ED QUICK QUIZ

WHAT IS THE DIAGNOSIS?

BACKGROUND

82 year old male brought in via ambulance with increasing drowsiness and lethargy. Family found patient unresponsive in bed this morning. Paramedic crew have GCS at 8 (M: 5 E: 1 V:2).

Patients past medical history includes: IHD, T2DM, Prostate Ca, HTN, and hypercholesteraemia. Currently taking Tamsulosin, Bisoprolol, Metformin, Aspirin, Ramipril, Zomorph and Oramorph.

EXAMINATION

Airway patent. Bradypnea. Heart sounds I + II + 0. Palpable suprapubic mass on abdominal examination. Patient clinically dehydrated. GCS remains at 8 as with the crew. Normal tone present. Pupils 2 non-reactive to light.

OBSERVATIONS

HR : 70, BP : 100/75, RR : 7, SaO2 : 92% on RA, T : 36.5^{o}C, BM : 10.

QUESTIONS

1. What is causative agent of this presentation?
2. What has changed to cause this presentation?
3. How would you manage this patient?

ANSWERS & DISCUSSION

1. Opioid toxicity secondary to accumulation of prescribed long and short acting opioids. A build up opioid plasma concentration has caused respiratory depression and a decreased level of consciousness. Opioids induce respiratory depression via activation of μ-opioid receptors at within the central nervous system including the pre-Bötzinger complex, a respiratory rhythm generating area located in the pons. Complete opioid agonists like morphine and fentanyl affect breathing with onset and offset profiles that are primarily determined by opioid movement to the receptor site, while partial opioid agonists such as buprenorphine action is governed by transfer to the receptor site as well as receptor kinetics, in particular dissociation kinetics.

2. The patient has gone into renal failure, most likely pre-renal but there is the possibility of renal and post-renal failure as well. The patient is taking nephrotoxics and has a diagnosis of Prostate Ca. As opioids are cleared by the renal system as renal clearance decreases a build up of opioids occurs in the plasma, which can rise to toxic levels. The altered pharmacokinetics of opiates in renal failure may result in the accumulation of the parent compound, an active metabolite or both parent compound and active metabolites. Morphine, for example, is metabolised to morphine-3-glucuronide and morphine-6-glucuronide, both of which are renally excreted. Morphine-6-glucuronide, which is more potent than morphine itself, has a half life of about 50 hours in patients with end stage renal failure compared with 3-5 hours in the presence of normal renal function. Patients with renal dysfunction are therefore susceptible to opiate toxicity unless doses are reduced or dosing intervals are lengthened appropriately.

3. Initial management of the airway with the possibility if tolerated of a simple air adjunct. Give O_2 via a non-rebreathe mask. Establish IV access taking a VBG, FBCs, U&E, CK, Bicarbonate, LFTs and Clotting. Give a test dose of Naloxone 400 μgrams. The patient will need a bladder scan and catheter. A CXR should be performed to rule out aspiration and to exclude pulmonary oedema. When the patient responds to initial treatment during their admission a hold of Ramipril and long acting opioids would be appropriate. Daily U&Es, strict input and output monitoring, daily weights, and if no element of overload IVF. Use of non-opioid drugs should be considered and when opiates are necessary, those that tend not to accumulate in renal disease, such as buprenorphine or alfentanil, may be preferred for mild and more severe pain, respectively.