POST-ANAESTHETIC APNOEA : AN EVENT OF THE PAST?

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Introduction

The pattern of emergence from general anaesthesia has changed substantially over the years. Modern anaesthetic techniques enable patients to regain consciousness, respiratory drive and protective airway reflexes within a short span of time. However, delayed recovery of ventilatory function may still occur because of several reasons which may or may not be anticipated by the anaesthesiologist. We present 2 cases of prolonged apnoea following anaesthesia.

Case Report

Case 1

A 26-year-old multigravida, weighing 49 kg was subjected to an emergency lower segment caesarean section for cervical dystocia. She was an unbooked case and her past clinical status was unknown. Initial evaluation revealed her to be a case of rheumatic heart disease with mitral stenosis and aortic regurgitation without evidence of cardiac failure. She was given 6 mg morphine iv and after preoxygenation general anaesthesia was induced with thiopentone 125 mg. Suxamethonium 75 mg was given to facilitate tracheal intubation and anaesthesia maintained with 0.5 per cent halothane, 60 per cent nitrous oxide in oxygen and pancuronium 4 mg. IV fluids were administered conservatively in view of cardiac lesions. There were no untoward events during anaesthesia and haemodynamic parameters remained stable during the hour long surgery. A live healthy male baby was extracted. At the end of surgery the urinary bag was noticed to have only 15 mL of urine. When reversal of neuromuscular blockade was attempted using neostigmine and atropine combination, the patient did not regain consciousness and there was no discernible spontaneous respiratory effort. Hence manual ventilation using the reservoir bag was continued for next 1 hour. Since urine output still remained below 20 mL, she was given lasix and low dose dopamine. She was placed on mechanical ventilatory support. Post operatively a marked rise in blood urea (90 mg/dL) and serum creatinine (2.5 mg/dL) was noticed. As acute renal failure was suspected, diuretics along with low dose dopamine were continued. At this stage further enquiries revealed her to be an old case of chronic glomerulonephritis (CGN).

She exhibited weak respiratory efforts by 12 hours post opera-

tively and soon became fully awake with adequate respiratory drive. Her cardiovascular status remained stable and she was successfully weaned off the ventilator. She continued an uneventful recovery subsequently.

Case 2

A 45-year-old male, known case of chronic renal failure, was posted for left sided ureterolithotomy. He was subjected to peritoneal dialysis 12 hours prior to surgery. On the day of surgery, blood urea, serum creatinine and potassium were 86 mg/dL, 5.0 mg/dL and 4.2 mEq/L respectively. Anaesthesia was induced with thiopentone 150 mg and oro-tracheal intubation accomplished with pancuronium and maintained with oxygen, nitrous oxide and halothane. He received a total of 6 mg of pancuronium and 3 mg of morphine. Surgery proceeded uneventfully and the urine output remained satisfactory. At the conclusion of surgery, reversal of neuromuscular blockade was attempted with neostigmine and atropine combination in standard dosages. There was no respiratory efforts or any response to verbal command, hence manual ventilation was continued with 100 per cent oxygen. After an hour, weak diaphragmatic excursions were observed on the anaesthesia bag which improved following a small repeat dose of atropine and neostigmine. Though he was able to maintain a saturation of 94 per cent, the respiratory rate was 30/minute and head lift was ill sustained. Hence, he was shifted to ICU on spontaneous ventilation with tracheal tube in situ. Over a period of 8 hours, he demonstrated a marked improvement in muscle power and sensorium. He was then given a small dose of neostigmine (1 mg) and atropine (0.6 mg) and the trachea was extubated. He had no complications thereafter.

Discussion

Common causes of apnoea in the post anaesthetic period are, residual narcotics causing central respiratory depression and poor respiratory function because of inadequate reversal of neuromuscular blockade. In addition, hypocarbia induced by hyperventilation, occurrence of medical complications during anaesthesia, such as stroke or embolism, respiratory diseases and occasionally atypical plasma choline-esterases can all contribute towards prolonged apnoea [1].

It is now firmly established that inadequate excretion of myoneural blocking agents can cause pro-

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longed apnoea in debilitated patients suffering from renal disease. In renal failure, protein binding and sensitivity of myoneural junction to muscle relaxants are essentially normal. In addition, volume of distribution of muscle relaxants is not significantly different in normal and anephric patients. Hence their half life is determined by their clearance. As about 50 to 60 per cent of pancuronium is eliminated in urine in the first 24 hours, prolonged paralysis following a single large dose is invariably seen in patients with renal dysfunction. Somogyi et al demonstrated the prolongation of elimination half life from 2.2 hours to 4.3 hours and reduction in clearance of pancuronium from 1.8 L/Kg/min to 0.9 L/Kg/min [2]. Hence it is recommended that in, such cases, maintenance doses of pancuronium should be drastically curtailed and interval between doses be prolonged till an assessed need for a supplement arises based on clinical or monitored criteria.

In such settings, vecuronium and atracurium offer attractive alternatives for achieving neuromuscular blockade. Vecuronium, an isomer of pancuronium, has an interesting response in patients with renal dysfunction. As almost 30 per cent of vecuronium is excreted unchanged in humans, it has a longer half life in patients with poor renal function [4]. One of its metabolites (3-desacetyl vecuronium) which is 70 per cent as potent, is also excreted through urine and thus further potentiates the blockade in renal failure. However, used in small doses, vecuronium is an entirely satisfactory method of neuromuscular blockade in end stage renal disease [4].

Atracurium, though expensive, remains the drug of choice for neuromuscular blockade in renal failure, being totally independent of the kidney for its elimination (Hoffman elimination) [5].

Presence of central respiratory depression, the other major factor, cannot be ruled out in both the instances. Morphine is biotransformed in the liver by phase II pathways and its metabolites, being water soluble, are easily excreted in urine. Retention of such compounds in patients with normal renal function is not harmful [6]. Nevertheless, it is considered prudent to reduce the initial dose of narcotics by 25-50 per cent in renal failure [7]. Fentanyl may be the drug of choice on account of its unchanged pharmacokinetics.

Alternative methods of intra- and post operative analgesia may be considered, with benefit, in patients with severe renal dysfunction. The use of continuous epidural blockade supplemented with light GA, is a much preferred technique. Epidural opioids, like bu-

prenorphine and fentanyl given single shot or continuous through catheter provide post operative analgesia of highest quality, but require high level monitoring.

Once confronted with prolonged apnoea, the anaesthesiologist should consider all the causes while continuing ventilatory and circulatory support. In cases of inadequate reversal of neuromuscular blockade, tracheal extubation should be delayed in order to facilitate mechanical ventilation. Neuromuscular transmission should be monitored depending upon the availability of the equipment, and anticholinesterases may be repeated if indicated. Central respiratory depression can be treated using small titrated doses of narcotic antagonist like naloxone. Large doses of the same may cause reversal of analgesia and hence should be avoided [8].

To summarise, the causes of post anaesthetic apnoea are manifold. Patients with renal failure are especially prone to such episodes because of their unpredictable response to neuromuscular blocking agents or narcotics. The first case manifested apnoea because of renal disease which escaped notice. Second case also had problems despite the awareness of the underlying disease process and judicious use of drugs. Thus, in presence of altered renal physiological state, a judicious choice of anaesthetic technique, and selection of correct non-depolarising muscle relaxants for a general anaesthetic, can be used with advantage.

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