Anaesthesia and ARV

RN Rodseth

As more and more HIV infected patients gain access to antiretroviral medication, this drug class and its patients have gained particular significance for anaesthetists. This paper offers an overview of antiretrovirals (ARV) with a specific focus on the implications for anaesthetic management.

The four main classes of ARV’s are as follows: Nucleoside analogue reverse transcriptase inhibitors – zidovudine, didanosine, stavudine; non-nucleoside reverse transcriptase – nevirapine, delavirdine and efavirenz; protease inhibitors – saquinavir, ritonavir, indinavir; fusion inhibitors – enfuvirtide.

Common group side effects are looked at in depth. The NRTI’s lactic acidosis, pancreatitis, peripheral neuropathies and hepatic failure are discussed together with their mechanisms of action and techniques to handle these complications. The protease inhibitors cause extensive inhibition of the cytochrome P450 system that result in a profound extension of many anaesthetic drug half-lives. In addition the PI have a host of metabolic complications including lipodystrophy, insulin resistance and dyslipidemia, which result in patients at a higher risk for cardiovascular morbidity and mortality.

Anaesthetic management of the patients are discussed and the importance of enforcing universal precautions together with ensuring that ARV dosing schedules are maintained is emphasised. An aspect of great concern which is also touched on is post-exposure prophylaxis and in particular the implications when the patient has been on ARV’s and may be carrying a resistant strain of HIV.

Coagulation revisited: Special focus on Prothrombotic states and anticoagulation

S Mayet

Abstract
In the daily practice of anaesthesia, it sometimes happens that a case arises that requires investigation. This is a review of such a case.

A 35 year old female presented with a surgical history of bilateral above knee amputations. A subsequent diagnosis of antithrombin III and protein C deficiency was made. As a consequence of inadequate anticoagulation the patient then presented for amputation of an arm.

In recent years, our understanding of the coagulation cascade and its mechanisms has evolved significantly from a classic separation of “intrinsic” and “extrinsic” pathway to one of initiation, amplification, propagation and stabilisation of the clot. We now have greater insight than ever before on the degradation of clot and how the different mediators like thrombin, antithrombin and protein C and S act on the cascade.

This review aims to provide an overview of the “new” coagulation pathway, consequences of abnormalities in the pathway and where the “new” anticoagulants act on the pathway so that we have an improved understanding for improved patient care.

References