Journal of the Postgraduate Institute of Medicine 2023; 10(1): E201 1-11

http://doi.org/10.4038/jpgim.8398

Case Report

A Case of Dapsone-induced DRESS syndrome and concomitant thyrotoxicosis

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Key words: **Dapsone, DRESS, thyrotoxicosis, hepatobiliary, anti TPO antibody, eosinophilia**

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Received: 04 Sep 2022, accepted revised version: 20 Oct 2022, Published: 16 Jan 2023

Competing Interests: Authors have declared that no competing interests exist

Introduction

Drug reaction with eosinophilia and systemic symptoms (DRESS) is a severe idiosyncratic drug reaction characterized by rash, eosinophilia, lymphadenopathy, visceral organ involvement and atypical lymphocytosis [1,2]. The condition is recognized as one entity of the severe cutaneous adverse drug reactions (SCARs). The disease carries a mortality close to 10% and has a close association with other diseases of autoimmune aetiology [2,3]. The disease is recognized to occur following the use of certain drugs more than others, one of them being dapsone which is commonly used to treat disease conditions such as leprosy (Hansens disease), dermatitis herpetiformis, cicatricial pemphigoid [4]. The disease is known to cause complications such as limbic encephalitis, thyroid disease, renal failure, splenic rupture, eosinophilic colitis, eosinophilic oesophagitis, enterocolitis, and fatal cytomegalovirus (CMV) disease [5]. Associated thyroid disease and endocrinopathy is recognized. Hypothyroidism is more common, although cases of hyperthyroidism are also reported [6].

The latency between the first exposure to the drug and the onset of symptoms can range from 2 to 8 weeks. The condition differs from a typical drug reaction as it may persist, and even worsen, despite discontinuation of the drug in question [7]. Although eosinophilia is included in the nomenclature, it is only present in 66%-95% of individuals, and atypical lymphocytes in 27%-67%. In the early phases of the disease, decreased number of lymphocytes and hypoglobulinemia has also been described [2,8,9]. Thus, in a patient coming with DRESS, the diagnosis is not always straight forward. Nevertheless early treatment is mandatory and should be initiated without delay to avoid fatal complications. As such, a high degree of clinical suspicion is necessary.

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There are many scoring systems and criteria to assist in making a diagnosis of DRESS syndrome. RegiSCAR and Japanese group's criteria are used, with the former more widely applied [9,3]. Our diagnosis was based on RegiSCAR criteria (Figure 1) as its sensitivity seems to be higher in comparative studies [10].

Item	Comment	SCORE			Patient score
		-1	0	1	
Fever >38.4◦		N/U	Y		0
Enlarged Lymph nodes	>1cm and ≥ 2 different sites		N/U	Y	1
Eosinophils	Score 2 when ≥1.5 ×10^9/L or >20% if WBC <4× 10^9/L		N/U	Y	2
Atypical Lymphocytes			N/U	Y	1
Skin rash >50% body surface area			N/U	Y	1
Skin rash suggesting DRESS		N	U	Y	1
Skin biopsy suggesting DRESS		N	Y/U		0
Organ involvement	Score 1 for each organ involvement, maximum Score 2		N	Y	1
Resolution≥15 days		N/U	Y		1
Evaluation of other potential causes	Score 1 if 3 of the following tests were performed and all were negative HAV, HBV, HCV, ANA, Mycoplasma, Chlamydia, Blood culture		N/U	Y	1
Total score					9

N: No, U: unknown, Y: yes, HAV: Hepatitis A virus, HBV: Hepatitis B virus, HCV : Hepatitis C virus, ANA: Antinuclear antibody
Final score <2 = No ,2-3 = Possible, 4-5 = Probable, 5>= Definit

Figure 1: RegiSCAR scoring system for DRESS syndrome

DRESS syndrome with dapsone in the setting of leprosy has been reported in Sri Lanka [3,5] and is a well-established phenomenon. But to our knowledge, there are no reports of concomitant or subsequent thyrotoxicosis in Sri Lanka. We present a case of dapsone-induced DRESS syndrome in a patient treated for multibacillary leprosy. His unusual

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presentation was a combination of symptoms of thyrotoxicosis and drug reaction. Thyrotoxicosis or thyroiditis is a well-recognized, but extremely rare, sequel of DRESS syndrome. To our knowledge, this is one of the very few reported cases where the patient presented with concomitant symptoms of thyrotoxicosis and DRESS.

Case Report

We present a case of a 45-year-old bus conductor from the suburbs of Kurunegala. One year previously he had noted lethargy, malaise and loss of weight. He then developed hypopigmented, anaesthetic patches on the back of his chest and an erythematous rash on his left upper limb. Upon referral to dermatology, a histological diagnosis of multibacillary leprosy was made and he was started on treatment. At this point, the patient was not febrile and there was no organomegaly, lymphadenopathy or biochemical abnormalities in his basic investigations. The patient did not have systemic symptoms or fever.

Following commencement of anti- leprosy treatment, he complained of progressive, low volume, loose stools associated with increased frequency of defaecation around 8 times a day. He had lost close to 20kg of weight from his original weight of 70kg. Exactly 21 days into leprosy treatment with dapsone and clofazamine, the patient developed severe arthralgia and myalgia with high grade fever around 101-102° F. The fever responded to paracetamol and usually resolved with drenching sweats. One week prior to admission, the patient developed a generalized erythematous rash with flushing. He was treated, 5 days prior to presenting to us, at a district general hospital where he was found to have pancytopaenia with fever and his dapsone was withheld. Three days prior to presentation, he noticed severe itching of the body with yellowish discoloration of eyes and dark urine. His fever was persistent and he complained of severe constitutional symptoms including arthralgia, myalgia and anorexia. He had left the local hospital, where he did not receive any specific drug therapy, against medical advice 4 days before,

On examination, the patient was thin, anxious and clammy with high grade fever. He was pale and deeply icteric. There were aphthous oral ulcers. He had a palpable goitre with a prominent left lobe with no overlying bruits. There was generalized lymphadenopathy with large firm lymph nodes ranging from 1cm to 3cm which were non-tender and matted but not fixed. He was flushed, with confluent erythema, in keeping with erythroderma without desquamation. There were hypopigmented patches on the back of the chest which were anaesthetic consistent with his diagnosis of leprosy. There were generalized scratch marks with polished nails. The common peroneal and ulnar nerves were thickened. Cardiovascular examination revealed a tachycardia around 120bpm with a bounding and collapsing pulse. The pulse pressure was wide, with a BP of 120/20mmHg. There was a systolic murmur in the left upper sternal border with no radiation. Abdominal examination revealed a non-tender, firm moderate hepatomegaly 3-4cm below the costal margin, with regular borders. Spleen was firm and 3 cm below the costal margin. No hepatic bruits or splenic rubs were heard.

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His investigations (Table 1) revealed a high white cell count with a lymphocytic predominance initially, later progressing to an eosinophilia (30%). His full blood count prior to admission had been consistent with pancytopaenia. His CRP was 62 with a normal ESR of 26mm. Blood and urine cultures were negative. His liver function tests revealed a predominantly cholestatic pattern with high transaminases (AST>ALT). His TSH was <0.015mIU/L (0.4-4.6mIU/L), free T4 was >90 (10-28.2pmol/L) and free T3, 33.7 pmol/L (4.26-8.1pmol/L). Ultrasound scan of the thyroid showed a diffusely enlarged gland with coarse echogenicity and significantly increased vascularity. Right Lobe 1.8×1.9cm, Left Lobe 2.2 ×1.9cm, Isthmus 0.35cm. The conclusion was of a diffuse goitre due to thyroiditis. Ultrasound scan of the abdomen showed hepatosplenomegaly with the liver slightly hypoechoic and 21cm in size and spleen 14×8.5cm in size.

Table 01: laboratory data

Parameter	Normal	Admission	In ward	Discharge
FBC				
WBC ($\times 10^9$/L)	4-10	21.53	7.61	8.01
Neutrophils($\times 10^9$/L)	2-7	7.32	3.78	3.03
Lymphocytes ($\times 10^9$/L)	0.8-4	12.64	2.58	1.15
Eosinophils($\times 10^9$/L)	0.02-0.5	0.28	0.48	2.98
Hb (g/dL)	11-16	9.2	10	9.6
Plt ($\times 10^9$/L)	150-450	179	221	359
MCV fL	80-100	91.8	86.5	
ESR (mm)		26		
CRP mg/L	<10	62	12	2.8
AST(U/L)	<45	104	147	81
ALT (U/L)	<31	136	195	147
ALP (U/L)	30-120	229	322	167
GGT (U/L)	11-61	124	516	202
T.Bilirubin (µmol/L)	5-19	190	458	178
D. bilirubin (µmol/L)	1.7-6.8	146	347	141
I.Bilirubin (µmol/L)		43	110	
T.Protein (g/dL)	6.6-8.3	5	5.6	5.4
Albumin (g/dL)	3.5-5.3	3	3	2.7
Globulin (g/dL)		2	2.6	2.7
INR		1.7	2.52	1.6
APTT (seconds)	26-36	42.4	52.3	38
Sodium (mmol/L)	136-146	137		136
Potassium (mmol/L)	3.5-4.5	3.9		4.5

Blood picture initially showed a reactive film with atypical lymphocytes. Bone marrow aspiration was consistent with a hypercellular marrow with normal cell trails consistent

with a reactive marrow, with no evidence of haematological or non-haematological malignancy. Erythropoiesis was active with normal maturation. There was active granulopoiesis with a left shifted myeloid lineage. There was only mild eosinophilia with vacuolations at the time of bone marrow biopsy with no excess blasts. Megakaryopoiesis was active and showed normal morphology. Lymphocytes and plasma cells, were normal in number and morphology on trephine biopsy. There was increased histiocyte number and activity. Serum LDH level was raised at 552 IU/L (135-225) and CPK was normal 20U/L (<171).

Mantoux test and sputum samples for AFB were negative. Retroviral studies, hepatitis serology and VDRL were negative. Bone marrow samples for culture and TB studies were negative. Malaria antigen was negative. Brewers test was negative. 2DECHO did not show evidence of infective endocarditis or any myocardial involvement. His anti TPO antibody was highly positive at 500IU/L (9 IU/L). ANA, and rheumatoid factor were negative.

A diagnosis of DRESS syndrome associated with dapsone was made according to the RegiSCAR scoring system (figure 1). He had fever, skin eruptions, lymphadenopathy, organomegaly with liver involvement, lymphocytosis with atypical lymphocytes and eosinophilia later and exclusion of other causes, giving a score of 9 to lead to a comfortable diagnosis (Figure 1). Initially, he was started on carbimazole 20mg twice a day and propranolol 20 mg twice daily for the thyrotoxicosis. Despite treatment for thyrotoxicosis, the patient's fever did not resolve, running very high spikes, and his general condition was static. Thus. we arranged an early bone marrow biopsy following which methyl prednisolone was commenced, initially as pulses of 1g for 3 days later converting to oral prednisolone 45mg/d as for DRESS syndrome.

He showed dramatic clinical and biochemical improvement with steroid therapy. With the commencement of carbimazole, there was an increase in cholestasis, thus a drug free interval was given till his liver functions normalized. There was resolution of fever, lymphadenopathy and hepatosplenomegaly soon after the commencement of steroids and the patient's constitutional symptoms and appetite improved drastically. Liver functions also improved. We were able to restart him on carbimazole and gradually increase the dose. His prednisolone was gradually tapered off. His anti-leprosy medications were restarted with a dapsone-free regimen that included ofloxacin.

During subsequent clinic visits, his eosinophilia resolved and on re-evaluation there was no residual lymphadenopathy or hepatosplenomegaly and he was clinically feeling very much better. The timeline of illness is depicted in figure 2.

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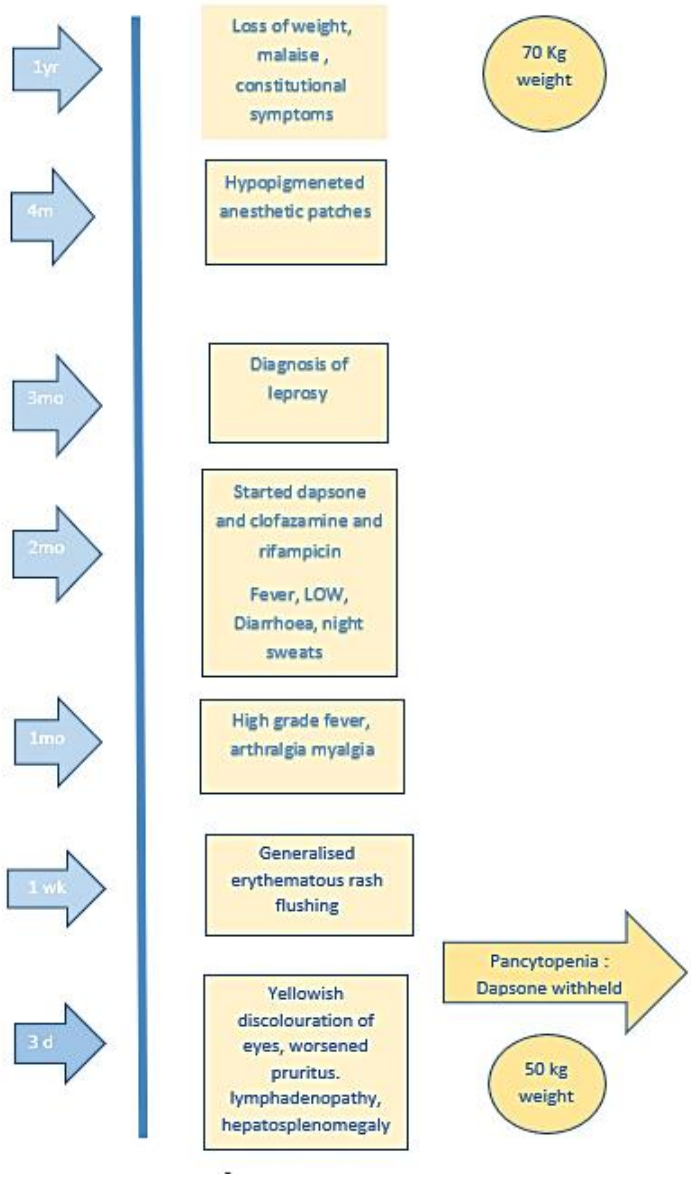

Figure 2: Timeline of illness

Discussion

DRESS syndrome secondary to dapsone occurs at a frequency of 0.2% to 0.5% of patients on dapsone therapy (5) This being a rare manifestation in itself, concomitant thyrotoxicosis is seldom reported. A case report of dapsone hypersensitivity with myocarditis and thyrotoxicosis published by Teo *et al*. in 2006, describes a case of thyroiditis where the symptoms manifested 12 days after admission. Our case is unique in that the patient gave a history of toxic features early on in the disease course and they were predominant at the time of admission. Other features, such as lymphadenopathy and hepato-splenomegaly were not noted during admission to the local hospital hence, presumably, they developed within the 5 days prior to admission.

The diagnosis in our case was made retrospectively, as the patient was not responsive to anti-thyroid treatment and features such as organomegaly, lymphadenopathy, high fever spikes and constitutional symptoms remained until steroids were commenced. The patient strongly fulfilled the RegiSCAR criteria for the diagnosis during the hospital stay but the manifestations were phasic, especially the eosinophilia. Thus the need for a high degree of clinical suspicion for early drug withdrawal cannot be emphasized enough as the disease and its complications are often fatal.

The pathogenic mechanisms of DRESS or drug induced hypersensitivity syndrome (DIHS) remain unclear to date but many mechanisms have been hypothesized. Reactivation of human herpes viruses (HHV6) have been postulated in several reports [11]. Although the mechanisms of reactivation of several herpes viruses and subsequent multiorgan involvement has not been unearthed completely, it is understood that the expansion of fully functional regulatory T cells in the acute phase of DRESS allows viral reactivation. Moreover, this leads to a loss of suppressive function upon clinical recovery. The subsequent development of autoimmune sequelae is thought to be due to the functional defects of T cells [12].

Certain ethnicities and HLA subtypes have also been associated with the development of DRESS, and these may be specific to the drug in question. Dapsone, for one, is known to have an association with B*1301, noted commonly in people of Chinese ethnicity [13]. Several studies have discussed about the role of pharmacogenetics in the pathogenesis of DRESS syndrome [14,15,2].

Thyroid dysfunction is the most commonly reported autoimmune consequence of DRESS syndrome amounting to approximately 4.8% of cases. The disease can manifest as Grave's thyroiditis, Hashimoto's thyroiditis or other forms of painless thyroiditis. There have been reported cases where patients were positive for thyroid autoantibodies (anti-thyroid peroxidase, anti-thyroglobulin) despite not having clinical features suggestive of thyroiditis. The delayed development of thyroid disease after 1-17months following DIHS is known to occur, emphasizing the need to monitor these patients even after complete resolution of symptoms [2,12].

Most cases of DRESS-induced thyrotoxicosis have been described as a sequel of the syndrome [16,12]. Concomitant toxicosis, where the patient presented initially with toxic features later developing eosinophilia and organ involvement, is scarce in the literature. The possibility of pre-existing thyroid disease in our patient cannot be ruled out entirely. But his symptoms manifested after the initiation of dapsone and the temporal nature was supportive of DRESS-induced toxicosis. Nevertheless, patients with pre-existing autoimmune disease may have a predilection for DRESS syndrome and this remains an area that needs further exploration. Our patient could be a case of undiagnosed Grave's disease, a case of thyroiditis with concomitant DRESS or thyrotoxic storm in the presence of DRESS.

Other than thyroid disease, several other autoimmune diseases have also been reported following resolution of DRESS. The mechanism described is similar to that seen in

autoimmune thyroid disease, where dysfunction of regulatory T cells during the resolution phase has been implicated. Furthermore, decreased production of free radicals, inhibition of phospholipaseA2 and altered expression of tumor necrosis factor and interferon- Y have been described [10]. Other autoimmune diseases described include systemic lupus erythematosus, vitiligo, alopecia areata, autoimmune hemolytic anemia and reactive arthritis [2,16]. Many case reports have described patients with DRESS who are initially negative for ANA who develop ANA positive lupus later [10]. Thus even in the context of negative autoantibodies, vigilance for future autoimmune sequelae should be emphasized in any patient with a diagnosis of DRESS syndrome.

Being a frequent phenomenon, liver injury is described as the prodrome of DRESS by some authors [17]. Although hepatocellular and cholestatic patterns are both seen, younger patients tend to develop a more hepatocellular picture(14)(2). Those with liver injury are shown to have higher atypical lymphocytes as seen in our patient. The description of R ratio is used to identify the injury pattern as cholestatic, hepatocellular or mixed [R ratio = (serum ALT/ALT ULN)/ (serum ALP/ ALP ULN) where ratio<2 is compatible with a cholestatic, more than 5 with hepatocellular and 2-5 with mixed pattern] revealed a mixed pattern, initially, but later on he developed a cholestasis with a ratio of 0.5 while on carbimazole. His liver functions failed to improve initially, and we had to withhold carbimazole until the liver functions recovered, later gradually introducing carbimazole under steroid cover. There was no clear guidance or literature on managing similar situations in the presence of DRESS but our management was based on patient response as most drugs used in thyrotoxicosis have an impact on liver functions. Thyrotoxicosis per se is a cause of cholestasis ([18] although in our patient the temporal nature was more in keeping with drug-induced cholestasis.

The disease might sometimes progress despite discontinuation of the offending agent or starting immunosuppression [19]. In some patients, a relapsing and chronic course has been seen following the first episode, despite avoiding the responsible drug. Our patient developed most of the symptoms following drug discontinuation. The eosinophilia was noted more than 3 weeks into the initial presentation and high grade fever continued until immunosuppression was commenced. Thus, although the trigger is related to a drug, the course is that of a typical monophasic autoimmune disease which might carry a chronic sequel or later turn out to be polyphasic.

The haematological abnormalities generally occur 2 weeks following the onset of drug reaction, mostly a leukocytosis with atypical lymphocytes followed by eosinophilia. The eosinophilia may have a role in the visceral organ involvement due to the substances released from the granules. Literature also describes leucopenia and lymphopenia that precede the leukocytosis. Some articles describe rare cases of anemia and thrombocytopenia as well [20,21]. These manifestations were all seen in our patient starting from leucopenia and the transition, in retrospect, helped us with making a diagnosis.

Although, initially, we were contemplating on lymph node biopsy in view of excluding a haematological malignancy, we deferred the procedure due to the altered clotting profile. The thyrotoxicosis, constitutional symptoms and the progressively worsening symptoms despite withdrawal of the offending drug which were all atypical in a drug eruption kept us vigilant regarding other possible diagnoses. The erythroderma noted in this dark skinned South Asian individual was not a frequently described lesion in DRESS. Nevertheless, the skin eruptions in DRESS are known to be diverse including diffuse maculopapular inflammatory reactions (most common), erythema multiforme (EM), SJS/TEN, pruritic eruptions and even erythroderma [21].

A high degree of clinical suspicion and usefulness of standardized diagnostic criteria in heterogenous presentations needs to be emphasized specially in case of drug- associated DRESS.

Conclusion

DRESS syndrome, being an atypical and rare drug-related hypersensitivity reaction with an autoimmune basis, can occur with a considerable latency. It is characterized by multiorgan involvement and, uncommonly, can be associated with autoimmune sequelae such as thyroid dysfunction. Concomitant symptoms of thyrotoxicosis at the outset has not been widely described but poses many unanswered questions on the pathophysiology including a possible association between these autoimmune diseases or a common genetic basis. The heterogenous nature of presentation can mask the primary disease and mimic other haematological and neoplastic diseases and delay drug withdrawal. Thyrotoxicosis in the presence of liver injury may pose a therapeutic challenge and management has little guidance in literature. Thus, it should be individualized. Patients with a diagnosis of DRESS should be followed up for future relapses and autoimmune sequel while exerting caution when using the culprit drugs in the future.

Acknowledgements

We express our gratitude to the patient who kindly gave consent for publication of this case report

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