Print ISSN: 2319-2003 | Online ISSN: 2279-0780

IJBCP International Journal of Basic & Clinical Pharmacology

doi: 10.5455/2319-2003.ijbcp20140238

Case Report

Clindamycin as trigger of uroporphyrinuria

Mario Stefanini*

Clinch Valley Medical Center, Richlands, Virginia USA

Received: 26 October 2013
Accepted: 14 November 2013

***Correspondence to:**
Dr. Mario Stefanini,
Email: devescogm@gmail.com

ABSTRACT

Administration of clindamycin for the management of a staphylococcal abscess of the abdominal wall was followed by development of a non-pruriginous, macular, blistery rash involving the areas of the body exposed to sunlight. This was accompanied by significant elevation of urinary uroporphyrin possibly related to the ability of clindamycin to inhibit the enzyme CYP3A4 of the P450 system.

Keywords: Clindamycin, Uroporphyrinuria, Rash

CASE REPORT

A 44 year-old man suffering from Gaisböck's syndrome (polycythemia hypertonica) developed an abscess of the abdominal wall. Culture grew *Staphylococcus aureus*, methicillin-resistant, and sensitive to clindamycin. The patient was placed on oral clindamycin (300 mg three times a day). After approximately 48 hours and the administration of six doses of the drug the patient was accidentally exposed to sunlight. He promptly developed a non-pruriginous, blistery red, macular rash involving only surfaces not covered by clothing. Vital signs and physical findings remained unchanged from previous examinations. Chemical parameters, including "hepatic enzymes" (SGOT, SGPT, GGT) and total serum bilirubin were within limits of normal. WBCs were 4550/cu mm with 82% neutrophils, RBCs 5.75 million/cu mm, hemoglobin 15.5 g/dl, hematocrit 46% and platelets 190,000/cu mm.

A sample of urine collected on arrival appeared to be darker than usual in the impression of the patient and fluoresced faintly under Wood's lamp. In this sample the level of uroporphyrin was elevated (Table 1). Clindamycin was discontinued and the rash disappeared approximately six hours later while the patient was being protected from sunlight.

About one week later the patient, who had been otherwise asymptomatic even when exposed to light, was given two doses of 300 mg of clindamycin orally with eight hours interval, after obtaining consent for such a step. Six hours later, this was followed by return of a less dramatic rash upon exposure to sunlight. Again, a random urine sample showed faint fluorescence under Wood's lamp and elevated uroporphyrin level (Table 1).

The rash disappeared after approximately eight hours. Levels of uroporphyrin returned to normal the following

day and there was no return of rash when the patient was exposed to sunlight.

The patient has now been asymptomatic over three months even in sunlight. The abscess of the abdominal wall has been successfully treated with the use of vancomycin.

Table 1: Levels of urinary porphyrins (mcg/L) in random samples of patient's urine before and after administration of clindamycin.

	A	B	C	D	Normal level
Urinary porphyrin	39	3	27	2	0-2
Urinary hepta carboxyl porphyrin	< 1	1.5	< 1	< 1	0-2
Urinary hexa carboxyl porphyrin	< 1	1	< 1	1	0-1
Urinary penta carboxyl porphyrins	< 1	< 1	< 1	1	1-2
Urinary coproporphyrins	17	14	9	13	0-15
Urinary coproporphyrins 3	3	2	3	2	0-99
A = at the time of appearance of rash B = 24 hours later C = 12 hours after clindamycin oral challenge D = 24 hours later					

DISCUSSION

Clindamycin (methyl 7-chloro-6,7,8-trideoxy-6-(1methyl-*trans*-4-propyl-L-2-pyrrolidinecarboxamido)-1-thio-L-*threo*-α-D-*galacto*-octopyranosidemonohydrochloride) is a widely-used antibiotic. Upon oral ingestion or intravenous administration clindamycin binds to plasma proteins, reaching a peak level at about 35-60 minutes. It has a lifespan of two to three hours in the bloodstream and it is metabolized primarily by oxidation.[1] In vitro studies with human liver and intestinal microsomes have shown that clindamycin inhibits the activity of CYP3A4 enzyme in the P450 enzymatic system.

Normally the body uses multiple processes of synthesis in the formation of hemes. Many of these steps involve porphyrins. This process may be interfered with by congenital absence or deficiency of needed enzymatic entities or by interference of the synthetic process by exogenous influences such as drugs.[2] Development of porphyrinuria is known to be triggered by multiple drugs, among them barbiturates, phenantoin, and sulfonamides. The ability of clindamycin to inhibit the activity of one or more enzymes in the P450 system makes it able to induce porphyrinuria. Clindamycin should be added to the list of drugs able to induce transitory porphyrinuria and potentially light sensitivity.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

REFERENCES

1. Wynalda MA, Hutzler JM, Koets M, Podoll T, Wienkers LC. In Vitro Metabolism of Clindamycin in Human Liver and Intestinal Microsomes. Amer Soc for Pharmacology and Experimental Therapeutics. 2003;31:878-87.
2. DeHaan RM, Metzler CM, Schellenberg D, VandenBosch WD, Masson EL. Pharmacokinetic Studies of Clindamycin Hydrochloride in Humans. Int J Clin Pharm. 1972;6:105-19.

doi:10.5455/2319-2003.ijbcp20140238
Cite this article as: Stefanini M. Clindamycin as trigger of uroporphyrinuria. Int J Basic Clin Pharmacol 2014;3:225-6.