# Medicinal mishap

## Possible acute hepatotoxicity from oral clindamycin

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### Case

A 52-year-old woman presented feeling giddy and generally unwell. She complained of episodic upper abdominal pain and headaches.

The patient had a past history of pulmonary embolism and was taking warfarin. She was also taking phenytoin to prevent seizures and long-term amoxycillin for cerebral abscesses. This infection had been slow to resolve so 36 hours before her presentation, clindamycin 450 mg three times daily had been added to her treatment.

Physical examination was unremarkable and her warfarin and phenytoin concentrations were in the therapeutic range. The woman's liver function had been normal before starting clindamycin but was now abnormal:

- alanine aminotransferase 340 U/L (normal range 5–40 U/L)
- aspartate aminotransferase 855 U/L (normal range 5–40 U/L)
- gamma-glutamyl transferase 524 U/L (normal range 12–43 U/L)
- alkaline phosphatase 159 U/L (normal range 30–150 U/L)
- lactate dehydrogenase 714 U/L (normal range 100–230 U/L).

The patient's bilirubin, albumin and alpha-fetoprotein concentrations were normal. Serology for hepatitis B and hepatitis C infection was negative. Apart from a previous cholecystectomy, the liver and biliary tree were normal on a CT scan.

Clindamycin was ceased, but no other changes were made to her drugs. Three days after stopping clindamycin, her symptoms had resolved and her liver function tests were almost back to baseline values.

### Comment

Clindamycin is a lincosamide antibiotic with antibacterial activity against anaerobes, protozoa and Gram-positive bacteria, including community-acquired methicillin-resistant *Staphylococcus aureus* (MRSA). Oral doses have a high bioavailability. Probably for these reasons, clindamycin is widely used. Most clinicians are aware that up to 20% of patients taking clindamycin will experience diarrhoea[1], however hepatotoxicity is less well recognised. Reversible subclinical transaminitis is not uncommon with parenteral clindamycin, however acute symptomatic hepatotoxicity with oral clindamycin is rare. Only one case has been published recently[2], the remaining few coming from the 1970s.[3,4] The recent case occurred with low-dose oral clindamycin while the earlier cases involved parenteral clindamycin. Hepatotoxicity resolved in all cases after stopping clindamycin, but unlike our patient the resolution took several weeks. Liver biopsies showed mixed hepatocellular and portal damage.[2,3] The hepatotoxicity is probably idiosyncratic since patients with underlying liver disease who were given clindamycin had no exacerbation of liver dysfunction.[5]

In summary, acute hepatotoxicity is a rare complication of clindamycin that may be seen more often with its increasing use. Clinicians should have a low threshold for checking liver function in their patients, particularly if they become unwell while taking clindamycin.

### References

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