07 - Wernickes encephalopathy

Wernicke’s encephalopathy

Addictions and substance misuse CHAPTER 4 Carbamazepine is an alternative to a benzodiazepine for managing withdrawal in situations where benzodiazepines are not a safe first-line option.15,16 Examples include: ■ ■A history of adverse reaction or allergy to benzodiazepine drugs. ■ ■A preference for carbamazepine because of a history of harmful or dependent use of benzodiazepines. Wernicke’s encephalopathy Wernicke’s encephalopathy is an acute neuropsychiatric condition caused by thiamine deficiency. In alcohol dependence, thiamine deficiency is secondary to both reduced dietary intake and reduced absorption. Risk factors for Wernicke’s encephalopathy in alcohol dependence are:16 ■ ■acute withdrawal ■ ■malnourishment ■ ■decompensated liver disease ■ ■emergency department attendance ■ ■hospitalisation for comorbidity ■ ■homelessness ■ ■memory disturbance ■ ■peripheral neuropathy ■ ■previous history of Wernicke’s encephalopathy. The ‘classic’ triad of ophthalmoplegia, ataxia and confusion is rarely present in Wernicke’s encephalopathy, and the syndrome is much more common than is recognised. A presumptive diagnosis of Wernicke’s encephalopathy should therefore be made in any patient undergoing detoxification who experiences any of the following signs: ■ ■ataxia ■ ■hypothermia ■ ■hypotension ■ ■confusion ■ ■ophthalmoplegia/nystagmus ■ ■memory disturbance ■ ■unconsciousness/coma. Any history of malnutrition, recent weight loss, vomiting or diarrhoea or peripheral neuropathy should also be taken into consideration.17 Prophylactic thiamine Low-risk drinkers without neuropsychiatric complications and with an adequate diet should be offered oral thiamine. The dose should be 300mg daily during assisted alcohol withdrawal and periods of continued alcohol intake.9 As thiamine is required to utilise glucose, a glucose load in a thiamine-deficient patient can precipitate Wernicke’s encephalopathy.

488 The Maudsley® Prescribing Guidelines in Psychiatry CHAPTER 4 Parenteral thiamine Historically it has been advised that patients undergoing in-patient detoxification should be given parenteral thiamine as prophylaxis against Wernike’s encephalopathy.2,9,18,19 In many countries, there are no licensed forms of parenteral thiamine available. In the UK, NICE20 recommends offering prophylactic parenteral thiamine followed by oral thiamine to those defined as ‘harmful or dependent drinkers’ if they are also known to: ■ ■be malnourished or at risk of malnourishment or ■ ■have decompensated liver disease and in addition: ■ ■they attend an emergency department or ■ ■are admitted to hospital with an acute injury or illness. People at high risk of Wernicke’s encephalopathy can have a range of conditions, including: ■ ■significant weight loss ■ ■poor diet

■ ■low body mass index (BMI) (<18) ■ ■other signs of malnutrition. Consider offering prophylactic parenteral thiamine to people at high risk following the dosing below. If Wernicke’s encephalopathy is suspected the patient should be transferred to a medical unit where intravenous thiamine can be administered. If untreated, Wernicke’s encephalopathy progresses to Korsakoff’s syndrome (permanent memory impairment, confabulation, confusion and personality changes). Treatment of somatic symptoms Somatic complaints are common during assisted withdrawal. Table 4.7 lists some remedies. Community setting doses ■ ■Give intramuscular thiamine 200–300mg once daily for at least 3 days Hospital setting doses ■ ■Give intramuscular or intravenous thiamine 200–300mg once daily for 3–5 days with daily review and monitoring for emergent signs of Wernicke’s encephalopathy

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Revision #1
Created 2026-01-04 20:16:00 UTC by Omar Ayman
Updated 2026-01-04 20:16:00 UTC by Omar Ayman