Balanescu SM, Balanescu AR. Stevens-Johnsons syndrome or drug-induced lupus - a clinical dilemma: a case report and a review of the literature. Biomed Rep. 2018 Jul;9(1):37-41. [PMC free article: PMC6007037] [PubMed: 29930803] 3.He Y, Sawalha AH. Drug-induced lupus erythematosus: an update on medications and mechanisms. Curr Opin Rheumatol. 2018 Sep;30(5):490-497. [PMC free article: PMC7299070] [PubMed: 29870500] 4.Atzeni F, Turiel M, Capsoni F, Doria A, Meroni P, Sarzi-Puttini P. Autoimmunity and Anti-TNF-alpha agents. Ann N Y Acad Sci. 2005 Jun;1051:559-69. 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