Extended Abstract

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10th International Congress on Clinical Virology, Fungal Infections & Infectious Diseases , December 04-05, 2017 Dubai, UAE -Antibiotic and mental status change

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Delirium or acute state of confusion is an organically caused decline from a previously attained baseline level of cognitive function, which occurs in up to half of hospitalized patients and up to 80% of patients at intensive care units. Increased length of hospital stays and in-hospital complications such as discharge to long-term care facilities, hospitalization from longterm care facilities, subsequent cognitive impairment, subsequent dependent and risk of in-hospital and 1-year mortality is associated with delirium. A review of medications as a potential contributing factor should always be prompted by any change in mental status. Neurologic symptoms, including sedation, sleep disturbance, confusion, delirium, seizures, mood changes, psychosis, and hallucinations, are often an overlooked etiology of antimicrobial agents. Increased dose of antibiotics and their different classes can change the type and frequency of mental status, concurrent central nervous system (CNS) disorders and renal dysfunction. The most common causative agents, with incidence varying from a few isolated case reports to 15% of patients at the intensive care unit, are fluoroquinolones, cephalosporins and macrolides in cefepime. Over 50% of elderly patients receive high-dose clarithromycin. In view of the fact that, antimicrobial agent use has a high frequency, awareness regarding the potential for antimicrobials to induce changes in mental status should be taken into consideration by clinicians. Therefore, recognition and management may reduce morbidity and patients and families should be appropriately educated regarding these adverse effects. Type 1 was characterized by seizures and most often associated with penicillin and cephalosporins. Type 2 was marked by symptoms of psychosis and associated with procaine penicillin, sulfonamides, fluoroquinolones and macrolides. Both Type 1 and Type 2 had a quick onset of symptoms, within days. Once antibiotics were stopped, symptoms also stopped within days. Type 3 was characterized by abnormal brain scans and impaired muscle coordination and other signs of brain dysfunction, and was only associated with the drug metronidazole. The beginning of noticeable symptoms took weeks instead of days. Symptoms also took longer to go away once the antibiotic was stopped. Bhattacharyya noted that all of the patients had an active infection that could not be ruled out as the cause of the delirium and other brain problems. A scale used to determine whether side effects can be attributed to a drug found that the association was possible in most cases. When infections that affected the central nervous system were not included, the association was probable.

“More research is needed, but these antibiotics should be considered as a possible cause of delirium,” said Bhattacharyya. “Recognition of different patterns of toxicity could lead to a quicker diagnosis and hopefully prevent of some of the negative consequences for people with delirium and other brain problems. Antibiotics are among the most frequently used pharmaceuticals in both the inpatient and outpatient setting. While these antimicrobial agents are generally well tolerated, these drugs are not without their associated side effects, both dose-dependent and idiosyncratic in nature. While diarrhoea is a commonly associated adverse effect of many antibiotics,

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toxic effects on the central nervous system are perhaps much less recognize. A danger for clinicians and patients alike, of not recognizing neurotoxic effects of antibiotics is that the neurological manifestations of toxicity may be confused with a different neurological condition. Correspondingly, in cases of drug-induced encephalopathy, change in mental status may be ascribed to a metabolic abnormality especially in hospitalized patients. With greater education regarding these neurotoxic effects, medical care providers can learn to recognize toxic effects more readily and make medication adjustments as necessary since it is often a readily reversible process. A high degree of suspicion is also essential for clinicians. Many factors of drug metabolism may increase susceptibility to neurotoxicity such as an individual's nutritional status, local blood flow and tissue uptake, and status of the blood-brain barrier, rate of absorption of the medication, route of drug delivery to target tissue, activation and elimination of the drug and its metabolites, as well as protective responses the individual may have. Other factors that may be implicated include genetic factors, altered drug pharmacokinetics in cases of renal insufficiency and central nervous system (CNS) penetration may also be relevant in causing neurotoxicity. In this article, we reviewed the neurologic adverse effects of different classes of antibiotics as they have been described in the medical literature over the last several decades, the potential mechanisms and management strategies. Aminoglycosides have been known to cause ototoxicity most commonly, though peripheral neuropathy, encephalopathy and neuromuscular blockade have also been reported. In the case of gentamicin, one case series outlined peripheral neuropathy and encephalopathy, with nerve biopsy revealing a lysosomal abnormality analogous to those changes seen in gentamicin-induced nephrotoxicity. Other case studies have detailed brain lesions following administration of intrathecal gentamicin, where the patient developed multiple small discrete lesions restricted to the pons and mesencephalon characterized by axonal loss, astrocytic and oligodendroglial loss as well as an inflammatory response. A concurrent experimental study in rabbits resulted in reproduction of similar characteristic lesions that were directly related to brain tissue and CSF concentrations of gentamicin. Aminoglycoside antibiotics are also associated with neuromuscular blockade. Since the original observations were made with streptomycin in patients with tuberculosis, many other aminoglycoside antibiotics have been implicated in neuromuscular and autonomic transmission blockade. They include amikacin, tobramycin, neomycim, gentamicin, and kanamicin. These neuromuscular blocking effects of aminoglycosides have implications in neurological conditions such as myasthenia gravis or Lambert Eaton myasthenic syndrome, where these antibiotics can worsen neuromuscular weakness and thus are contraindicated in these patients. The mechanism of ototoxicity is thought to be the result of excitotoxic activation of NMDA receptors within the cochlea [10]. This results in formation of oxidative radicals, which are postulated to contribute to cell death. Intrastriatal neomycin is shown to cause gliosis that was dose-dependent and diminished when NMDA antagonists were co-administered. It stands that there is a theoretical dose-dependent risk of CNS toxicity with aminoglycosides, particularly in individuals with increased CNS permeability. The mechanism of neuromuscular blockade on the other hand appears to be inhibition of quantal release of acetylcholine in the neuromuscular junction pre-synaptically, and also binding of the drug to the acetylcholine receptor complex post-junctionally. Calcium seems to prevent this suggesting calcium depletion may occur as well

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