Symmetrical drug-related intertriginous and flexural exanthema (Baboon syndrome) associated with Infliximab: a case-based review.

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ABSTRACT

Background: SDRIFE is a rare cutaneous eruption characterized by symmetrical intertriginous dermatitis, caused by delayed Type-IV immune reaction, with several reported drug-triggers. Objective: We present a case of SDRIFE associated with infliximab in a 70-year-old female with rheumatoid arthritis, and review cases of SDRIFE associated with TNF-inhibitors. Methods: A literature review about SDRIFE cases associated with TNF-inhibitors was performed. Articles published in English from inception to January 6th, 2022, restricted to humans, and directly related to this review were included. Results: Ours is the third reported case of SDRIFE associated with TNF-inhibitors, and second with infliximab. SDRIFE can occur anytime during treatment with TNF-inhibitors, and presents with similar clinical and histopathological features as SDRIFE secondary to other drugs. No systemic manifestations have been reported, and the rash resolves after discontinuation of the TNF-inhibitor without any long-term sequelae. Conclusion: SDRIFE is benign, and an accurate diagnosis and discontinuation of the responsible drug remain the cornerstone of management.

Keywords: Biological therapies; Skin; Rheumatoid arthritis

INTRODUCTION

Symmetrical drug-related intertriginous and flexural exanthema (SDRIFE) is a rare benign cutaneous drug-eruption characterized by symmetrical dermatitis in the intertriginous areas of the body, most commonly reported in association with antibiotics. Tumor necrosis factor(TNF)-inhibitors can cause several cutaneous reactions, and so far, only 2 cases of SDRIFE associated with TNF inhibitors have been reported[1,2]. We report a case of SDRIFE associated with infliximab in a patient with rheumatoid arthritis (RA).

CASE PRESENTATION

A 70-year-old Caucasian female with past medical history of asthma, osteoarthritis, nephrolithiasis, deep vein thrombosis, obesity, seborrheic keratosis, hypertension, and hyperlipidemia presented for follow up of rheumatoid arthritis. She was diagnosed with RA 19 years ago and had previously failed etanercept, adalimumab, methotrexate, sulfasalazine, and leflunomide. Her current RA medications included hydroxychloroquine 400mg daily (for 15 years) and infliximab 5mg/kg every 8 weeks (initiated 2 years ago). She was not using any non-steroidal anti-inflammatory drugs. She had low disease activity (Clinical Disease Activity Index of 5, Disease Activity Score-28-CRP of 2.16) at her visit. Her other medications included losartan 50mg daily, furosemide 20mg daily, simvastatin 40mg daily, apixaban 2.5mg twice daily, tramadol 50mg twice daily, ergocalciferol 25mcg daily, albuterol 2 inhalations 4 times daily as needed, and tizanidine 4mg twice daily as needed.

The patient presented with an itchy rash that started 2-months ago as a spot on the right posterior calf, and rapidly evolved and spread to her bilateral lower extremities, abdominal fold, groin, chest, and arms. She denied any recent infections or new medications. The rash was non-

responsive to over-the-counter antihistamines (diphenhydramine 25mg twice daily). Review of systems was otherwise negative. Examination revealed sharply-demarcated, deeply erythematous moist coalescing papules and plaques involving lower abdominal folds, inguinal folds, intergluteal cleft, buttocks, posterior thighs, bilateral popliteal/antecubital fossae (Figure 1). There was minimal inframammary/axillary involvement and no involvement of scalp, face, and oral/vaginal mucosa. Punch biopsy revealed epidermal spongiosis with focal microvesicle formation, rare keratinocyte necrosis, and superficial-mid dermal perivascular lymphohistiocytic inflammation with eosinophils (Figure 2). Periodic acid-Schiff (PAS) stain with appropriate positive control did not demonstrate spore/hyphal forms. Laboratory work-up including complete blood counts, hepatic and renal function were normal. Based on clinical features and histopathology, SDRIFE was diagnosed, and treatment with topical hydrocortisone 0.2% twice daily was initiated. On reviewing her medications using the Naranjo Adverse Drug Reaction Probability Scale to ascertain the probability of her drug reaction being associated with one of her current medications, infliximab was the only medication that reached a score suggesting it was the probable cause of SDRIFE, and was discontinued (Appendix 1). At follow-up 4-weeks after discontinuing infliximab, her rash had completely resolved without any residual hyperpigmentation and without need for topical corticosteroids. The patient was started on abatacept for the treatment of RA.

LITERATURE REVIEW

Search strategy and selection criteria

Data for this review were identified by searches of MEDLINE, Scopus, Google Scholar, and references from relevant articles using the search terms “Symmetrical drug-related intertriginous and flexural exanthema”, “SDRIFE”, “Baboon syndrome”, “infliximab”, “adalimumab”, “golimumab”, “etanercept” and “certolizumab”. Articles presenting and describing cases of SDRIFE associated with TNF-inhibitors were included in this review. Articles

published in English from inception to January 6th, 2022, restricted to humans, and directly related to this review were included.

RESULTS

Our literature search yielded a total of 2 articles, which were included in this review. The first case of SDRIFE associated with infliximab was published in 2015, and the second case of SDRIFE associated with golimumab was published in 2017[1,2]. (Table I)

Demographics

The first report was of a 50-year-old-male with psoriasis who developed SDRIFE secondary to infliximab 2 days after the 10th dose of infliximab. The second report was of a 70-year-old-female with RA who developed SDRIFE 2 weeks after the first dose of golimumab. Our patient was a 70-year-old female with RA who developed SDRIFE 3 weeks after the 16th dose of infliximab. Given the paucity of reports of TNF-inhibitor induced SDRIFE, at this time, it cannot be determined if age, sex, underlying rheumatic illness, or choice of TNF-inhibitor have any association with the development of this rare adverse effect.

Clinical presentation

In both cases of infliximab-induced SDRIFE, rash onset was after several doses of infliximab, while in the case of golimumab-induced SDRIFE, rash onset was 1 week after first dose of golimumab. Thus, it can be assumed that SDRIFE can develop anytime during the course of treatment with TNF-inhibitors. Clinical presentation of the rash in all 3 cases of TNF-inhibitor induced SDRIFE was similar to that seen with SDRIFE due to other drugs. All 3 patients presented with acute-onset sharply-defined erythematous macules or papules distributed symmetrically

in the intertriginous areas including inguinal and gluteal areas in a characteristic “V-shaped” pattern. Other sites involved include abdomen, chest, axilla, neck, and antecubital areas. There was notable sparing of palmar, plantar, facial, and mucosal areas. Vesiculobullous lesions were reported with golimumab. Pruritus was present in all patients, while systemic symptoms were absent and blood tests were normal.

Histopathological features

All 3 cases had histopathological features of superficial perivascular infiltration with mononuclear cells admixed with neutrophils and/or eosinophils, vacuolar interface dermatitis and spongiosis. Other findings reported include dermal edema and subcorneal pustules. These histopathological reports are in alignment with histopathology seen in SDRIFE induced by other drugs.

Treatment

In all 3 cases, discontinuation of the TNF-inhibitor led to complete resolution of rash. Topical corticosteroids and oral antihistamines were used as supportive care, while none of these cases required systemic corticosteroids. In the previously reported case of infliximab-induced SDRIFE, retrial of infliximab led to a more severe eruption non-responsive to topical treatments. Interestingly, this patient was switched to adalimumab and no recurrence of rash was observed, suggesting this to be a drug-specific phenomenon rather than being associated with TNF-inhibition.

DISCUSSION

In 1983, Nakayama *et al* described 15 cases of mercury inhalation-induced systemic contact dermatitis, characterized by diffuse symmetrical erythematous rash involving the flexural areas, 1-2 days after breaking a clinical thermometer or during a dental procedure in patients, most of

whom had a previous history of contact dermatitis to Mercurochrome[3]. In 1984, Anderson *et al* described 3 cases of systemic contact dermatitis due to mercury, nickel, and ampicillin, and introduced the term "Baboon Syndrome", given the resemblance of the rash to red bumps of baboon[4]. In 2004, Häusermann *et al* proposed the criteria distinguishing SDRIFE from systemic contact dermatitis, which included (a) first or repeated exposure to systemic drug excluding contact allergens, (b) sharply demarcated erythematous rash in gluteal/perianal or inguinal/perigenital areas, (c) involvement of another intertriginous site, (d) symmetrical involvement, (e) lack of systemic signs/symptoms[5].

Pathophysiology of SDRIFE is secondary to type-IVa delayed hypersensitivity immune response involving CD4+ lymphocytes and macrophages, and type IVc reaction involving cytotoxic CD8+ lymphocytes and CD4+ lymphocytes[6–9]. There is immunohistochemical evidence of increased endothelial and keratinocyte expression of CD26P-selectin, which recruits type-1 helper T-cells to the sites of inflammation. At the dermo-epidermal junction, main effector cells are neutrophils, eosinophils, and cytotoxic TIA1+ cells while in the dermis, both CD4+ and CD8+ lymphocytes are present with a CD4/CD8 ratio from 1:1 to 2:1 in addition to macrophages, granulocytes, plasma cells, and B-cells[6].

A recent review of 73 reported cases of SDRIFE suggested average age of 51 years (range 18 months to 88 years), with slight male to female predominance in a ratio of 1.5:1[10]. The latency between drug initiation and onset of rash has ranged from 1 hour to 120 days with an average of 3 weeks, and the rash can occur as soon as the agent is introduced, or after repeated exposures[6,11,12]. Beta-lactams and sulfa antibiotics have been the most commonly reported drugs (50%) responsible for SDRIFE[10]. Several other drugs have been reported to cause SDRIFE, including other anti-infective agents, antihypertensives, non-steroidal antiinflammatory drugs, iodine radio-contrast, chemotherapeutic agents, and topical bufexamac and 5-fluorouracil[5,6,13,14]. Cases of SDRIFE have been reported after COVID-19 infection and COVID-19

vaccination[15–17]. Clinical presentation of SDRIFE is characteristic and patients present with acute-onset sharply-defined, symmetrical erythematous maculopapular rash involving intertriginous areas including inguinal and gluteal areas in a characteristic "V-shaped" pattern, axilla, inframammary folds, abdomen, neck, popliteal fossa, and antecubital areas[5]. Pustules, vesicles, bullae, erosions, lichenified lesions, and urticaria-like lesions have also been reported. Pruritus is present in up to 90% of cases, and the absence of systemic signs and symptoms is a hallmark of this syndrome[10,12].

In contrast to homogeneity observed in clinical features, the histopathology of SDRIFE is rather heterogeneous and non-specific. Superficial perivascular lymphocytic infiltrate with eosinophils/neutrophils is present in most cases[7]. Dermal eosinophils and spongiosis are commonly reported. Less commonly-reported findings include subcorneal or intraepidermal pustules, apoptotic keratinocytes, papillary dermal edema, vacuolar interface changes, and extravasated erythrocytes[7,18,19]. Muresan *et al* reported many histopathological patterns, sometimes more than one pattern in a single patient with SDRIFE[7]. Most commonly present were interface dermatitis, spongiotic dermatitis, and psoriasiform dermatitis, some cases also had pustular dermatitis, perivascular and interstitial neutrophilic dermatitis, and interstitial granulomatous dermatitis. Leukocytoclastic vasculitis was reported in one case of amoxicillin-induced baboon syndrome but has not been reported in any other cases of SDRIFE since[4].

Differential diagnosis of SDRIFE is wide but typical characteristics of the rash and histopathological features can differentiate SDRIFE from other drug eruptions such as acute generalized exanthematous pustulosis (AGEP), drug reaction with eosinophilia and systemic symptoms (DRESS), erythema multiforme, intertriginous form of toxic erythema of chemotherapy (TEC), and fixed drug eruption (FDE)[6,7]. AGEP and DRESS are associated with widespread rash as well as systemic symptoms, while FDE presents with asymmetrical round or oval patches and plaques with residual hyperpigmentation, involving acral, genital, and mucosal

surfaces[2,20]. Psoriasis (new-onset or worsening) has been reported secondary to TNF-inhibitors and can be differentiated from SDRIFE based on clinical and histopathological features such as presence of neutrophils in the stratum corneum and upper layer of the epidermis in psoriasis[21]. Other differentials include seborrheic dermatitis, intertrigo, allergic contact dermatitis, granular parakeratosis, candidiasis, tinea, Darier disease, and Hailey-Hailey disease[22]. Patch tests and drug provocation tests can be considered, however, sensitivity of patch tests is poor due to incomplete absorption of a systemic agent when applied to the skin during patch test, and drug provocation tests are not always practical[23].

SDRIFE has a benign course, and the cornerstone for treatment is identifying and discontinuing the offending drug which leads to spontaneous resolution of the rash without residual hyperpigmentation within 1-2 weeks[24]. In rare cases, the rash has been reported to last longer but eventually resolves without hyperpigmentation. Topical corticosteroids and oral antihistamines have been reported to provide symptomatic relief, and systemic corticosteroids are rarely needed in severe cases.

TNF-inhibitors have been associated with several systemic and cutaneous adverse effects. The most commonly reported cutaneous adverse reactions include injection-site and allergic reactions. Other less common cutaneous adverse effects include Stevens-Johnson Syndrome, cutaneous/leukocytoclastic vasculitis, urticaria, linear IgA bullous dermatosis, acute generalized exanthematous pustulosis, erythema multiforme, erythema nodosum, toxic epidermal necrolysis, psoriasis, alopecia, and lichenoid skin reaction[25]. However, SDRIFE has not been frequently reported with TNF-inhibitors. Delayed type-IV immune reactions have been reported secondary to TNF-inhibitors, and could be the likely pathophysiology for development of SDRIFE. However, given the rarity of this adverse effect, the exact immunologic mechanisms cannot be ascertained. There does not seem to be cross-reactivity between TNF-inhibitors as in the case of infliximab-induced SDRIFE by Bulur *et al*, the patient was able to tolerate adalimumab without

any recurrence of SDRIFE. Prompt identification of the etiology and discontinuation of the TNF-inhibitor has led to complete resolution of the rash in all the known cases of TNF-inhibitor-induced SDRIFE.

CONCLUSION

SDRIFE is a rare drug eruption with characteristic clinical features, antibiotics being the most commonly-reported causative agent. TNF-inhibitors have been associated with several cutaneous and systemic adverse effects. However, SDRIFE secondary to these drugs is rare, and to our knowledge, this is only the second reported case of SDRIFE associated with infliximab, and the third associated with TNF-inhibitors. Physicians should be vigilant about this rare adverse effect as prompt diagnosis and discontinuation of the TNF-inhibitor leads to complete resolution of the rash.

Abbreviations

SDRIFE: Symmetrical drug-related intertriginous and flexural exanthema

RA: Rheumatoid arthritis

TNF: Tumor necrosis factor

CDAI: Clinical disease activity index

CRP: C-reactive protein

Patient consent statement

Informed and written consent of the patient was taken prior to writing this case, and the patient has graciously given us permission to present and report her case.

Ethical statement

The Mayo Clinic Institutional Review Board (IRB) acknowledges that based on the responses submitted for this new activity through the Mayo Clinic IRB Human Subjects Research Wizard tool, and in accordance with the Code of Federal Regulations, 45 CFR 46.102, the above-noted activity does not require IRB review.

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Conflict of interest

We confirm that the authors have no financial disclosures, competing interests, or conflict of interest.

Data availability and deposition

The authors confirm that the data supporting the findings of this study are available within the article [and/or] its supplementary materials.

Author contributions

All authors contributed to the study design, critically reviewed the first draft, approved the final version, and agreed to be accountable for the work. All authors have full access to the manuscript and all the data in the study.

Table and Figures legends

Figure 1: (A) Well demarcated erythematous plaques in the right inguinal fold. (B) Erythematous plaques with fine scales in the left inguinal fold. (C) Well-demarcated erythematous eroded plaques in the right popliteal fossa. (D) Erythema across chest and neck.

Figure 2: (A) H&E stain, 20x original magnification: Superficial dermatitis with perivascular inflammation and spongiosis. (B) H&E stain, 200x original magnification: Epidermal spongiosis with microvesicle formation (single arrow), rare keratocyte necrosis (double arrow), and superficial perivascular lymphohistiocytic inflammation (triple arrow) with eosinophils (quadruple arrow).

Table I: Review of cases of TNF-inhibitor induced SDRIFE. TNF: Tumor necrosis factor, SDRIFE: Symmetrical drug-related intertriginous and flexural exanthema, RA: Rheumatoid arthritis.

Figure 1.

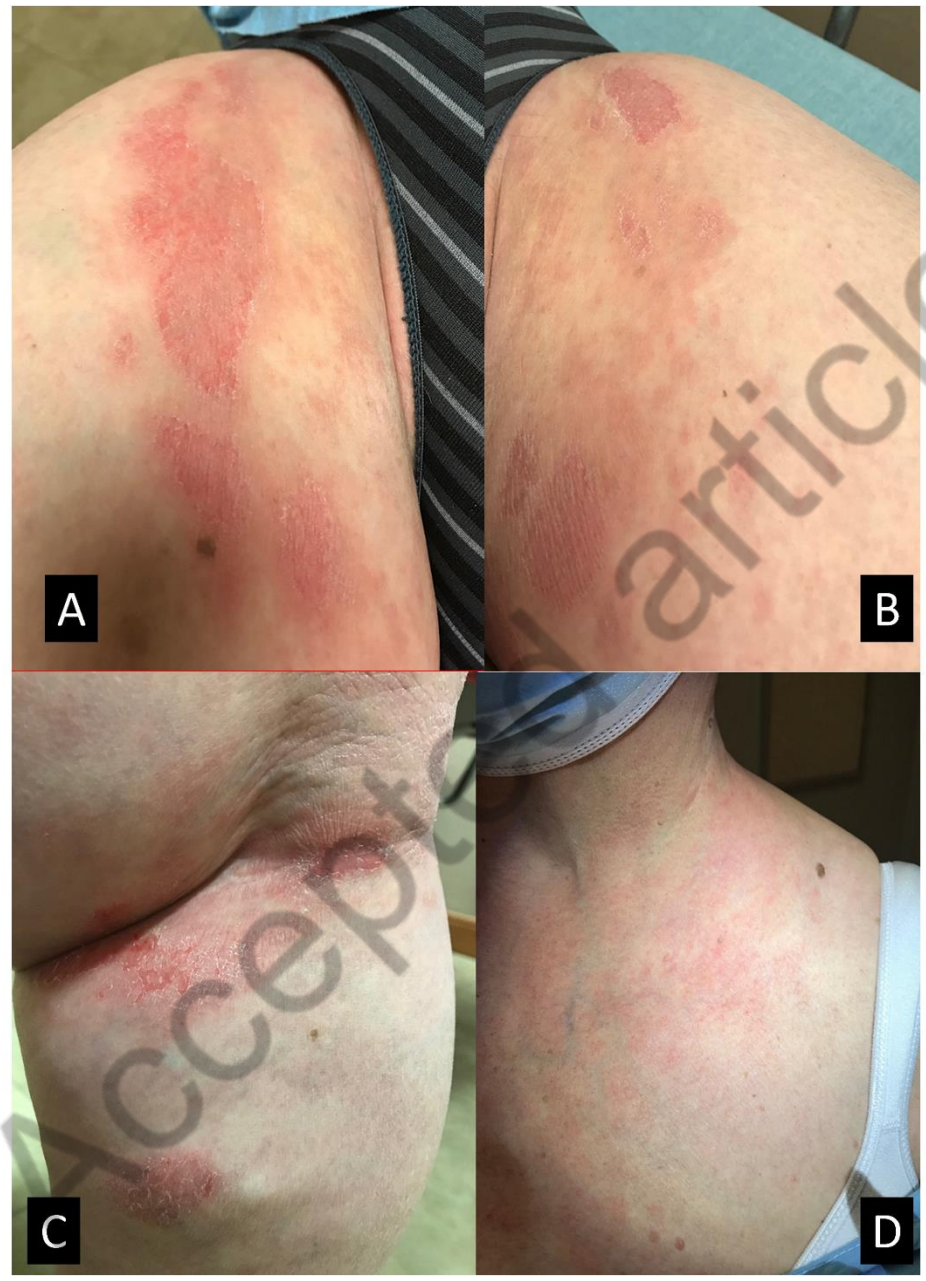
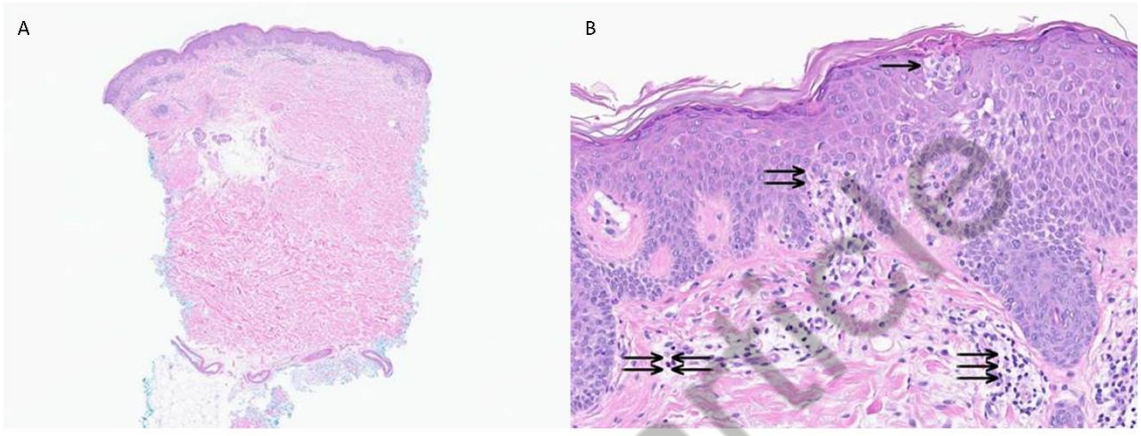


Figure 2.



	Infliximab	Losartan	Furosemide	Simvastatin	Apixaban	Tramadol	Tizanidine	Albuterol	Ergocalciferol
1. Are there previous conclusive reports on this reaction?	Y (+1)	N (0)	N (0)	N (0)	N (0)	N (0)	N (0)	N (0)	N (0)
2. Did the adverse event appear after the suspected drug was administered?	Y (+2)	Y (+2)	Y (+2)	Y (+2)	Y (+2)	Y (+2)	Y (+2)	Y (+2)	Y (+2)
3. Did the adverse event improve when the drug was discontinued or a specific antagonist was administered?	Y (+1)	D (0)	D (0)	D (0)	D (0)	D (0)	D (0)	D (0)	D (0)
4. Did the adverse event reappear when the drug was readministered?	D (0)	D (0)	D (0)	D (0)	D (0)	D (0)	D (0)	D (0)	D (0)
5. Are there alternative causes that could on their own have caused the reaction?	N (+2)	Y (-1)	Y (-1)	Y (-1)	Y (-1)	Y (-1)	Y (-1)	Y (-1)	Y (-1)
6. Did the reaction reappear when a placebo was given?	D (0)	D (0)	D (0)	D (0)	D (0)	D (0)	D (0)	D (0)	D (0)
7. Was the drug detected in blood or other fluids in concentrations known to be toxic?	D (0)	D (0)	D (0)	D (0)	D (0)	D (0)	D (0)	D (0)	D (0)
8. Was the reaction more severe when the dose was increased or less severe when the dose was decreased?	D (0)	D (0)	D (0)	D (0)	D (0)	D (0)	D (0)	D (0)	D (0)

9. Did the patient have a similar reaction to the same or similar drugs in any previous exposure?	N (0)	N (0)	N (0)	N (0)	N (0)	N (0)	N (0)	N (0)	N (0)
10. Was the adverse event confirmed by any objective evidence?	Y (+1)	Y (+1)	Y (+1)	Y (+1)	Y (+1)	Y (+1)	Y (+1)	Y (+1)	Y (+1)
Total Score	**7**	**2**	**2**	**2**	**2**	**2**	**2**	**2**	**2**

Appendix Table I: Naranjo Adverse Drug Reaction Probability Scale. Y: Yes. N: No. D: Do not know. For questions 1, 3, 7, 8, 9 and 10: Yes = +1, No = 0, Do not know = 0. For questions 2 and 4: Yes = +2, No = -1, Do not know = 0. For question 5, Yes = -1, No = +2, Do not know = 0. For question 6, Yes = -1, No = +1, Do not know = 0. Total scoring: ≥9 = Definite. 5 to 8 = Probable. 1 to 4 = Possible. ≤0 = Doubtful.

Adapted from *LiverTox: Clinical and Research Information on Drug-Induced Liver Injury [Internet]. Bethesda (MD): National Institute of Diabetes and Digestive and Kidney Diseases; 2012-. Adverse Drug Reaction Probability Scale (Naranjo) in Drug Induced Liver Injury. [Updated 2019 May 4]. Available from: https://www.ncbi.nlm.nih.gov/books/NBK548069/*

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