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Case Report

Add on dexmedetomidine in the treatment of severe alcohol withdrawal in a patient of emergency laparotomy

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ABSTRACT

According to American statistics 90% of people drink alcohol at some time in life. The estimated prevalence of alcohol abuse among hospitalized in patients is 20 % and 10- 33 % in patients admitted to the ICU. Approximately 18% of these patients will develop alcohol withdrawal syndrome (AWS) whose symptoms can include physical and psychological manifestations that range from mild to life threatening. Although AWS has been reported in literature in post-operative periods and in intensive care unit, there is less information on treatment and preparing of a patient with AWS, coming for emergency surgical procedure. The surgical stress and deranged liver functions possess an additional challenge to the anesthesiologist. Here we are reporting the successful management of a case of delirium tremens by using Dexmedetomidine in pre, intra and post-operative period in a patient with hollow viscous perforation for emergency laparotomy.

Keywords: Alcoholic withdrawal syndrome, Delirium tremens, Dexmedetomidine, Emergency laparotomy

INTRODUCTION

Alcohol is one of the most frequently abused drugs throughout the world.[1] The American medical association considers alcoholism as a disease and supports a classification that includes both physical and mental components.

Alcohol intake produces changes in the brains structure and chemistry, such as tolerance and physical dependence. These changes maintain the person with alcoholism's compulsive inability to stop drinking and result in alcohol withdrawal syndrome (AWS) if the person stops.

There is a wide spectrum of manifestations of alcoholic withdrawal ranging from anxiety, decreased cognition and tremulousness through increasing irritability and hyperactivity to full blown delirium tremens and withdrawal seizures that typically develop in alcohol dependent individuals in 6-24 hours of their last drink.[2] People who are abusing alcohol tend to have many complications starting from malnutrition to arrhythmias, congestive heart failure, coronary artery disease, gastro intestinal bleeding and ulcerations, infections, liver disease, nervous system impairment and pancreatitis. Here we are reporting the successful management of a case of delirium tremens presenting with hollow viscous perforation for exploratory laparotomy by using intravenous (IV) dexmedetomidine in intra and post-operative intensive care.

CASE REPORT

A 38 years man was admitted to our institution with severe pain in abdomen diaphoresis, tremors and vomiting since 3 days. The patient was restless, agitated, talking irrelevantly. Relatives provided with history of

chronic alcoholism & cigarette smoking for 20 years. On examination pulse rate was 140/minute, BP 156/92mmHg and respiratory rate 36/minute. The patient was icteric with dry tongue. Auscultation revealed bilateral ronchi in all lung fields. The IV access was secured Patient was thoroughly investigated. Significant case related results were as follows.

Erect X- ray abdomen taken which revealed gas under diaphragm. Liver function tests revealed serum billirubin 3.2 mg/dl, serum albumin 2.8 gm./dl and gamma glutamyl transpeptidase 70 IU/lit, aspartate transaminase (AST) 55 IU/L, alanine amino transferase (ALT) 60 IU/L. Coagulation profile revealed International normalized ratio (INR) 1.5. Kidney function tests revealed blood urea 50 mg/dl, serum creatinine 1.2 mg/dl, and serum ions within physiological limits. USG abdomen revealed mild hepatomegaly with free fluid in the peritoneal cavity. ECG revealed tachycardia with normal ST segment. With the confirmation of diagnosis & high risk consent from the relatives, patient was posted for emergency laparotomy.

As patient was agitated IV Lorazepam 2 mg was administered with pulse oximeter monitoring. The patient was calm with this dose. The pulse oximeter and non-invasive blood pressure were connected followed by IV Pantoprazole 40 mg and IV Ondansetron 4mg, IV Glycopyrolate 0.2 mg IV as antisialogogue. IV Dexmedetomidine 50mg slowly was administered over 20 mins. With pulse oximetry, HR and BP monitoring. IV Fentanyl 50mcg was given. Following this preoxygenation was done. Induction with IV Propofol 80mg with loss of eyelash reflex was achieved, plus IV Succinyl choline 100mg.

The airway was secured with no.9 cuffed oral endotracheal tube and the patient was ventilated with O_2:N_2O in 50/50 with sevoflurane 1-2 %.

IV Fentanyl 50mcg was administered. Other monitors like electrocardiogram capnography urinary catheter and temperature probes were connected.

As the patient was in alcoholic delirium immediate postoperative extubation was not considered. Surgical relaxation was maintained with IV Atracurium 25 mg bolus and 5mg top up to a total 45 mg. Intraoperative fluid management was done with dextrose normal saline (DNS) 1.5 lit and ringer lactate (RL) 1.5 lit. Multivitamine infusion containing thiamine 100 mg was given, and Vit. K was injected IM.

Intraoperative surgical diagnosis was anterior duodenal perforation. As the urine output was low and high coloured IV Furosemide 10 mg and 20% Mannitol 100ml was administered to prevent heapatorenal syndrome.

Postoperatively the patient was shifted to the surgical intensive care unit for ventilatory support. In the SICU patient was administered IV Lorazepam 1mg 8 hourly plus Dexmedetomidine 0.5 mcg/kg/hr.

Analgesia was maintained with IV Fentanyl infusion at the rate of 30 mcg/ hour for the first 24 hours which was later reduced to 10 mcg/ hour. The patient was comfortable and calm with normal vitals when extubated at 48 hours and was continued with IV dexmedetomidine 0.3mcg/kg/hr. for next two days after extubation.

DISSCUSION

Chronic alcohol exposure exerts numerous pharmacological effects by means of interaction with various neurotransmitters and neuromodulators. Alcohol abuse causes an imbalance in inhibitory gamma amininobutyric acid (GABA) neurotransmission and excitatory N- Methyl–D–Aspartate (NMDA) receptor stimulation. Chronic alcohol activation of the GABA receptor results in decreased endogenous GABA release and down regulation of receptors.[3] Alcohol also inhibits the activity of NMDA receptors. Overtime this inhibition results in up regulation of receptors sensitivity in a compensatory attempt to maintain homeostasis. Additionally, alcohol has inhibitory effects on the adrenergic system which results in systemic up regulation and decrease regulation of neuronal firing.[3,4] Abrupt cessation of alcohol intake results in drastic decrease in inhibitory signaling for which the bodies endogenous GABA signaling cannot compensate rebound glutamate signaling at NMDA receptors and increased central nervous excitatory tone leading to a state of hyper excitability presenting as the autonomic hyperactivity to alcohol withdrawal syndrome (delirium tremens).[3,5]

AWS can manifest between 24 to 96 hours after abstaining from alcohol. It has been reported in literature in postoperative care.[6] Spies et al have reported a 16 % incidence of AWS after surgery while there was 31% incidence in post- trauma patients.[7] Imdahl et al have shown that in 70% of 672 surgical patient's prophylactic treatment was administered based mostly on a combination of benzodiazepines chlormethimazole, haloperidol, clonidine or ethanol.[6]

Benzodiazepines including lorazepam, diazepam and midazolam are the most commonly used and recommended agents for AWS.[4] Though the benzodiazepines have shown to be effective, increasing doses of BZDs place a patient at a risk of respiratory depression. So in a patient with AWS, BZD monotherapy may not be effective enough to control symptoms and may worsen delirium.[4,8]

Alpha 2 agonist

Both Clonidine and Dexmedetomidine are presynaptic alpha 2 receptors agonists and exert their effect through a negative feedback mechanism. Stimulation of presynaptic alpha2 receptors inhibits the release of nor epinephrine

and result in sympatholysis.[3] Considering Excitatory effect of alcohol withdrawal on adrenergic system, alpha 2 receptor agonist may have role in therapy.

Dexmedetomidine is an 8 times more selective lipophillic derivative of clonidine, for the alpha 2 receptors.[3,5] It was approved by FDA in 1999 for sedation in mechanically ventilated Patients in ICU and for procedural sedation in non-intubated patients for maximal 24 hours. Dexmedetomidine produces a state of cooperative sedation and has an anesthetic, anxiolytic, analgesic and sympatholytic properties.[9] Common adverse event associated with its use include bradycardia and hypotension. Notably however, it does not cause respiratory depression due to its short half-life (2 hours). Dexmedetomidine is administered as a continuous infusion which allows for quick titration.[3]

Rayners et al published a retrospective review of 20 consecutive patient admitted to ICU and administered dexmedetomidine solely for AWS. In addition to dexmedetomidine patient also received lorazepam as needed. Five patients initially received a bolus dose of dexmedetomidine and none experienced any adverse events associated with bolus. A mean dose dexmedetomidine was 0.530 mcg/kg/hr. and mean length of therapy was 49.1 hours. The mean daily lorazepam dose fell 62% from the 24 hours prior to the dexmedetomidine therapy to first 24 hours after.[8]

Dailey et al published a retrospective chart review of 10 patients with AWS who were treated with dexmedetomidine. The mean dose was 0.7 mcg/kg/hr. and the mean infusion time was 50 hrs. After initiation of dexmedetomidine, patient's mean Clinical Institute Withdrawal Assessment for alcohol score decreased from 26+5 to 13+9. The mean diazepam usage 24 hrs. Prior to dexmedetomidine initiation was 13 mg/hr. falling to 3 mg/hr. in 24 hrs after treatment.[10]

In this case the patient was already in delirium tremens, so patient had already given IV Lorazepam 2mg which acts through GABA receptor. It is a logical intervention for abstinence associated CNS excitation. Patient with significant tolerance to alcohol exhibit cross tolerance to GABA mediated effects of BZDs. And the requirement for high doses of BZDs can lead to over sedation, respiratory insufficiency and worsening of delirium.[11] Dexmedetomidine has ability to produce arousable sedation without respiratory depression.[8] Its alpha2 receptor agonistic activity leads to sedation, analgesia, sympatholysis and increased vagal tone.[8] All these characteristic make dexmedetomidine choice of treatment for hyper adrenergic state during AWS. The patient induced with IV Propofol which acts through both GABAergic and glutamatergic pathways in the CNS is effective in the management of BZD refractory AWS.[8]

CONCLUSION

AWS remains the widespread problem in the hospitalized patients. Although the BZDs remain the mainstay of treatment, the ability of dexmedetomidine to provide sedation and reduce autonomic hyperactivity with potentially less respiratory distress and delirium tremens makes a better choice. It also helps to reduce the requirement of other anesthetic agents and their adverse effects. Arousable sedation without respiratory distress helps to minimal duration of ventilator support and early extubation. Hence dexmedetomidine can be used as adjuvant to the standard care of AWS.

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