"Unraveling the Enigma: Dress Syndrome as Severe Idiosyncratic Drug Reaction"

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Abstract:
Drug allergies, including eosinophilia and systemic symptom syndrome, are acute, delayed-acting reactions to specific drugs. It is described in many words; however, burns with eosinophilia and systemic disease seem more appropriate. This infection causes clinical symptoms anywhere from 2 to 8 weeks after starting the offending drug. Standardized diagnostic criteria have been developed; However, its benefits have been demonstrated. Unfortunately, strong evidence-based statements do not adequately control the management of burn medications for eosinophilia and systemic disorders.

Index Terms:
Drug hypersensitivity, Anticonvulsants, Drug reaction, DRESS, DIHS, Eosinophilia.

Introduction:
Numerous systemic and cutaneous responses were brought on by the introduction of new medications[1]. Drug Reaction with Eosinophilia & systemic symptoms, known in English by the acronym Dress (Drug Reaction with Eosinophilia and systemic symptoms). One serious disadvantage is that delayed drug administration with hypersensitivity of many organs [2] Delayed drug administration hypersensitivity, and more recently, drugs delayed hyperactivity disorder (DIHS), I’s confusing however, this syndrome is of particular importance to acknowledge Mortality is 10% [3] DRESS has later onset and longer duration of action than other drugs, including latent period. It takes 2 to 6 weeks. It affects both adults and children and may be associated with injury or even death(5)
Anticonvulsants (especially phenobarbital, phenytoin & sulfonamides are the most common stimulants, but antibiotics are reported to increase up to 30%; in the number of children Cases [9] Drug reaction with eosinophils & systemic (Dress) is also a severe, systemic, T cell -drug reaction characterized by a combination of cutaneous, hematologic and visceral drugs for fever, rash, and eosinophilia recognized since the 1930s There is a term Dress is more recent The term “rash” was In Dress later changed in response because of this skin types.[10][12][13]
Fig 1: dress syndrome

Epidemiology:

The estimated incidence in the general population is more than one case for every 10,000 drug exposures. Additional information reveals an incidence of 10 cases per million in the general population. In children 1 case in 1000 and 1 case in 10000 drug exposure. The incidence in hospitalized patients varies from 2.18 to 40/100000 in patients. The death rate in dress syndrome can vary from 3.8% to 10% even with treatment in place [2][10]. There is little epidemiological data on Dress because of its complex nomenclature and wide range of clinical presentations. The likelihood of receiving an aromatic antiepileptic prescription for first or second time is through to be between 1:1000 and 1:10000, however this greatly depends on patients ethnic origin[10].

Etiology:

50% of cases of pediatric dress syndrome are induces by aromatic anticonvulsants such as carbamazepine & lamot anticonvulsants NSAIDs. Sulfasalazine, and antibiotics all have a Function Adults who use aromatic anticonvulsants account for (35%) of instances while other antibiotics account For 11% [1][9][19]. One drug is typically the cause of DRESS [5]. medication used to treat seizures (e.g. Lamotrigine, phenytoin, carbazepine, phenobarbital, oxcarbazepine); Infections, common skin disorders (e.g. Vancomycin, trimethoprim sulfamethazole, minocycline, ampicillin levofaxcin, Amoxicillin which are antibiotics) gout (e.g. allopurinol and acne are among the drugs that Frenquently induce DRESS. Also anti TB drugs (e.g. Ethambutanol, isoniazid, pyrazinamide, rifampin) NSAIDs (e.g. Aspirin, celecoxib, diclofenac, ibuprofen, piroxicam cause DRESS syndrome [2][5][9]

Pathology:

Dress Syndrome is not adequately managed Known and is hypothesized to include a Complex interaction. A hereditary weakness in detoxifying enzymes results in the accumulation of drug metabolites, which form covalent bonds with cells, causing cell death and generating secondary immunological abnormalities. Interleukin-s release from drug-specific T-cells can activate both eosinophilic and inflammatory cascades (1).Multiple explanations have been presented for why viral reactivation occurs in Dress. In the acute phase, a highly immunocompromised profile is observed, with fast increase of immune suppressive T regulatory cells (particularly CD4+CD25+FoxP3+) and concurrent declines in immunoglobulins and peripheral B cells. This clonal proliferation may impair the function of antiviral T cells, leading to viral reactivation. Others have proposed that viral reactivation could be caused by immunosuppression corticosteroid
Clinical manifestations:

- Rash: typically maculopopular or morbilliform widespread [2].
- Fever: often high grade fever usually above 38°C [2].
- Lymphadenopathy: enlarged lymph nodes especially cervical [2][5].
- Hematological abnormalities: leukocytosis, atypical lymphocytes, thrombocytopenia [2].
- Reactivation of herpesviruses: reaction of HHV-6, HHV-7 or CMV][3]
- Eosinophilia: elevated eosinophil count in blood [5].
- Facial edema: swelling of the face[5]
- Systemic involvement: hepatitis, nephritis, myocarditis, pneumonitis[10]

Diagnosis:

In order to diagnose Dress syndrome in hospitalized patient’s with a medication rash, the RegiSCAR group proposed criteria. Another set of diagnostic criteria, which includes HHV-6 activity, was proposed by a Japanese group [1][10]. Original diagnostic criteria were proposed by Bocquetetal[2]. These three diagnostic criteria given below.
### Diagnostic Criteria for Dress Syndrome

**Fig 3:** diagnostic criteria for dress syndrome.

Some other diagnostic methods:

<table>
<thead>
<tr>
<th>Diagnostic methods</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Genetic testing</td>
<td>Optional testing to identify predisposing factors or genetic markers associated with Dress syndrome.</td>
</tr>
<tr>
<td>Blood test</td>
<td>Checking for elevated eosinophils, liver enzymes (AST, ALT) and other relevant markers (e.g. CRP, ESR).</td>
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<tr>
<td>Imaging studies</td>
<td>Performing ultrasound, CT scans or other imaging modalities to evaluate organ involvement (e.g. liver, spleen).</td>
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<tr>
<td>Skin biopsy</td>
<td>Obtaining a sample of affected skin for histopathological examination to assess for characteristics features of Dress.</td>
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<tr>
<td>Clinical evaluation</td>
<td>Assessing patient history, symptoms (fever, rash, lymphadenopathy, organ involvement) and medication exposure.</td>
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Treatment:

1. Stop the medication suspected to causing the reaction immediately [1][5].
2. Immunosuppressive therapy:
   - have been treated with high dose of IVIG, cyclosporine [2].
3. To prevent DRESS from returning therapy is gradually reduced over several weeks to months [5].
4. Systemic corticosteroid
   - Prednisone or prednisolone orally or intravenously [10]
5. Provide supportive care to manage symptoms complications
   - Monitoring for organ involvement (e.g. liver, kidney) [16].
   - Fluid and electrolyte management [17]
6. Recognize clinical symptoms and confirm eosinophilia and systemic involvement [17].

Conclusion:

In conclusion, Dress Syndrome, though rare, presents significant challenges in diagnosis and management due to its diverse clinical manifestations and potential for life-threatening complications. Early recognition, prompt withdrawal of offending drugs, and appropriate supportive care are crucial for favorable outcomes in affected individuals. Further research is needed to better understand the underlying mechanisms and improve treatment strategies for this complex syndrome. The review aims to provide the latest info on Dress, a syndrome caused by certain medications. It covers everything from how common it is, to how it happens, what might increase the risk, what symptoms to look for, how to diagnose it, what the outlook is, and how to treat it.

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