Drug-Induced Urticaria: Causes and Clinical Courses

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ABSTRACT

The authors retrospectively reviewed medical records of patients who were diagnosed as having drug-induced urticaria at Siriraj Hospital of Mahidol University (Bangkok, Thailand) between October 2004 and April 2007. One hundred and forty-seven drugs were presumed as causing acute urticaria. Females were affected more commonly than males. The most frequent drug groups were antibiotics, followed by non-steroidal anti-inflammatory drugs (NSAIDs). The most common culprit drugs were ceftriaxone, cephalexin, amoxicillin and diclofenac, respectively. The median duration of onset and of clinical remission were 18 hours and 24 hours, respectively. Antibiotics were the most frequent causes of drug-induced urticaria, of which cephalosporins were the most common causative drugs. Oral NSAIDs significantly had the shortest median onset of urticaria. After discontinuing the culprit drugs, the reactions usually disappeared within a few days.

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INTRODUCTION

The most frequent adverse drug reactions are cutaneous drug reactions, with incidence ranges from 1–3 percent.[1-3] Urticaria has been reported to be the second most common cutaneous drug reaction, following morbilliform reactions.[3-9] It accounts for 5–22 percent of all cutaneous drug reactions.[3-5,7] The most common causative agents were different in each study.[5,10,11]

Drug-induced urticarial rash can occur alone or associated with other symptoms, such as angioedema, systemic symptoms or anaphylaxis. Drug-induced urticaria may be caused by either an immunologic or non-immunologic process. Three mechanisms have been described: 1) immunoglobulin (Ig) E-mediated drug reactions, 2) circulating immune complex-mediated drug reactions (serum sickness) and 3) non-immunologic activations or pseudoallergic reactions.

Although Nettis et al. reported a large number of cases with drug-induced urticaria, they did not discuss the clinical course of individual drug groups.[11] The purpose of this study was to identify the drugs which induced urticaria and the clinical courses of patients in the setting of a large university-based hospital.

MATERIALS AND METHODS

This study was approved by the Siriraj Institutional Review Board, Siriraj Hospital, Mahidol University, Bangkok, Thailand. We retrospectively reviewed data records of patients diagnosed as having drug-induced urticaria at the adverse drug reaction (ADR) center of Siriraj Hospital between October 2004 and April 2007. Patients 18 years of age and above were included in the study. Urticaria, a wheal-and-flare reaction with individual lesions which come and go within 24 hours,[12,13] was diagnosed by attending physicians and dermatologists.

Medical records were reviewed for demographic data, history of previous adverse drug reactions and previous urticaria, clinical characteristics, suspected drugs, clinical features, course, treatment and outcome.

Causality assessment of ADR was carried out by dermatologists and pharmacists at the ADR center. The assessment was classified into six levels: certain, probable, possible, unlikely, unclassified and unclassifiable according to World Health Organization (WHO) guidelines.[14]

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The Mann-Whitney test and Kruskal-Wallis test were used, respectively, for comparison of two independent groups and more than two independent groups. The statistical significance was $P<0.05$.

RESULTS

One hundred and ten patients were enrolled. The demographic data of these patients are shown in Table 1. Mean age of the patients was 44.8 years old with the range of 18 to 82 years old. Most patients had urticarial lesions without angioedema or systemic symptoms. Twenty-four patients (22%) had previous history of urticarial attacks.

One hundred and forty-seven suspected drugs were presumed as causing acute urticaria (according to WHO assessment classification), as shown in Table 2. It should be noted here that one patient might have taken more than one suspected drug. The causality assessments of ADRs were certain, 2.7 percent; probable, 41.5 percent; and possible, 55.8 percent. The median duration from starting the suspected drug until developing urticaria was 18 hours (range: four minutes to 10 days). The median duration from cessation of using suspected drugs to clinical remission was 24 hours (range: 30 minutes to 22.5 days).

TABLE 1.

Demographic and Clinical Data of Patients With Drug-Induced Urticaria (n=110)

Characteristics	No. of patients (%)
Sex	
Female	73 (66.4)
Male	37 (33.6)
Department visited	
Out-Patient Department	54 (49.1)
In-Patient Department	56 (50.9)
Diagnosis	
Acute urticaria	90 (81.8)
Acute urticaria with angioedema	16 (14.5)
Acute urticaria with systemic symptoms	3 (2.7)
Acute urticaria with anaphylaxis	1 (0.9)
History of previous adverse drug reactions (n=107)	
No	73 (66.4)
Yes	34 (30.9)
Urticaria	3 (8.8)
Urticaria with angioedema	3 (8.8)
Angioedema	5 (14.7)
Maculopapular rash	3 (8.8)
Unspecified rash	6 (17.6)
Treatment	
None	8 (7.3)
Topical treatment	24 (21.8)
Systemic antihistamine	99 (90.0)
intravenous	23 (23.2)
oral	35 (35.4)
both	41 (41.4)
Systemic corticosteroids	27 (24.5)
Others	14 (12.7)
Type of oral antihistamines	
H1 antagonists	74 (67.3)
H2 antagonists	4 (3.6)

> This data may help physicians to identify the most suspected drugs causing urticaria in patients who are taking multiple drugs.

Antibiotics were the most frequent cause of drug-induced urticaria in this study. The first three most common suspected antibiotics were ceftriaxone, cephalexin and amoxicillin, respectively. Three common drug groups causing urticaria were antibiotics, non-steroidal anti-inflammatory drugs (NSAIDs) and opioids. The median onset and remission times of urticaria were compared as shown in Table 3. The different route of administration of antibiotics gave a different clinical course (Table 4). Both median onset and remission of urticaria due to intravenous antibiotics were statistically significantly shorter than those of the oral form. Similar results were detected in the opioids group (data not shown in table). The clinical courses of oral drug-induced urticaria were compared in Table 5. NSAIDs had the shortest median onset of urticaria and had statistically significant differences among three common drug groups.

In this study, sixteen patients had urticaria with angioedema. The most frequently suspected causative drug group was antibiotics (50%). Three patients had urticaria with systemic symptoms. The suspected drugs were intravenous ceftriaxone, intravenous cloxacillin and oral norfloxacin. The reactions developed within five to ninety minutes after receiving the suspected drugs.

Only one patient had anaphylaxis. She was 47 years old. Previously, she had taken carboplatin treatment for ovarian carcinoma several times without a problem. At the 9th cycle, after taking carboplatin for forty minutes, she developed urticaria with hypotension. She was treated with intravenous antihistamine and corticosteroids. The urticarial lesions were markedly improved within one day. Causality assessment was possible.

Among patients who had a personal history of atopy (mostly allergic rhinitis), three-fourths had NSAID-induced urticaria.

DISCUSSION

Similar to previous studies, our study showed that drug-induced urticaria occurred predominantly in females.[3,11] Stubb et al.[10], Nettis et al.[11] and Patel et al.[15] reported that NSAIDs were the most frequent causative agents in drug-induced urticaria, whereas Hunziker et al. reported that penicillins were the most

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TABLE 2.

Suspected Drugs that Induced Urticaria (n=147)	
Characteristics	**No. of patients (%)**
Antibiotics	76 (51.7)
Aminoglycosides	
Amikacin	1 (0.7)
Cephalosporins	
Ceftriaxone	11 (7.5)
Cephalexin	8 (5.4)
Cefazolin	6 (4.1)
Ceftazidime	4 (2.7)
Cefditoren	1 (0.7)
Cefepime	1 (0.7)
Cefminox	1 (0.7)
Cefoperazone/sulbactam	1 (0.7)
Cefoxitin	1 (0.7)
Carbapenams	
Imipenem	1 (0.7)
Tetracyclines	
Doxycyclin	1 (0.7)
Tetracyclin	1 (0.7)
Fluoroquinolones	
Ciprofloxacin	4 (2.7)
Levofloxacin	2 (1.4)
Norfloxacin	2 (1.4)
Macrolides	
Roxithromycin	2 (1.4)
Clarithromycin	1 (0.7)
Penicillins	
Amoxycillin	7 (4.8)
Amoxycillin/ clavulanic acid	3 (2.0)
Cloxacillin	3 (2.0)
Ampicillin	2 (1.4)
Ampicillin/ Sulbactam	2 (1.4)
Dicloxacillin	1 (0.7)
Piperacillin/ Tazobactam	1 (0.7)
Others	
Clindamycin	5 (3.4)
Metronidazole	2 (1.4)
Teicoplanin	1 (0.7)

NSAIDs=non-steroidal anti-inflammatory drugs.

Continued	
Characteristics	**No. of patients (%)**
NSAIDs	**28 (19)**
Diclofenac	7 (4.8)
Celecoxib	6 (4.1)
Ibuprofen	5 (3.4)
Indomethacin	2 (1.4)
Meloxicam	2 (1.4)
Aspirin	1 (0.7)
Etoricoxib	1 (0.7)
Mefenamic acid	1 (0.7)
Naproxen	1 (0.7)
Parecoxib	1 (0.7)
Rofecoxib	1 (0.7)
Opioids	**10 (6.8)**
Pethidine	3 (2.0)
Tramadol	3 (2.0)
Codeine/acetaminophen	1 (0.7)
Fentanyl	1 (0.7)
Morphine	1 (0.7)
Tramadol/acetaminophen	1 (0.7)
Muscle relaxants	**4 (2.7)**
Orphenadrine/ acetaminophen	4 (2.7)
Miscellaneous	**29 (19.7)**
Serratiopeptidase	2 (1.4)
Bupivarcain	2 (1.4)
Omeprazole	2 (1.4)
Atenolol	1 (0.7)
Bupropion	1 (0.7)
Bromhexine	1 (0.7)
Carboplatin	1 (0.7)
Cisplatin	1 (0.7)
Clorazepate	1 (0.7)
Cyproterone acetate/ ethinyl estradiol	1 (0.7)
Diltiazem	1 (0.7)
Ergotamine	1 (0.7)
Esomeprazole	1 (0.7)
Magesto	1 (0.7)
Metformin	1 (0.7)

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TABLE 2. (CONT'D)

Continued	
Characteristics	**No. of patients (%)**
N-Acetylcysteine	1 (0.7)
Ondansetron	1 (0.7)
Perindopril	1 (0.7)
Phenytoin	1 (0.7)
Salbutamol	1 (0.7)
Sertraline	1 (0.7)
Tegaserod	1 (0.7)
Theophylline	1 (0.7)
Thymosin α	1 (0.7)
Triamcinolone acetonide	1 (0.7)
Xylocaine	1 (0.7)

frequent causes.[5] In this study, the most common causes were antibiotics (cephalosporins were the most common culprit drugs), followed by NSAIDs and opioids, respectively. Pauvilai et al. also reported that cephalosporins were the most frequent culprit drugs causing urticaria in Thailand.[16] These findings may perhaps be due to the increased use of cephalosporins instead of penicillins in Thailand.

The onset of urticaria is more rapid than that of other cutaneous drug reactions. It occurs from within minutes to 36 hours after drug administration.[17] Our data showed that NSAIDs significantly had the shortest median time to inducing urticaria after oral drug administration. Even though there was no statistically significant difference, opioids had the longest median time to inducing urticaria after drug ingestion, however, usually not longer than a few days. This data may help physicians to identify the most suspected drugs causing urticaria in patients who are taking multiple drugs. In most cases, the rash disappeared within one to a few days after the culprit drugs were discontinued. Our results supported previous data, which indicated that atopic patients had increased risk of developing NSAID-induced urticaria.[18]

Gastrointestinal (GI) and respiratory drugs were the common culprit drugs in the miscellaneous group. Proton pump inhibitors (PPIs) caused three out of seven events among the GI drug group. Although PPIs were used extensively in clinical practice, hypersensitivity reactions (HSRs) were rarely reported.[19] It was not so easy to diagnose PPI allergies because PPIs were commonly used in combination with other drugs, such as antibiotics or NSAIDs, which were the common causes of drug allergies. Therefore, physicians should be aware that PPIs could be a cause of HSRs. The cross-reactivity among PPIs diagnosed by skin prick testing or oral challenge testing had been reported.[20-23] Lobera et al. reported that lansoprazole was a good alternative treatment in omeprazole allergic (type I HSRs) patients when compared with pantoprazole.[19]

Urticaria is one of the opioids' side effects, and commonly occurs by direct mast cell degranulation[24]; skin prick testing (SPT) is useless for diagnosis.[25] Because most reactions were not a true allergy, the management of mild histamine-related adverse effects involves changing the drug to a different class combined with using antihistamines and/or steroids.[24]

This study showed four cases of urticaria induced by combination of orphenadrine and acetaminophen. None of them had a history of NSAIDs allergy. The HSRs of muscle relaxants were mostly reported during the perioperative period. Muscle relaxants were involved in 62–75 percent of anaphylaxis during surgery.[26-28] Orphenadrine citrate is a derivative of diphenhydramine. It has been used as an analgesic and muscle relaxant.[29] Orphenadrine-induced urticaria was rare. As far as we knew, no previous published report existed of orphenadrine-induced urticaria. Acetaminophen is widely used as an analgesic and antipyretic. Acetaminophen HSRs are not uncommon and are mostly associated with aspirin intolerance; however, true allergies are rare.[30,31]

In carboplatin allergy, the incidence of carboplatin HSRs correlated with increase in the number of treatment cycles.[32,33] The

TABLE 3.

Clinical Course of Drug-Induced Urticaria: Comparing Among Drug Groups (Including Intravenous and Oral Administration)

	Drug groups				
Characteristics	**Antibiotics**	**NSAIDs**	**Opioids**	**Miscellaneous drugs**	***P* value***
No. of drugs (%)	76 (51.7)	28 (19)	10 (6.8)	29 (19.7)	
Median onset (hours)	21.99	10.5	30.5	24	0.833
(min, max)	(0.08, 216)	(0.07, 192)	(0.07, 72)	(0.25, 240)	
Remission (hours)	24	48	30.5	48	0.090
(min, max)	(0.5, 480)	(24, 504)	(24, 48)	(1, 246)	

* Compare among antibiotics, NSAIDs and opioids.

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TABLE 4.

Clinical Course of Antibiotic-Induced Urticaria: Comparing Between Routes of Administration

	Routes		
Characteristics	**Intravenous**	**Oral**	***P* value**
No. of drugs	38	38	
Median onset (hours)	3	36	<0.001
(min, max)	(0.08, 72)	(0.33, 216)	
Remission (hours)	23.98	36	0.013
(min, max)	(0.5, 96)	(1, 480)	

reaction rate significantly increased between the sixth to eighth cycle, with the highest rate occurring at the eighth cycle.[34-36] Koshiba et al. reported that incidence of HSRs significantly increased after receiving carboplatin for more than nine cycles.[33] Furthermore, they also reported that patients with ovarian carcinoma had higher incidence of HSRs than patients with uterine carcinoma. The exact mechanism of carboplatin hypersensitivity was unclear, but may occur through either IgE-mediated or non-immunologic processes.[34] Clinical symptoms ranged from only flushing or itching to anaphylaxis.[32] Similarly, our patient developed anaphylaxis at the ninth cycle of carboplatin for ovarian cancer therapy. Sliesoraitis et al. suggested the performance of SPT before the sixth cycle for prevention of carboplatin HSRs.[32]

Corticosteroid HSRs were uncommon; however, a number of reports have been published.[37,38] The reactions were proposed to occur by either immunologic or non-immunologic mechanisms.

In conclusion, antibiotics were the most frequent drugs causing acute urticaria, followed by NSAIDs. Cephalosporins were the most common drugs causing urticaria in Thai patients. Oral NSAIDs had the significantly shortest median time in inducing urticarial reactions. After discontinuing the culprit drugs, the reactions usually disappeared within a few days.

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DISCLOSURES

The authors have no relevant conflicts of interest to disclose.

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TABLE 5.

Clinical Course of Oral Drug-Induced Urticaria (n=92): Comparing Among Drug Groups

	Drug groups				
Characteristics	**Antibiotics**	**NSAIDs**	**Opioids**	**Miscellaneous drugs**	***P* value***
No. of drugs (%)	38 (41.3)	24 (26.1)	6 (6.5)	20 (21.7)	
Median onset (hours)	36	14	48	24	0.019#
(min, max)	(0.33, 216)	(0.98, 192)	(13, 72)	(0.5, 240)	
Remission (hours)	36	48	48	48	0.895
(min, max)	(1, 480)	(24, 504)	(24, 48)	(24, 246)	

*Compare among antibiotics, NSAIDs and opioids.
#Antibiotics vs. NSAIDs, *P*=0.02; Antibiotics vs. opioids, *P*=0.51; NSAIDs vs. opioids, *P*=0.015.

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