

CORRESPONDENCE

Intramyometrial terlipressin in atonic postpartum hemorrhage: A uterine salvage decision

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Terlipressin is a long acting analog of vasopressin mainly used in the treatment of upper gastrointestinal bleeding with minimal cardiovascular effects.¹ We report a case where intra myometrial terlipressin effectively controlled post partum hemorrhage (PPH) refractory to conventional uterotonic drugs.

A 27 year old primigravida parturient was posted for emergency cesarean section (CS) in view of fetal bradycardia under general anesthesia. Rapid sequence induction with cricoid pressure application was performed with thiopental sodium and paralysis achieved with succinylcholine. Anesthesia was maintained with oxygen-nitrous oxide-isoflurane with atracurium keeping minimum alveolar concentration at 0.7-1. She developed PPH after CS and went into hemorrhagic shock with invasive arterial blood pressure (ABP) of 66/40 mmHg and heart rate of 130 /min. Traumatic PPH and gross coagulopathy were excluded from detailed local exploration and previous routine coagulogram (normal platelets count and prothrombin time). No history of any comorbidity or coagulation disorder was noted in her antenatal record. Oxytocin 25 units in 500 ml saline was started after delivery of the baby in titrated fashion. The surgeon reported a non-contractile uterus which did not respond to 25 U oxytocin by intravenous infusion. Intramuscular ergometrine 0.2 mg was injected after placental removal. Manual uterine massage was performed and clots from the uterus were removed by obstetrician. Hypotension was managed with crystalloid and hydroxy ethyl starch. Three units of packed red blood cells and 1gm of tranexamic acid were given. A total of 4 litres of fluids including blood products was transfused. After resuscitation, ABP improved to 90/50 mmHg and heart rate to 115/min. Forced air warming was used to prevent hypothermia. Injection 15 methyl PGF₂α (Carboprost™) 250 µg was given intramuscularly with a repeat dose injected into myometrium. Even after

two repeat doses of Carboprost™ (both intramuscular and intramyometrial) uterus remained atonic. Rectal 1000 µg misoprostal also failed to improve the uterine tone. Uterine tamponade was also unsuccessful. Uterine artery ligation was performed but due to continued bleeding, a consent for hysterectomy was taken. In the mean time we decided to give a trial of intramyometrial 1 mg terlipressin (Remestyp™, Ferring Pharmaceuticals Pvt. Ltd. Mumbai, India) injection after taking consent from relatives. Surprisingly the uterine tone improved after 5 min of terlipressin injection. We repeated second dose after 20 min and with this second dose uterus became more firm and PPH was controlled. So hysterectomy was deferred and patient was shifted to high dependency unit. Her vital signs improved further with fluids and blood products. She was discharged home 7 days after CS.

Intramyometrial vasopressin has been used in myomectomy operation due to its potent vasoconstrictive action, but it is associated with serious adverse effects like hypotension and severe bradycardia.^{2,3} Terlipressin when applied regionally is metabolized to lysine-vasopressin by endopeptidases and provides vascular smooth muscle constriction for longer duration as compared to vasopressin. It has less cardiac effects due to higher affinity for V1R vasopressin receptors. Intrauterine route reduces its systemic side effects and have minimum effects on postpartum breast feeding. Carboprost is usually effective to stop bleeding when used intramyometrial route but in our case it failed probably due to undiagnosed chorioamnionitis or hidden coagulopathy (conventional coagulation parameters were normal but thromboelastography may be abnormal) where vasopressin is better choice. We used terlipressin over vasopressin to avoid serious side-effects.³⁻⁵ So, this new indication of terlipressin needs to be validated by further trials with large number of patients to assess the clinical efficiency in PPH

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'Lignocaine stylet': Tips for difficult arterial cannulation with minimum arterial spasm

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Commonly used methods for arterial cannulation are over the guide wire or over the needle technique.¹ First attempt is the best attempt in arterial cannulation. In many conditions, it is not possible to cannulate artery in first attempt. Puncture site hematoma, spasm of artery result in disappearance of pulse and it creates difficulty in immediate further attempts. We describe a simple technique to overcome the difficulty related to repeated attempts in arterial cannulation.

We attempted radial artery cannulation in left hand with 20G cannula (BD Venflon™ Pro IV Cannula, Becton Dickinson, Helsingborg, Sweden) in a patient with pancreatitis. First attempt was unsuccessful in negotiating the cannula into the artery. With repeated attempts radial artery went into spasm and pulse disappeared. Before trying to another artery we applied a simple modification to the technique in the same artery. We punctured the artery with a new cannula after distal white stopper removal (Figure 1) at the previous puncture point.



Figure 1: Cannula with separated distal white stopper

As blood came into the hub, we tried to negotiate the catheter as much as possible till free arterial flow was there. Then we attached 2cc syringe filled with preservative free

2% lignocaine to distal hub of the cannula with or without stylet (Figure 2) and injected 1-1.5ml into artery and left the cannula for 45-60 sec.



Figure 2: Preservative free lignocaine filled 2 ml syringe at the distal end injecting as lignocaine stylet



Figure 3: Cannula with stylet without stopper. Small drops of blood trickling without pulsatile flow

We could able to thread the cannula into the artery after the stipulated time successfully. Some people remove stylet to check arterial flow after puncturing artery with cannula. It causes profuse bleeding and soiling of the bed

as sometimes it is difficult to reintroduce the stylet into cannula. To overcome this problem our suggestion is to remove the white stopper from the distal end of cannula and withdraw the stylet only a few millimeter before reducing angle to check free blood flow and threading while keeping stylet inside cannula. If the cannula with stylet is inside the artery, continuous small drops of blood will trickle from the stylet opening due to arterial pressure effect (Figure 3) but no trickling if the cannula is inside the vein. Thus it reduces blood loss with minimum soiling. We describe our modification as 'Lignocaine stylet' technique.

A "liquid-stylet" created by a slow arterial injection of saline through the catheter from distal end attached with saline filled syringe, has also been shown to be effective for insertion of arterial cannula.² Different methods of identification of arteries like, hypodermic needle localization,³ and last ultrasound-guided arterial puncture

have also been reported.⁴ But none will be effective when artery is in spasm. We have applied 'Lignocaine stylet' method in different arteries during difficult cannulation and got favourable results. It has been reported that vasospasm of carotid artery during aneurysm embolization surgery can be relieved by lidocaine infusion.⁵ Lignocaine can cause vasodilatation by blocking sodium channel in sympathetic nerves as well as release of nitric oxide from endothelium.⁶ This method may also be helpful in children, infants and neonates with the use of 24G or 26G cannula and small volume of lignocaine 1% solution to limit the toxic dose. We have tried with other different agents like papavarine, nimodipine but lignocaine is promising and easily available with rapid onset of action.

So, we suggest anesthesiologists and intensivists to try this technique in spasmodic artery with difficult cannulation.

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Central venous catheter insertion site infection leading to subcutaneous emphysema

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Key words: subcutaneous emphysema; skin wound; ball-valve mechanism

A 20-year-old lady presented to our hospital with pain and distension of abdomen, feculent vomiting and inability to pass faeces and flatus since 15 days. She looked sick, was febrile, and had a pulse rate of 140/min, blood pressure 76/40 mmHg, respiratory rate 34/min and SpO₂ 87% (room air). On auscultation, chest had bilateral crepitations. Abdominal examination revealed guarding and rigidity with absent bowel sounds. A diagnosis of intestinal obstruction with septic shock was made. Oxygen therapy, fluids and noradrenaline infusion was started. A central venous catheter (CVC) was inserted in right subclavian vein. Standard

general anesthesia was administered for laparotomy. The small bowel had adhesions, stricture and a perforation for which loop ileostomy with urobag laparostomy was done. After completion of surgery, in view of her poor general condition and unstable vitals, anesthesia was not reversed and she was shifted to the ICU for further management. In the ICU, she received ventilatory support (SIMV+PS), broad spectrum antibiotics, vasopressors, parenteral nutrition and other supportive care.

On tenth day, a slight swelling was noted primarily around the CVC insertion site, right upper chest and neck. On palpation there was a crackling sensation under the skin suggesting subcutaneous emphysema (SE). She

correspondence

had no chest pain or discomfort. No change in airway pressures and other vital parameters was noted. The chest radiograph showed evidence of SE involving the right chest wall and neck, with no evidence of pneumothorax or pneumomediastinum (Figure 1). The right subclavian vein cannulation was done ten days ago and no subsequent intervention was done. On examining the CVC insertion site, a small skin wound around the CVC was seen (Figure 2).

The CVC was removed. Blood samples, CVC tip and wound swab were sent for culture. Wound was cleaned and air tight surgical dressing was done. Left internal jugular vein was subsequently cannulated. Blood culture showed no growth. *Acinetobacter* species was isolated from both CVC tip and wound cultures. Antibiotics were changed according to sensitivity. There was no further increase in SE and it subsequently resolved over two weeks.

Perhaps, in our case, air was sucked in through the small skin wound around the CVC insertion point due to negative

intrathoracic pressure being transmitted to the perivascular interstitial space. A large amount of air may have entered the soft tissues due to a ball-valve mechanism.¹ The absence of barriers in the subcutaneous tissues throughout the body allow free movement of air under the skin.²

Cutaneous ulcers and small skin wounds can cause SE.¹ There are reports of SE involving the upper limb following trivial laceration on the dorsum of hand³ and elbow⁴, air being trapped from the skin wound due to a flap valve and a ball-valve mechanism, respectively. The treatment is directed at the underlying cause; subcutaneous air eventually gets absorbed over time. However, massive SE leading to respiratory embarrassment⁵ and requiring insertion of subcutaneous drains has been reported.⁶

To conclude, CVC insertion site infection may be a potential cause of SE and daily inspection for signs of infection should be done. Transparent bio-occlusive dressings protect from infection and allow inspection without the need to remove dressing.

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Counselling is important in treatment of deliberate self-harm.

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The article on poisoning with amitraz was an interesting read.¹ The authors have reported a case of deliberate self-poisoning with amitraz; detailing its presentation with an approach to its management. They have rightly indicated that there are no known antidotes. In this case though there was a suspicion of the agent used, it might not always be the case. In poisoning, when the agent is unknown, the clinical syndrome of symptoms and signs may suggest one of the recognizable toxidromes, which can guide treatment; resuscitation, of course, remains an

important component of the management.^{2,3} It should be recognized that an accurate history might not be gathered at presentation to the emergency room.^{4,6} When etiological agent is known or is subsequently revealed, specific antidotes may be available and these offer the opportunity to reduce morbidity and mortality.² Importantly, in cases of deliberate self poisoning, treatment should not end with decontamination and reversal of symptoms alone. A routine referral and follow up with a psychiatrist is to be encouraged.

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Endotracheal tube cuff leak: yet another unusual cause!

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Despite routine practice of pre-use visual inspection and testing of ETT for physical defects, some manufacturing defects still go unnoticed and may lead to partial or complete failure of ventilation in intubated patient.¹⁻⁴ A 36-year-old female, ASA physical status I, weighing 45 kg, was scheduled for elective laparoscopic cystectomy for ovarian cyst. Standard GA and monitoring techniques were employed. After routine pre-use check for any visible physical defects, a 7.5 mm internal diameter cuffed ETT (InTube® Intersurgical Ltd., UK) was selected for orotracheal intubation. Correct ETT position was confirmed by end-tidal carbon dioxide (EtCO₂) trace. A close circle system was used and ventilator adjusted to achieve effective oxygenation (SpO₂ ≥ 95%) and ventilation (EtCO₂ 35-40 mmHg) with volume-controlled mode. Pneumoperitoneum with carbon dioxide was created and intra-abdominal pressure was held at 12 mmHg. Patient was then placed in Trendelenberg's and lithotomy position. Within few minutes after the commencement of surgery, we noticed the collapsing ventilator bellows and air leak from the mouth as evident by a bubbling sound. Surgery was stopped and pneumoperitoneum released. Patient was placed in supine position and ETT position was confirmed by chest auscultation and ETT marking at the level of the lips. The pilot balloon was found partially deflated and was re-inflated with air. However, after few minutes, air leakage with a deflated pilot balloon was noticed again. Suspecting a possible damage of the ETT cuff or inflation assembly,

the ETT was immediately replaced by a fresh ETT of same size. Subsequently no further problems were observed and the surgery was completed uneventfully.

We checked the removed ETT by inspecting and inflating the cuff but could not find any visible defect in the cuff or in the inflation assembly including pilot balloon. Then the tube was immersed in water to identify the exact point of air leakage. We observed a continuous air leakage, visible as bubbles, from the distal most end of the inflation channel (Figure 1).



Figure 1: Arrow showing air leakage from distal end of ETT

correspondence

Air leakage around the cuff may be seen due to inadequate cuff inflation or use of a smaller tube. It may be seen in clinical situations such as cephalad migration of ETT,² tracheal misplacement of nasogastric tube,⁵ or high peak airway pressures.⁶ Rarely, air leakage may be seen due to structural defects in the cuff and/or cuff inflation assembly.¹⁻⁴ The resultant inadequate tidal volume may necessitate immediate ETT replacement.² In our case, an increased intra-abdominal pressure secondary to

pneumoperitoneum coupled with Trendelenburg plus lithotomy positions may have increased the peak airway pressure and subsequent intracuff pressure leading to cuff leakage from the defective inflation channel.

It was noticed that even after proper pre-use check, some untoward manufacturing defects may still go unnoticed leading to unwanted consequences.

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Pulmonary calcification or...?

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Key words: Pulmonary calcification; Aspiration pneumonia; Barium swallow.

The presence of diffuse pulmonary calcific lesions in a critically ill patient with respiratory difficulties raises suspicion of potentially serious underlying conditions. However, this need not always be the case.

A 62 year old male was admitted to intensive care with worsening dyspnoea and progressive hypoxia. He had had nasopharyngeal carcinoma 10 years back and had received radiotherapy, which led to pharyngeal damage, swallowing difficulty and recurrent aspirations requiring a percutaneous gastrostomy feeding tube. Provisional diagnosis was pneumonia secondary to aspiration and infection. In addition to changes suggestive of pneumonia, the chest x-ray also showed dense, speckled calcified opacities within the lower zones bilaterally (Figure 1). The opacities were caused by residual barium from aspiration after an oral contrast study done over a year ago.

Aspiration is a well-recognised complication of oral contrast studies. Barium sulphate is inert and relatively insoluble, therefore can remain within the lung tissues for a prolonged period of time. Aspirated barium tends to gravitate and hence is seen predominantly in the basal



Figure 1: Opacities marked by arrows

regions. It is phagocytosed by alveolar macrophages and may lead to local fibrosis. Barium aspiration can be acutely fatal both due to physical obstruction and pulmonary inflammation.¹ However, if the patient survives the

initial phase, the barium itself does not lead to significant problems with gas exchange.

Differential diagnosis of bilateral 'diffuse' pulmonary calcification includes healed varicella pneumonia, pulmonary silicosis, amyloidosis, pulmonary alveolar

microlithiasis, metastatic calcinosis and pulmonary interstitial ossification.² It was interesting that the opacities in this case were reported as 'calcification' by a radiologist, who did not have the benefit of the full history.

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OBITUARY

Germán Amaya Ochoa

Dr. German Amaya Ochoa died on Aug 5, 2014. He was a founding member of the Colombian Association for the Study of Pain (ACED) in 1988 and served as ACED's President twice, during the terms 1993-1996 and 1998-1999. He was a co-founder and first president of FEDELAT, a Latin American federation of regional chapters. He remained a member of the International Association for the Study of Pain (IASP) since 1988, and was active in the IASP education initiatives working group, and a strong advocate for changes in pain management policy in Colombia.

Upon completion of international training in Mexico City, the United States, and Europe, Dr. Ochoa returned to Colombia and started to work in the pain field in 1986 as a spine surgeon and pain specialist. In 1986 he and other pain specialists founded a Back School Clinic in Bogotá and worked on acute and chronic pain, with an emphasis on low back and neuropathic pain.

A publisher of numerous articles, chapters, and books on pain management, Dr. Ochoa coordinated the meeting of FEDELAT with the Ministers of Health from the Latin American countries and members of the Community of Andean Nations in 2007 in an effort to improve pain treatment in Latin America.

May his soul rest in peace!

