From bench to bedside: Colistin nephrotoxicity and vitamin E

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Introduction

By increasing the prevalence of infections caused by multi-drug resistant gram negative bacillus bacteria and mortality due to hospital acquired infections, we are using an old but not forgotten antibiotic called colistin [1-3]. Based on previous studies it was reported that about half of the isolated gram negative bacillus bacteria or even more are resistant to most of the antibiotics in our country and we are facing some problems in controlling and managing them [4,5]. Colistin, a polymyxin structured antibiotic is sometimes our only option. It was first discovered in 1949. By increasing bacterial cell membrane permeability by acting like a cationic detergent in gram negative bacteria colistin could have its bactericidal effect [6-8]. It is eliminated from kidney by renal tubular secretion, and also 80% of secreted medication reabsorbed in tubules [9]. Colistin nephrotoxicity as a serious adverse drug reaction which is mostly reversible but sometimes irreversible need special attention [10]. The exact mechanism of this reaction is not fully understood but, data from clinical studies suggest that oxidative stress and cell apoptosis play a role in acute tubular necrosis due to colistin consumption and this oxidative stress increase activity of Endothelial Nitric Oxide Synthase and Inducible Nitric Oxide Synthase which could lead to apoptosis and necrosis [11,12]. It also causes cellular permeability control dysfunction which could lead to cell lysis and acute tubular necrosis [13,14]. Although the exact mechanism is not recognized yet, it seems Reactive Oxygen Species (ROS) play an important role in tubular cells apoptosis [15]. In animal studies, it was shown before that using an antioxidant like N-acetyl cysteine or ascorbic acid could decrease ROS level an apoptosis [12,16-18]. Using ascorbic acid by dose of 3000 milligrams daily concomitantly by colistin or receive colistin alone. It also causes cellular permeability control dysfunction which could lead to cell lysis and acute tubular necrosis [13,14]. Although the exact mechanism is not recognized yet, it seems Reactive Oxygen Species (ROS) play an important role in tubular cells apoptosis [15]. In animal studies, it was shown before that using an antioxidant like N-acetyl cysteine or ascorbic acid could decrease ROS level an apoptosis [12,16-18]. Using ascorbic acid by dose of 3000 milligrams daily concomitantly by colistin or receive colistin alone. It also causes cellular permeability control dysfunction which could lead to cell lysis and acute tubular necrosis [13,14]. Although the exact mechanism is not recognized yet, it seems Reactive Oxygen Species (ROS) play an important role in tubular cells apoptosis [15]. In animal studies, it was shown before that using an antioxidant like N-acetyl cysteine or ascorbic acid could decrease ROS level an apoptosis [12,16-18]. Using ascorbic acid by dose of 3000 milligrams daily concomitantly by colistin or receive colistin alone.

References


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