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Original Articles

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Editorial

Happy New Year dear colleagues.

Precision of medical interventions usually result in safer predictable and better outcomes in the perioperative space. The ability to measure the physiological response of a patient to pharmacological exposure and noxious stimuli is key in ensuring the conduct of a safe anaesthetic, patient's comfort and ideal operating conditions in the face of coexisting life threatening systemic disease. In this edition, we highlight evacuation of a subdural hematoma under sedation with entropy monitoring in a patient with attendant end stage renal disease.

Aortic aneurysmal surgery is fraught with many potential complications which are multisystemic either due to preexisting illness or resultant from the surgical and anaesthetic intervention: in this issue we discuss the diagnostic dilemma of

coagulopathy and thromboembolic phenomena that ensued prolonged aneurysmal surgery.

Physical manifestation of hereditary medical conditions may arise at various ages and will occasionally demand repeated surgical intervention and resultant challenges for the anaesthetic team as highlighted in a case report on the anaesthetic challenges present in Von Recklinghausen disease.

Acute pancreatitis as well as pancreatic surgery have long been known to have an association with thoracic complications, the most feared being acute respiratory distress syndrome (ARDS). Often, these thoracic complications manifest early in pancreatic disease though the exact mechanism is not yet well understood. ARDS may also present well after disease onset and still have other predisposing factors as highlighted in this edition.

Living in a world where resources are scarce means that we have to be innovative and leverage on technology to maximize on what we have available in a bid to ensure the safe practice of medicine. The use of telemedicine to facilitate real-time patient reviews, patient ward rounds and order procedures is one method of bridging this gap in access to the specialist human resource: this is covered in the article of Tele-ICU with a broad discussion on its use, draw backs and medico-legal challenges.

On matters transition, I would like to welcome the incoming editor-inchief, Dr. Idris Chikophe, who will be taking over to run the KJACCM and inject fresh impetus into the journal. I would like to appreciate all the editorial board, article reviewers, readers, advertisers and contributors who have made the KJACCM a veritable success.

Entropy Monitored Sedation For Burr Holes And Evacuation Of Chronic Subdural Hematoma In A Patient With End Stage Renal Disease

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Abstract

Introduction

Assessment of depth of anaesthesia is the mainstay of anaesthetic practice. Entropy measurement is an objective way of assessing both hypnotic levels (State Entropy- SE) and state of analgesia (Response Entropy -RE) during anaesthesia.

Objective

The objective of this case report was to demonstrate the utility of entropy in monitoring anaesthetic depth during monitored anesthetic care for high risk patients.

Materials and Method

This is a case of a 58-year old male patient with end-stage renal disease, diabetes and hypertension who was diagnosed with right sided chronic subdural hematoma. Given the increased risk of morbidity associated with general anaesthesia in this case, monitored anaesthetic care with entropy (State and Response) was used. Pulse rate, blood pressure, respiratory rate and oxygen saturation were also monitored and recorded.

Results

After scalp block and sedation, there was a general reduction in both state (SE) and response entropy (RE). The maximal reduction in RE and SE was by 63% and 57% from the baseline. There was a general reduction in blood pressure, heart rate and respiratory rate, the nadir of which were within physiological limits.

Conclusion

Entropy with monitored anaesthetic care may provide a safe form of intraoperative management in a high-risk patient where general anaesthesia is a relative contraindication.

Background

Chronic Subdural Hematoma is an encapsulated collection of blood between the dura mater and the arachnoid. (Adhiyaman, Asghar, Ganeshram, & Bhowmick, 2002) Trauma to the brain is the commonest cause of chronic subdural hematoma. Other systemic factors like use of anti-platelets (clopidogrel and aspirin) may increase the risk of this disease. Ageing is associated with shrinkage of the brain and stretching of the small veins located between the brain surface and the dura mater. Once these small veins tear or break, the blood leaks over time to form a hematoma. Depending on size and location, the hematoma manifests with the symptoms and signs which vary from simple headache to confusion and loss of consciousness with severe brain damage in some cases

Anaesthesia and surgery for chronic subdural hematoma drainage may be associated with high risk of peri-operative morbidity especially in the setting of co-existing diseases like end stage renal disease. The anaesthesia practitioner has to ensure peri-operative safety of the patient by choosing the most appropriate mode of anaesthesia.(Singh, Bansal, Kumar, Gupta, & Thakur, 2017) In this case, entropy monitoring and conscious sedation for awake burr holes and evacuation of chronic subdural hematoma was used.

Entropy Monitoring

Anaesthesia involves the reversible pharmacologic depression of the neuro-endocrine system leading to loss of response and reaction to noxious stimulus (surgical incision). The objective is to render loss of awareness, analgesia, immobility and blunting of autonomic reflexes.(Brown, Lydic, & Schiff, 2010) An ideal anaesthetic agent would fulfil all the above; in addition to easy titratability to achieve desired levels of anaesthesia.(Eger, 2004) Both light and deep anaesthetic are undesirable. Light anaesthesia is associated with increased risk of awareness and pain. Very deep anaesthesia may lead to cardio-medullary depression.

Entropy is an anaesthetic depth monitoring modality. It involves the processing of electro-encephalography (EEG) and frontal electromyography (FEMG) signals into digital values, state (SE) and response (RE).(Viertiö-Oja et al., 2004) RE is based on both EEG and FEMG and indicates patient's response to external stimulus. It may signal early awakening. SE is based on EEG and indicates the hypnotic sate of the patient. It may signal awareness. RE is always higher than or equal to SE. Entropy analysis has a high specificity and sensitivity in assessing the depth of anaesthesia.(Singh et al., 2017)

Table 1: Entropy Values

RE	SE	Comment
100	90	Awake
60	60	Low probability of recall
40	40	Clinically adequate for most surgical procedures
0	0	Burst suppression

Materials and Methods

Case Presentation

A 58-year old male patient with end stage renal disease (on maintenance dialysis), diabetes and hypertension; was admitted for pre-kidney transplant work up. He complained of headache, confusion and left hemiparesis for one week. His previous surgical and anaesthetic history included burr holes and evacuation of a right sided chronic subdural hematoma four weeks prior to the current admission.

Figure 1: CT Scan of the Brain



The patient was scheduled for evacuation of the subdural hematoma. Additional investigations revealed a haemoglobin of 12.3g/ dL, urea of 64.31 mg/dL and creatinine 5.37 mg/dL. His serum sodium concentration was 125 mol/L, potassium concentration of 5.07 mol/L and chloride concentration of 96.1 mol/L. His pulse rate was 83 per minute with a blood pressure of 190/90 mmHg. His respiratory system was unremarkable.

Intraoperative Management

Standard American Society of Anaesthesiologists intraoperative monitoring (electrocardiography, non-invasive blood pressure and pulse oximetry) was used. In addition, entropy electrodes were attached on the right side of the scalp. 2 litres per minute of supplemental oxygen was administered via nasal prongs.

50 mcg of intravenous fentanyl was given after which a scalp block was performed on the right side. Supraorbital, supratrochlear, zygomaticotemporal, auriculotemporal and posterior auricular nerves were blocked with a mixture of 15 millilitres of 0.5% bupivacaine and 15 millilitres of 2% lidocaine with adrenaline. An average of 3 millilitres of the local anaesthetic mixture was given per nerve.

A loading dose of 1 mcg kg-1 of dexmedetomidine was infused intravenously over a period of 10 minutes followed by a maintenance dose 0.5 to 0.7 mcg kg-1 hour-1. The rate of infusion was increased to 0.7 mcg kg-1 hour-1 if the response entropy (RE) value went above 40 and reduced to 0.5 mcg kg-1 hour-1 with RE below 40.

Table 2: Intraoperative Vital Trends

	After Scalp Block		During loading with Dexme- detomidine (variability from original value)		Dexme- detomidine maintenanc infusion (variability from origin value)	e al	Post opera- tively (variability from original value)	
Pulse Rate (bpm)	83	72 (-15 to +4)		68 (-10 to -15)		67 (-14 to -19)		
SPO2 (%)	95	99 (+4)		100 (+5)		99 (+4)		
sBP (mmHg)	190	154 (-20%)		136 (-28%)		146 (-18%)		
dBP (mmHg)	90	88 (-2%	%)	69(-23%)		82(-9%)		
RR (bpm)	18	16(-2)		15(-3)		20(+2)		
Entropy RE	100	57(-43)		37(-63)		-		
Entropy SE	90	41(-49))	33(-5	57)	-		

Depth of Anaesthesia Assessment

Both response (RE) and state (SE) values followed a decreasing trend following infusion of dexmedetomidine. The RE lowest value recorded was 37 (from 100), while that of SE was 33 (from 90). There was a concurrent reduction in pulse rate, respiratory rate, both systolic and diastolic blood pressure. However, their nadir values were well within physiologic values.

Discussion

General anaesthesia involves the pharmacologic depression of the central nervous system to achieve a state of analgesia, amnesia, immobility and loss of awareness.(Brown et al., 2010) Its wide application in intra-operative care is related to its routine use and ease of monitoring. The drawbacks of general anaesthesia include the need for advanced airway management and the potential for adverse drug interaction and altered pharmacokinetics and pharmacodynamics in the setting of organ dysfunction e.g. chronic kidney disease. (Gottschalk, Aken, Zenz, & Standl, 2011)

Alternatives to general anaesthesia include local and regional anaesthesia; and monitored anaesthetic care. The need for advanced airway management is greatly reduced when these methods are employed. Assessment of adequacy of local and regional anaesthesia may be not be as easy.

Monitored anaesthetic care (MAC) involves the use of local or regional anaesthesia and sedation. Traditionally, monitoring during MAC was through observing clinical parameters such as response to voice, surgical stimulation and pain. Clinical parameters as the sole method of monitoring may be associated with lack of predictability as far as anaesthetic depth is concerned. More direct and reliable method of monitoring anaesthetic effect of a drug on the brain is desirable. The use of modalities such as entropy together with vital signs makes it easy to monitor the depth of anaesthesia and guide the titration of anaesthesia to the desired effect. (Singh et al., 2017) This is even more important in patients with unpredictable response to anaesthesia as a result of the primary disease or concurrent significant organ dysfunction.

In this case, Monitored Anaesthetic Care (MAC), under routine vital sign and entropy monitoring was safely employed in a patient with chronic subdural hematoma and end stage renal disease. This was used as an alternative to general anaesthesia.

Conclusion

Monitored anaesthetic care using entropy and routine vital sign may be a safe alternative to general anaesthesia in high risk surgical patients.

- Adhiyaman, V., Asghar, M., Ganeshram, K. N., & Bhowmick, B. K. (2002). Chronic subdural haematoma in the elderly. Postgraduate Medical Journal. https://doi.org/10.1136/ pmj.78.916.71.
- Brown, E. N., Lydic, R., & Schiff, N. D. (2010). General anesthesia, sleep, and coma. New England Journal of Medicine. https://doi. org/10.1056/NEJMra0808281.
- Eger, E. I. (2004). Characteristics of anesthetic agents used for induction and maintenance of general anesthesia. American Journal of Health-System Pharmacy. https://doi.org/10.1093/ ajhp/61.suppl_4.s3.
- Gottschalk, A., Aken, H. Van, Zenz, M., & Standl, T. (2011). Is Anesthesia Dangerous? Deutsches Aerzteblatt Online. https:// doi.org/10.3238/arztebl.2011.0469.
- Singh, S., Bansal, S., Kumar, G., Gupta, I., & Thakur, J. R. (2017). Entropy as an indicator to measure depth of anaesthesia for Laryngeal Mask Airway (LMA) insertion during sevoflurane and propofol anaesthesia. Journal of Clinical and Diagnostic Research. https://doi.org/10.7860/JCDR/2017/27316.10177.
- Viertiö-Oja, H., Maja, V., Särkelä, M., Talja, P., Tenkanen, N., Tolvanen-Laakso, H., ... Meriläinen, P. (2004). Description of the EntropyTM algorithm as applied in the Datex-Ohmeda 5/5TM Entropy Module. Acta Anaesthesiologica Scandinavica. https:// doi.org/10.1111/j.0001-5172.2004.00322.x.

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DVT In A Patient With Prolonged Routine Coagulation Tests And Low Platelets Counts Post Abdominal Aortic Aneurism Repair

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Abstract

This is a case a of 76-year-old woman who presented with ruptured infrarenal abdominal aortic aneurism (AAA). After open surgical repair of the aneurysm, she was diagnosed with bilateral deep venous thrombosis despite coagulopathy and thrombocytopenia, reaching nadir on 4th day postoperative (platelet count= 44*10⁹L⁻¹, PT= 16.0 sec, INR=1.213, fibrinogen=457, APTT=29.1sec). She was anticoagulated with enoxaparin and made satisfactory recovery. This case demonstrates ruptured AAA is prothrombotic and that DVT can develop despite coagulopathy. It is recommended that screening for thrombosis should be done and anticoagulation be initiated early.

Introduction

Ruptured Abdominal aortic aneurism is associated with high mortality not only from hemorrhage but also from ischemia induced organ failure. Coagulopathy is prevalent further worsening hemorrhage. However, it is unusual to find thrombosis concurrently with coagulopathy. Here a case of bilateral DVT in the leg veins is described in association with prolonged routine coagulation tests and thrombocytopenia.

Case Description

A 76-year-old lady was being treated for peptic ulcer disease at a primary care facility. She later presented at tertiary hospital with a few days history of worsening abdominal pain. At admission she had tender abdominal distension. Her other past medical history was unremarkable. An abdominal ultrasound scan revealed ruptured aortic aneurism. Initial laboratory investigations included a hemoglobin concentration of 9.4 g dL⁻¹, platelet count of 13 x 10⁹ L⁻¹, creatinine concentration of 103 micromoles L⁻¹; and a random blood sugar of 11.6 millimoles L⁻¹ (Table 1). Her condition deteriorated necessitating endotracheal intubation, mechanical ventilation and inotropic support. In addition, tranexamic acid infusion was started while awaiting emergency open surgical repair of ruptured aortic aneurysm.

General anesthesia was induced with ketamine and atracurium; and was maintained on isoflurane with stable hemodynamics. Following laparotomy, three liters of hemoperitoneum was evacuated arising from ruptured infra-renal aortic aneurysm together with a tear in the inferior vena cava.

Hemodynamically, the patient required infusion of noradrenaline and dopamine despite transfusion with 4 units packed cells, 6 units of platelets, 6 units of fresh frozen plasma, and two liters of normal saline. Heparin was given just before application and release of aortic cross clamp. Mannitol was administered after clamp release for renal protection.

The duration of surgery was four hours with an aortic cross clamp of 120 minutes. At the end of surgery, the intestines were noted to be edematous and therefore abdomen was partially closed with 'Bogota Bag'. The patient was taken to ICU for continued ventilation and inotropic support. Empiric antibiotic treatment with clindamycin was started and mechanical thromboprophylaxis was by TED stockings. The abdomen was closed two days later after intestinal edema had settled.

Her stay in ICU was complicated by acute renal injury requiring renal replacement therapy; pleural effusion managed by chest tube; and difficulty initiating enteral feeding. She had coagulopathy right from 1st post-operative day that persisted despite infusions of several units of fresh plasma and platelets (Table 1). She was started on prophylactic enoxaparin on 4th post-operative day; and extubated successfully on the 6th postoperative day.

On the 9th post-operative day, Doppler ultrasound scan showed bilateral deep venous thrombosis in the calf veins after which enoxaparin was increased to 40mg twice daily and adjusted to 60mg twice daily after two days. Subsequently, she developed features of fluid overload: pulmonary edema, pleural effusion and pitting edema. Pulmonary embolism was ruled out after a CT pulmonary angiography. She was treated with furosemide and bronchodilators and discharged home on Dabigatran 40 mg twice daily in stable condition after 33 days of hospital stay.

Day (from admission)		0(admission) 00:23 Hrs	O(admission, repeat) 11.55 Hrs	1	2	3	4	5	6	7	8	9	14	18
Hb g/dL		9.6	10.5	11.7	12	12.3	10.8	10.3	11.1			ĺ	12.8	12.7
Platelets		133	122	79	51	76	58	44	64	93	132		306	276
Prothrombin time (sec)	Test	14.6	14.4	17.7	20.5	16.4	15.1	16.0	15.2	16.1		19.4		
	Control	13.4	13.4	13.4	13.4	13.4	13.4	13.4	13.4	13.4		13.4		
	Activity (%)	91	93	75	65	81	88	83	88	83		69		
INR		1.098	1.082	1.354	1.59	1.246	1.139	1.213	1.147	1.222		1.536		
Fibrinogen (mg/dL)		413	395	469	827	781	617	457	384	289				
APTT (sec)	Test	27.7	26.7	34.8	30.8	28.7	29.8	30.6	27.5	26.8				
	Control	29.1	27.1	29.1	29.1	29.1	29.1	29.1	29.1	29.1				
Thrombin time (sec)														
	Test	15.7	18.1	21	16.4	20.9								
	Control	15.9	15.9	15.9	15.9	15.9								

Discussion

This case is presented to illustrate three main medical conundrums-frequently missed AAA diagnosis at primary settings where most patients present; associated thromboembolic complications that often prolong hospital stay for those who survive surgery; and emphasis that DVT may develop despite coagulopathy in vascular disease and surgery. Unlike many cases of AAA who rupture and die before diagnosis, she was lucky that she landed in a tertiary hospital with vascular surgery services where prompt diagnosis of AAA was made and surgical intervention thereafter.

The reported mortality of ruptured AAA varies even in best centers and regions at between 33-80%, but may be higher if rupture occurs in the community before arriving at the hospital¹. The wide variability could be due to referral networks, comorbidities such as hypertension, diabetes mellitus, smoking and loss of physiological reserve due to old age. Known complications associated with AAA before or after surgical repair depend on the size and location of the lesion². These are due to interruptions of blood supply by the aneurism, vascular clamping during surgery, and thromboembolism associated with stasis below occlusive clamps. It is usual practice to administer heparin anticoagulation just before application of cross clamps, and mannitol and sodium bicarbonate just before release for free radical scavenging effects as well as to correct metabolic acidosis respectively. However, thromboembolic complications still occur.

Empiric pre-operative administration of Tranexamic acid in this patient may be justified by consideration to decrease operative blood loss. This is supported by protocols based on results of CRASH-2 trial which established its beneficial role in trauma if given within three hours of injury ³, though it is still controversial in non-trauma cases ⁴. The objection against blanket administration of tranexamic acid revolves around the concept of 'fibrinolytic shut down' ⁵ and therefore most authorities now advocate for judicious use unless supported by laboratory evidence ⁶. When faced with clinical hemorrhage it is usually difficult to distinguish fibrinolysis from other causes of coagulopathy. Clinical signs of fibrinolysis are often non-specific and include generalized ooze especially from IV access sites, mucosa or urinary tract which were not evident in this patient. Laboratory confirmation of fibrinolysis is not always available, difficult to perform and time consuming. The laboratory tests in evaluation of fibrinolysis includes thromboelastography, assays of t-PA, PAI-1, D-dimers, eugloblobulin and fibrin plate lysis test. A caution should be sounded in administration of anti-fibrinolytics, that although useful in preventing blood loss, fibrinolysis is usually transient, and followed by fibrinolytic shutdown that may be associated with thrombus formation and increases mortality ⁵.

This patient had laboratory evidence of coagulopathy but without associated bleeding complications. Similar changes were observed by others in abdominal aortic aneurism cases ^{2 7 8}. It is not unusual to find patients with elevated PT or APTT with clinical evidence of DVT/PE²⁹. The prolonged coagulation test results could be explained on the basis of release of natural anticoagulants such as antithrombin III, thrombomodulin and syndecan in the so called 'endotheliopahty' triggered by hypotension, high circulating catecholamines augmented by iatrogenic vasoppressors ¹⁰. By blocking coagulation tests, a state called hypocoagulability. In this case, despite laboratory evidence of hypocoagulability, there was no increased haemorrhage, and instead paradoxically, the patient developed clinical thrombosis.

In an analysis of coagulopathy in aortic aneurism, Franson ¹¹ found that although coagulopathy was found in 30 out of 55 patients, of the 12 deaths that occurred, only 2 were due to haemorrhage and 10 from thrombosis despite coagulopathy. Blood from ruptured aortic aneurism is prothrombotic, as evidenced by presence of enhanced thrombin generation, suppressed fibrinolysis ¹², elevated vWF ¹³ and nuclear extracellular traps formation (NETs) ¹⁴. The mechanism of thrombosis in such patients is due to elevated plasma hyperactive vWF multimers owing to low ADAMPTS 13 and hyperactive platelets. In deed prolonged PT and APTT in the background of low platelets ⁷, high vWF:Ag and vWF:Rco ⁸ ¹⁵ have been reported. These illustrates the fact that in as much as the routine tests may be prolonged, the patients are still procoagulant through vWF axis. Therefore, deranged routine tests are not protective against thrombosis. The procoagulant changes always correlate with release of aortic cross clamp suggesting influence of ischaemia-reperfusion effects on coagulation reactions ¹⁶.

The platelet count changes in this patient are in keeping with other cases of aortic aneurisms reported in the literature ¹⁷. In the reported case series, the extent of fall in Platelets counts were inversely associated with duration of cross-clamping, but not to duration of surgery, pre-procedure platelet counts, heparin or protamine administration, contour or size of aneurism, operative blood loss or transfusion requirements. The phenomenon of thrombocytopenia coexisting with hypercoagulability in relation to aortic cross-clamping has been explained by hypothesis of ischemia-reperfusion whereby inflammatory cytokines as well as reactive oxygen species lead to platelet activation ¹⁸. In the process, the released neutrophil proteases mediate platelet apoptotic hyperactivation and endothelial injury releasing procoagulant vWF as well as anticoagulants thrombomodulin Brudzyn ¹⁹.

It is not clear at what point this patient developed DVT, which presents a limitation to this case description. It may probably have developed before presentation but remained asymptomatic only to be found during routine imaging. DVT can develop preoperatively²⁰, intraoperatively or post operatively² ²¹, or PE in unruptured AAA ²². Nonetheless, the risk factors for DVT were also present in this patient-transfusion of multiple blood products- FFP, platelets and packed red blood cells. Stored red blood cells are thrombogenic that increases with duration of blood storage owing to molecular changes such as microparticles, decryption of Tissue factor and externalization of phosphatidyle serine (PS).

In the largest case series of thromboembolism in AAA reported so far, Davenport ²³ and Lee ²⁴ found that the 30 day VTE rate was 1.1% of which 30% were post discharge, and the peak period was 6-7th day post-surgery. The identified risk factors were duration of surgery lasting>4 hours, amount of blood products administered>5 units, age and co-morbidities. The risk of VTE was increased if the aneurism was ruptured and open laparotomy repair done.

Although Routine tests such as PTI, APTT and Thrombin time were used for coagulation status in this patient, they suffer from a number of limitations. They are not predictive, neither specific nor sensitive for diagnosis of either thrombosis or haemorrhage. They are not predictive of bleeding prior to surgical procedures or interventions. Nevertheless, they may have a role in determining need for factor replacement in cases of massive haemorrhage ²⁵. One needs to understand the context in which the PT or APTT were designed-they were tested in liver disease or neonates to detect single factor deficiencies in haemophilia, not to predict bleeding risk or thrombosis²⁶. They do not evaluate the contribution of cellular

elements such as platelets, RBC or leukocytes which are now known to participate in haemostasis. Furthermore, they are not sensitive to contribution of vWF and endothelium in haemostasis.

In conclusion, given the context in which thrombosis develops in ruptured AAA in this case and from the literature, early initiation of thromboprophilaxies should not be withheld despite prolonged PT or APTT. The role of Mechanical thromboprophylaxis remains undetermined to prevent lower limb DVT in this clinical case. It is recommended that further prospective research be conducted in our local setting to define the coagulopathies that occur in association with aortic aneurism, and analyse the composition of thrombus to elucidate therapeutic targets.

- Adam, J. D. Coagulation , fibrinolysis and endothelial cell activation in abdominal aortic aneurysm repair. (University of Edinburgh(United Kingdom), 2003).
- Adam, D. J., Ludlam, C. A., Vaughan, C. & Bradbury, A. W. Coagulation and fibrinolysis in patients undergoing operation for ruptured and nonruptured infrarenal abdominal aortic aneurysms. J vasc surg 30, 641–650 (1999).
- 3. CRASH-2 Collaborators et al. Effects of tranexamic acid on death , vascular occlusive events , and blood transfusion in trauma patients with significant haemorrhage (CRASH-2): a randomised ,. Lancet 376, 23–32 (2010).
- 4. Binz, S. et al. CRASH-2 Study of Tranexamic Acid to Treat Bleeding in Trauma Patients : A Controversy Fueled by Science and Social Media. J. Blood Transfus. 2015, (2015).
- Moore, E. E. et al. Postinjury fibrinolysis shutdown : Rationale for selective tranexamic acid. J Trauma Acute Care Surg 78, S65-S9 (2015).
- Johnston, L. R., Rodriguez, C. J., Elster, E. A. & Bradley, M. J. Evaluation of Military Use of Tranexamic Acid and Associated Thromboembolic Events. JAMASurg 153, 169–175 (2018).
- Adam, D. J., Haggart, P. C., Ludlam, C. A. & Bradbury, A. W. Coagulopathy and Hyperfibrinolysis in Ruptured Abdominal Aortic Aneurysm Repair. Ann. Vasc. Surg. 18, 572–577 (2004).
- Campbell, V., Marriott, K., Stanbridge, R. & Shlebak, A. Successful Aortic Aneurysm Repair in a Woman with Severe von Willebrand (Type 3) Disease. Case Rep. Hematol. 2015, (2015).
- 9. Spencer, A., Pearce, M. I. & Ames, P. R. J. Sequential thrombosis and bleeding in a woman with a prolonged activated partial thromboplastin time. Thromb. J. 9, 2-5 (2011).
- Ostrowski, S. R. et al. Sympathoadrenal activation and endotheliopathy are drivers of Hypocoagulability and Hypofibrinilysis in Trauma: A Prospective observational study of 404 severely injured patients. J Trauma Acute Care Surg 82, 293–301 (2017).
- Fransson, M., Rydningen, H. & Henriksson, A. E. Early Coagulopathy in Patients With Ruptured Abdominal Aortic Aneurysm. Clin. Appl. Thromb. 18, 96–99 (2012).
- Adam, D. J., Haggart, P. C., Ludlam, C. A. & Bradbury, A. W. Hemostatic markers before operation in patients with acutely symptomatic nonruptured and ruptured infrarenal abdominal aortic aneurysm. J. Vasc. Surg. 35, 661-665 (2002).

- Skagius, E., Siegbahn, A., Bergqvist, D. & Henriksson, A. Activated Coagulation in Patients with Shock due to Ruptured Abdominal Aortic Aneurysm. Eur. J. Vasc. Endovasc. Surg. 35, 37-40 (2008).
- Meher, A. K. et al. Novel Role of IL (Interleukin)-1 in Neutrophil Extracellular Trap Formation and Abdominal Aortic Aneurysms. Arterioscler. Thromb. Vasc. Biol. 38, 843–853 (2018).
- Kokot, M. et al. Endothelium injury and inflammatory state during abdominal aortic aneurysm surgery : scrutinizing the very early and minute injurious effects using endothelial markers – a pilot study. Arch Med Sci 9, 479–486 (2013).
- Holmberg, A., Siegbahn, A., Westman, B. & Bergqvist, D. Ischaemia and Reperfusion During Open Abdominal Aortic Aneurysm Surgery Induce Extensive Thrombin Generation and Activity. Eur J Vasc Endovasc Surg 18, 11-16 (1999).
- Bradbury, A.; Adam, D.; Garrioch, M.; Brittendon, J.; Ruckley, C. V. Changes in Platelet Count, Coagulation and Fibrinogen Associated with Elective Repair of Asymptomatic Abdominal Aortic Aneurysm and Aortic Reconstruction for Occlusive Disease. Eur. J. Vasc. Endovasc. Surg. 13, 375–380 (1997).
- Norwood, M. G. A., Bown, M. J. & Sayers, R. D. Ischaemia-Reperfusion Injury and Regional Inflammatory Responses in Abdominal Aortic Aneurysm Repair. Biomed Res. Int. 245, 234–245 (2004).
- Budzyn, M.N.; Gryszczy, N.B.;Majewsky, W.; Krasinki, N. Z. M. P., Formanowicz, D., Wojciech, K. & Iskra, M. The Association of Serum Thrombomodulin with Endothelial Injuring Factors in Abdominal Aortic Aneurysm. Biomed Res. Int. 2017, (2017).
- Otsui, K., Yamamoto, M. & Aoki, H. A super-elderly case of abdominal aortic aneurysm associated with chronic disseminated intravascular coagulation. J. Cardiol. Cases 11, 48–51 (2015).
- 21. Eadara, S. et al. The Rate of Deep Venous Thrombosis in Patients Undergoing Endovascular Aneurysm Repair or Open. Clin. Surg. 4, 1-4 (2019).
- 22. Sajjad, J., Ahmed, A., Coveney, A. & Fulton, G. A large unruptured abdominal aortic aneurysm causing pulmonary embolism. J. Surg. Case Reports 7, 1-4 (2015).
- 23. Davenport, D. L. & Xenos, E. S. Deep venous thrombosis after repair of nonruptured abdominal aneurysm. J. Vasc. Surg. 57, 678-683.e1 (2009).
- 24. Lee, F. et al. Increased risk of deep vein thrombosis and pulmonary thromboembolism in patients with aortic aneurysms : A nationwide cohort study. PLoS One 12, 1-14 (2017).
- 25. Watson, H. G.; Greaves, M. Can We Predict Bleeding ? Semin. Thromb. Hemost. 34, 97-103 (2008).
- 26. Thachil, J. Is coagulopathy a contraindication for thromboprophylaxis ? Q J Med 106, 1155–1156 (2013).

Case Presentation

Anaesthetic Management of a Patient with Neurofibromatosis

Anne-Marie N. Githaiga, Wangari Waweru-Siika, Devi Mong'are

Abstract

The neurofibromatoses are a group of autosomal dominant hereditary diseases characterized by the formation of tumors of ectodermal and mesodermal tissue.³ This case report highlights salient points in the management of the patient with neurofibromatosis: the importance of a thorough history and physical examination, relevant investigations, the active search for associated complications or disease associations, and the integration of all this for the formulation and delivery of a safe anesthetic plan.

Introduction

Although eight subtypes of neurofibromatosis (NF) have been proposed to date, only two distinct types have been defined: a peripheral and central form.³ They differ by the predominant localization of the tumors in relation to the peripheral and central nervous systems. Type 1 von Recklinghausen disease (affects about 85% of patients) and Type 2 (affects about 10% of patients). ³ The incidence of NF1 is 1 in 2,500-3,300 and that of NF 2 1 in 33,000-40,000. ⁵ NF1 is characterized by diffuse tumors in dermal and subcutaneous tissue and is composed of Schwann cells, fibroblasts and perineural cells.¹ Symptoms vary in severity, but may affect all organ systems, with neurofibromas not only in the neuroaxis but in the oropharynx and larynx as well, leading to difficulty in laryngoscopy and tracheal intubation.¹ Pulmonary pathology includes pulmonary fibrosis and cystic lung disease. ⁴ The cardiovascular manifestation of NFI include hypertension which may be associated with pheochromocytoma or renal artery stenosis. ² Neurofibromas may also affect the gastrointestinal tract and carcinoid tumors may be found in the duodenum.²

Case Presentation

A 22-year-old female presented to our institution with a 20year history of multiple, progressively enlarging masses on her trunk, lower back and left arm. The growth on the left arm was of concern as it was heavy and caused her imbalance, pain and social ridicule and she was scheduled for left arm debulking surgery. She had no known co-morbidities.

There was no history of difficulty in breathing, hoarseness of voice or stridor and she denied any history of chest pain, orthopnea, paroxysmal nocturnal dyspnea or lower limb swelling. Her exercise tolerance was reasonable as she could walk for long distances without breathlessness but was limited by the weight of her left arm.

She had a history of global, dull headaches on and off associated with blurred vision, but did not have seizures, tremors, syncopal episodes or focal neurological deficits. There was no yellowness of eyes or skin, nausea, or vomiting, and she reported normal bowel movements and urinary function.

Surgical debridement of her left arm had been performed three years prior under general anesthesia with no complications.

She was an only child and there was no positive family history of a similar condition.





On examination we found a young lady who appeared depressed. Her entire left arm was grossly enlarged and deformed (figure 1 and 2) and she had areas of hyperpigmentation and nodules throughout her body (Figure 3). Her blood pressure was mildly elevated at 145/100 mmHg. The rest of her vital signs were normal and systematic examination was unremarkable. She weighed 50 kg and was 122 cm tall.

Airway examination revealed limited mouth opening with an inter-incisor gap of 2 cm and a high arched palate. She had a Wilson's score of 3 and she was classified as a Mallampati grade IV. Her neck was short with a thyromental distance of 4 cm but with good range of movement.

Her preoperative hemogram was normal with a baseline hemoglobin of 11g/dl. Her urea, creatinine and extended electrolytes were also within normal range.

Our anesthetic plan included endotracheal intubation of an anticipated difficult airway, hypotensive anaesthesia using Total Intravenous Anesthesia (TIVA), invasive blood pressure monitoring in addition to standard ASA monitoring, thromboprophylaxis using graduated compression stockings intraoperatively, and recovery in the general surgical ward post-operatively. The blood bank was alerted to the possibility of massive hemorrhage due to the size of the arm and the extent of the planned surgery. Three units of packed red cells and three units of fresh frozen plasma (FFPs) were ordered for intraoperative use.

A peripheral nerve block was considered for post-op pain relief. However, a pre-operative MRI of the affected arm showed that the brachial plexus on the left was encased by the neurofibroma. A nerve block was therefore considered a relative contraindication due to the likelihood of injury during surgical dissection that would potentially then be attributed to the regional anesthetic technique. A pain specialist was invited to guide post-op pain management, and a postoperative consultation with the hospital psychologist organized for management of a major depressive disorder.

The calculated allowable blood loss was 1,200 ml, based on a maximum acceptable drop in hemoglobin to 8 g/dl. Two 18-gauge cannulas were inserted on the right hand and anesthesia induced using midazolam 2 mg, remifentanil 50 mcg/min for 10 minutes and propofol 150 mg. Cisatracurium 10 mg was administered after adequate mask ventilation was confirmed and a size 7.0 endotracheal tube inserted under direct laryngoscopy. A Cormack-Lehane Grade II was recorded. Difficult airway adjuncts such as a stylet, a bougie and a laryngeal mask airway were available in the event of a difficult intubation but were not required. Ventilation was performed to normocapnia and oxygen saturations maintained above 94%.

A 20-gauge right radial arterial cannula, a urethral catheter and a temperature probe in the right nostril were inserted.

TIVA with remifentanil at 12.5-25 mcg/minute and propofol at 2.5 mg- 5mg/minute were used for maintenance and 10mg morphine given. Tranexamic acid 2g was administered before surgical incision and a tourniquet at 200mmHg placed on the left upper limb. Cefuroxime 1.5g was given at induction and repeated four hours later intraoperatively. Active warming was provided using forced air warming via a Bair hugger®.

During surgery, there was profound blood loss of approximately 2 liters with brief episodes of hypotension that responded to 50-100mcg boluses of phenylephrine, 500ml of Voluven® and commencement of blood transfusion. A sample from the arterial line revealed a hemoglobin of 5.6 g/dl with a lactate of 2.2mmol/l. (Figure 4). The patient was transfused three units of packed red cells, three FFPs and a unit of platelets, and a repeat dose of 2g of tranexamic acid administered. Total fluid input was 5 liters of Hartmann's solution and 500 ml of Voluven®. Urinary output was measured hourly and exceeded 50 ml/hour with a total collection of 1,100ml. Total anesthesia time was 8 hours.

Tourniquet time was six hours in total, with intermittent breaks of 30 minutes every two hours. Upon release of the tourniquet at the end of the surgery, 500ml of fresh blood was noted to enter the drain that had been left in the left arm. This however ceased following the application of a pressure dressing. The patient was successfully extubated following reversal of neuromuscular blockade. An additional unit of packed red blood cells was given in the post-anesthesia care unit (PACU) in view of the previously active drain.

Arterial Blood Gases

	12:26pm (4 hrs. intra-op)	2:45pm (6.5 hrs. intra-op)
рН	7.42	7.41
PCO2 (mmHg)	35	35
PO2 (mmHg)	178	90
Glucose (mmol/l)	4.6	4.4
Lactate (mmol/l)	2.2	1.5
Hematocrit (%)	18	29
HCO3 (mmol/l)	20.9	22
Base Excess (mmol/l)	-5.2	-4.9
SO2 (%)	100	100
Hemoglobin (g/dl)	5.6	9.4



Her vital signs were stable post-operatively and a check hemoglobin revealed a hemoglobin of 12g/dl. Her other blood works were within normal limits. Postoperative pain was managed with morphine 6 mg subcutaneously 6-hourly, paracetamol 1g 6-hourly and lornoxicam 8 mg 12-hourly. Rescue pethidine was prescribed for breakthrough pain.

Discussion

The Neurofibromatoses are a group of conditions that vary in severity, and which have fundamental implications for anesthesiologists, physicians and surgeons. NF1 is one of the most common genetically transmitted diseases with an incidence of 1 in 2,500-3,300. ⁵ Anaesthetists are therefore likely to encounter these patients, and an appreciation of the implications of this condition on the planned anaesthetic is vital. ³ Although the manifestations of NF1 are often mild, there may be associated pathology of direct relevance and importance to the anesthetic management of patients with the disease. It is therefore important to have a working knowledge of the clinical manifestations of the disease so that a systematic approach to the preoperative assessment of these patients can result in sound perioperative management. ^{1,3}

The detection of complications associated with NF calls for thorough history and physical examination, followed by targeted tests and imaging as guided by the clinical exam. ¹⁴ These include cranial computerized tomography (CT) or magnetic resonance imaging (MRI) to assess for neurofibromas affecting central or peripheral nerves that would affect regional anesthetic or the risk of herniation with intracranial masses , pulmonary function tests due to the possibility of airway or lung neurofibromas or lung fibrosis, echocardiogram due to the possibility of aortic aneurysms and hypertrophy complicated by outflow obstruction, blood urea nitrogen/ creatinine measurement, detection of abnormal electrolytes . ¹⁴ These investigations are guided by symptoms and physical examination.^{2,3}

Comments

Hirsch et al.

System

Table 3 Anaesthetic considerations of NF1

Pharmacology

There is controversy surrounding sensitivity to neuromuscular blocking agents and both increased and decreased sensitivity has been reported. The recommendation is to always use a peripheral nerve stimulator to monitor neuromuscular activity in patients with NF intraoperatively and at reversal. 3

Pregnancy

Pregnancy is associated with an increase in the number and size of neurofibromas. There is also potential for rapid increase in size of CNS tumors. Which should be considered in neuraxial anesthetic techniques for these patients.³

Conclusion

It is important to be aware of the clinical features of Neurofibromatosis and the likelihood of systemic involvement. Thorough preoperative evaluation and a multidisciplinary approach to care is essential in the care of these patients. Major blood loss is of particular concern in debulking surgeries associated with neurofibromatosis due to the mass of tissue excised and vessels involved. Massive Transfusion should be anticipated, and anesthetic plan should minimize blood loss.

- Anaesthetic Considerations in a Patient with Von Recklinghausen Neurofibromatosis Kishan Rao Bagam, Vijaya Durga S, Mohan K, Swapna T, Maneendra S, Murthy SGK. J Anaesth Clin Pharmacol 2010; 26(4); 553-554
- Neurofibromatosis: clinical presentations and anaesthetic implications. N.P. Hirsch, A. Murphy and J.J Radcliffe. Br J Anaesth 2001; 86:555-64
- 3. Neurofibromatosis: S. NAIDU University of Kwazulu-Natal
- Inan N, et al. The anaesthetic approach in a patient with type 1 neurofibromatosis with multiple deformities. Turk J Med Sci 2008; 38(5): 477-80
- Perioperative Management of Neurofibromatosis Type 1; Charles J. Fox, Samir Tomajian MD, Aaron J. Kaye, Stephanie Russo, Jacqueline Volpi Abadie MD, Alan D. Kaye PhD MD

Airway	Neurofibroma of tongue, pharnyx or larynx may interfere with tracheal intubation				
	Suspicion raised by history of dysphagia, dysarthria, stridor or change of voice				
Respiratory system	Intrapulmonary neurofibroma, pulmonary fibrosis may produce cough and dyspnoea				
	Right ventricular failure may be present				
	Scoliosis/kyphosis may compromise lung function				
Cardiovascular system	Raised arterial pressure usually essential hypertension but consider phaeochromocytoma or renal artery stenosis				
	Hypertrophic cardiomyopathy may occur				
	Mediastinal tumours may result in superior vena caval obstruction				
Central nervous system	Cerebral and spinal neurofibromas common				
1104-4	Increased incidence of epilepsy and learning disorders				
	Cerebrovascular disease may co-exist				
Gastrointestinal tract	Intestinal tumours may present with pain, gastrointestinal haemorrhage or perforation				
	Carcinoid tumours occur in duodenum and may result in jaundice and carcinoid syndrome				
Genitourinary system	Neurofibromas may cause ureteric/urethral obstruction				
Musculoskeletal system	Vertebral deformities or spinal cord tumours may make spinal/extradural techniques difficult				

Case Report:

Severe ARDS following Csytogastrostomy For a Pancreatic Pseudocyst

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Abstract

While pulmonary complications are common after major pancreatic surgery e.g. pancreatoduodenectomy, severe acute respiratory distress syndrome after cystogastrotomy is a rare occurrence. Acute pancreatitis is strongly associated with Acute respiratory distress syndrome. We report a case of a twenty-four (24) year old female who presented with alcohol related chronic pancreatic pseudocyst who underwent cystogastrostomy with consequent fatal acute respiratory distress syndrome.

Case Presentation

Acute respiratory distress syndrome (ARDS) is a diffuse, sudden-onset inflammatory process involving both lungs that results in increased lung pulmonary vascular permeability, decreased lung compliance, and alveolar edema and damage(1).

Postoperative pulmonary complications such as pneumonia and ARDS substantially increase the risk of morbidity and mortality, length of hospitalization and related financial burden (2). Acute respiratory distress syndrome in particular is often associated with multiple organ dysfunction and carries a high risk of mortality and financial cost (3).

Acute respiratory distress syndrome is characterized by the development of acute dyspnea and hypoxemia within hours to days of an inciting event, such as trauma, pneumonia, sepsis, drug overdose, massive transfusion, acute pancreatitis, or aspiration (4). Approximately one third of severe acute pancreatitis patients develop acute respiratory distress syndrome (ARDS) that account for 60% of all deaths within the first week(5).

Here we describe the clinical presentation, surgical and critical care management of a patient who presented with alcohol related chronic pancreatitis pseudocyst with gastric outlet obstruction.

Case Description

A twenty-four-year old female patient presented with twomonth history of progressive abdominal distension with epigastric pain radiating to the back. This was associated with early satiety, post prandial vomiting and weight loss. She consumed more than twelve units per week of alcohol (several bottles of whisky) on a regular basis for the past four years and smoked tobacco (two pack years). There was no associated yellowness of eyes, itchiness, dark urine nor clay colored stool. She was allergic to sulphur containing drugs. Physical examination revealed a moderately wasted patient with a blood pressure of 112/68 mmHg, a pulse rate of 92 bpm and an oxygen saturation of 98% on room air. Abdominal examination revealed globally distended abdomen that was mildly tender but with no guarding or rebound tenderness. It was dull to percussion with no shifting dullness and reduced bowel sounds. The rest of systemic examination findings were normal.

Blood tests showed elevated plasma amylase level of 1917 IU (normal \leq 220 IU) and lipase level of 174.9 U/I (normal \leq 60 U/I) with normal levels of aspartate transaminase, alanine transaminase, gamma glutamyl transferase, total protein and albumin. Elevated direct bilirubin (4.56 mmol/I, normal \leq 4.3), C-reactive protein (17.4, normal 0-8, and CA 125 (123.7, normal 0-35) levels were also noted. VDRL and serology for HIV and hepatitis B were negative. Her random blood sugar was 6.0 mmol/I. Total blood count was normal with mild granulocytosis (87.1%, normal 50-75%) and thrombocytosis (477 x 10⁹ cells/L, normal 150-450 x 10⁹ cells/L. Her renal function tests and electrolytes were normal except for a mild hypernatremia.

Computerized tomography (CT) scan of the abdomen showed a large peritoneal cystic lesion around the stomach, spleen and Morrison's pouch with minimal mesenteric inflammation. The rest of the abdominal organs appeared normal.

Patient was subsequently prepared for abdominal surgery under general anesthesia.

Anesthesia Induction

General anesthesia was induced with propofol and maintained with atracurium and halothane with F_1O_2 0.5 of oxygen/nitrous oxide mixture. Ventilation was on volume-controlled mode with a tidal volume of 400ml (8 ml/kg), a rate of 14 bpm and a positive end-expiratory pressure (PEEP) of 5 cmH₂O.

Surgical Procedure

Laparotomy via a left subcostal incision revealed a large cystic retrogastric mass. The mass was accessed via the stomach and about 3600 ml of dark colored cystic fluid drained, irrigated with warm saline and subsequently fashioned to the posterior gastric wall. The anterior gastric wall was repaired and abdomen closed in layers. Procedure was completed uneventfully.

Anesthesia Maintenance

Anesthesia was maintained with halothane/nitrous oxide and oxygen mixture. Analgesia management consisted of intravenous tramadol, diclofenac and paracetamol. The total fluid given during the 2-hour surgery was 2.5 liters of crystalloids (normal saline and ringer's lactate). Vital signs and oxygenation remained normal over the time span of surgery. Blood loss was approximately 150 ml and transfusion was not warranted. After reversal of neuromuscular blockade, the patient was extubated successfully. She was conscious, with normal respiratory and hemodynamic parameters at the time of transfer to the postoperative room.

Postoperative Care and ICU Management

Her first hour in the postoperative room was uneventful. She was fully awake with oxygen saturations above 95% on room air. In the second hour she developed tachypnea, labored breathing. Her oxygen saturations dropped to 54%. Initial attempts to stabilize her condition including administration of intravenous furosemide 60 mg and high flow oxygen (15litres/min via a non-rebreather mask) were not successful. She was subsequently re-intubated and transferred to the intensive care unit.

The diagnostic work-up in the course of her intensive care management included Chest CT Scan, CT-Pulmonary Angiogram, and Echocardiogram. Chest imaging showed a marginally enlarged pulmonary trunk (29mm), but no features of acute pulmonary thromboembolism or right heart strain, dense peri-hilar consolidations with background ground glass opacification suggestive of ARDS. Echocardiogram showed a normal systolic function ejection fraction of 53%, moderate pulmonary hypertension with a functional tricuspid regurgitation.

Arterial blood gas analysis after fifteen minutes of volume controlled mechanical ventilation (tidal volume 400 i.e. 8mls/kg), respiratory rate of 28 breaths per minute, a Positive End Expiratory Pressure of 8 and a fraction of inspired oxygen of 0.95 revealed a mixed metabolic acidosis and respiratory alkalosis with significant hypoxia (PH 7.32 PCO₂ 24mmHg PO₂ 49mmHg BE -6mmol/L HCO₃ 17mmol/L). The partial pressure of arterial oxygen: F_iO_2 (P/F) ratio was 51 qualifying a diagnosis of severe acute respiratory distress syndrome.

The patient was noted to have a hypokalemia of 2.64mmol/l (range 3.5-5.5), a gamma-glutamyl transpeptidase of

450U/l(range 9-48) and a low albumin level of 26g/dl(range 35-50). The rest of her Renal Function Tests, serum electrolytes and liver function tests were normal.

Cultures of tracheal tube, urine and blood specimens were negative. The patient was ventilated with a standardized ARDSNet protocol that focused on low tidal volume (6 ml/kg of ideal body weight and a target pH > 7.25), with a positive end-expiratory pressure (PEEP) in creased to 18 cm H₂O. Deep sedation and muscle paralysis were required to achieve target respiratory parameters for the first 6 days of her ICU stay. In the subsequent 5 to 6 days F_iO_2 was gradually reduced to 0.6 and positive end expiratory pressure reduced to 8 cmH_2O . Major (renal, liver, heart) organ functions were preserved through this period.

From the 13th day in ICU, the patient was noted to have purulent tracheal secretions, new lung infiltrates (on chest X-ray), fever (up to 39° C) and increased oxygen requirements (F_iO_2 increased to 0.9). Inflammatory markers were also on the rise with a white cell count of 24x10°/L and a procalcitonin level of 14. Based on a clinical pulmonary infection score of 7, an impression of ventilator associated pneumonia and samples for tracheal aspirate, urine and blood cultures collected. Despite initiation of appropriate antibiotics the patient's condition progressed to septic shock necessitating inotropic support. She succumbed 2 days later.

Discussion

Acute pancreatitis is defined as an inflammatory process of the pancreas with variable involvement of regional tissues or remote organs. According to its severity, acute pancreatitis is designated as mild or severe (6).

Chronic pancreatitis on the other hand is a syndrome characterized by chronic progressive pancreatic inflammation, fibrosis, and scarring, resulting in damage to and loss of exocrine (acinar), endocrine (islet cells), and ductal cells (5). Acute pancreatitis, recurrent acute pancreatitis, and chronic pancreatitis represent a disease continuum.

Pancreatic pseudocysts are organized accumulations that are rich in pancreatic enzymes, which arise as a consequence of and remain after an episode of acute pancreatitis or after exacerbation of chronic pancreatitis (7).

For acute fluid accumulations, no intervention is necessary, since most of these tend to resolve spontaneously. If pseudocysts develop, depending on their characteristics and location, they can be treated expectantly unless they present related symptoms or complications: abdominal pain, early satiety, weight loss and persistent fever.

The classical surgical options are cystogastrostomy, cystoduodenostomy and Roux-en-Y cystojejunostomy, depending on the location of the cyst.

Either open or laparascopic surgical approaches can be used. Other surgical options include endoscopic, percutaneous and ultrasound-guided drainage (7). In this case cystogastrotomy was preferred by the surgeon as the choice of surgical approach.

The patient described in this case had a history of heavy alcohol use over several years which may have been the key risk factor for chronic pancreatitis. Pancreatitis-associated ARDS has been reported to be related to the effects of pancreatic enzymes. Phospholipase A2 is particularly thought to play a role in ARDS by damaging pulmonary surfactant, which is a substrate for phospholipase A2 (8). In our opinion, ARDS in this patient may have been triggered by spillage of the contents of the pseudocyst into the peritoneal cavity.

Concurrent smoking is an independent risk factor (more than 2-fold) for development of postoperative pulmonary complications even in the absence of chronic obstructive pulmonary disease. This risk remains elevated up to 1 year after smoking cessation (2).

The distance of surgical incision site from the diaphragm is inversely related to the postoperative pulmonary complications. Incidences of pulmonary complication are highest in aortic aneurysm repair, followed by cardio-thoracic and upper abdominal surgeries, whereas, the lower abdominal and peripheral surgeries were associated with low incidence of pulmonary complications (9).

Pancreatitis associated with smoking, alcoholism and upper abdominal surgery are major predictors of post-operative pulmonary complications. The ARISCAT score is a predictive tool for postoperative pulmonary complications. It stratifies patients based on age, preoperative SPO_2 , history of respiratory infection in the last one month, anemia, duration of surgery and whether or not the procedure is an emergency. Using this score, the risk of respiratory dysfunction was highly underestimated in this case (10).

Anesthesia and surgery are associated with alterations in hemodynamic, metabolic, endocrine and immunological function, which may lead to significant fluid shifts. Endothelial injury with increased capillary permeability promotes transfer of intra-vascular fluid to interstitial space. Inappropriate choice of the amount (fluid overload) and type of intravenous fluid in the perioperative period may worsen organ injury. The lungs are very susceptible to this mechanism of injury; and present in the form of non-cardiogenic edema. This compromises gas exchange, prolongs mechanical ventilation and increases the risk of pneumonia (2).

Scores with better predictive utility would trigger earlier institution of preventive measures like judicious infusion of intravenous fluids, lung protective ventilation in patients with moderate to high risk developing ARDS. This improves clinical outcomes and reduce health care resource utilization(11).

Conclusion

We encountered a patient who developed severe ARDS after cystogastrostomy who eventually succumbed despite early diagnosis and appropriate management. Acute respiratory distress syndrome is a rare but critical complication following noncomplex surgery that involves the pancreas. Young patients presenting with complicated pancreatitis particularly pseudocyst are still at a risk of developing ARDS in the absence chronic medical condition. Caution should be applied when such patients are undergoing drainage procedures with attention to prevention of respiratory complications.

Disclosures

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- ARDS Definition Task Force, Ranieri VM, Rubenfeld GD, et al. Acute respiratory distress syndrome: the Berlin definition. JAMA. 2012, 307:2526-33.
- Surjya PU, Ulka S, Sudhakar ST, Himanshu C and Piyush NM. Prevention of Postoperative Acute Lung Injury (ALI) - The Anaesthetist Role. Int J Anesthetic Anesthesiol. 2015,2:027
- Bellani G, Laffey JG, Pham T, et al. Epidemiology, patterns of care, and mortality for patients with acute respiratory distress syndrome in intensive care units in 50 countries. JAMA. 2016;315(8):788-800.
- Eloise M Harman, Acute Respiratory Distress Syndrome Clinical Presentation Medscape accessed on 3rd August 2019 available from: https://emedicine.medscape.com/article/165139clinical#b1.
- Murli M, Alok KV, Sathisha UV, Nathan LS, and Anil Mishra. Chronic Pancreatitis Associated Acute Respiratory Failure. MOJ Immunol. 2017; 5(2).
- Hasibeder, W.R. et.al. Critical care of the patient with acute pancreatitis. Anaesth Intensive Care 2009; 37: 190-206.
- Rone Antônio AA and Manlio BS (2015). Acute and Chronic Pancreatitis – Complications, Acute and Chronic Pancreatitis, Luis Rodrigo, IntechOpen, Available from: https://www. intechopen.com/books/acute-and-chronic-pancreatitis/ acute-and-chronic-pancreatitis-complications accessed on 11th December 2019.
- Vadas P. Elevated plasma phospholipase A2 levels: correlation with the hemodynamic and pulmonary changes in gramnegative septic shock. J Lab Clin Med 1984; 104: 873-881.
- Rudra A, Das S. Postoperative pulmonary complications. Indian Journal of Anesthetics. 2006;50(2):89–98. [Google Scholar].
- Tilak KM, Litake MM and Shingada KV Study of risk, incidence and mortality associated with postoperative pulmonary complications using assess respiratory risk in surgical patients in catalonia score. Int Surg J. 2019 Sep;6(9):3215-3222.
- Futier E, Constantin JM, Paugam-Burtz C, Pascal J, Eurin M. A trial of intraoperative low-tidal-volume ventilation in abdominal surgery. N Engl J Med. 2013, 369: 428-437.

Telemedicine ICU (Tele-ICU) Consultation in Kenya

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Key Message

Existing internet connectivity and widely available social media applications can support Tele-ICU rounding between intensivists Kenya and the United States. Tele-ICU rounding promotes best practice, adherence to evidence-based guidelines and improved morale in the ICU in Kenya. Utility of Tele-ICU consultation within Kenya merits additional study.

Abstract

Using a passive system of tele-ICU consultation with available wireless internet connectivity and free social media, we report on what is now the first routine regular use of tele-ICU in the region. We identify realized or potential benefits of tele-ICU such as improved morale of ICU staff who perceive benefit. Kenyan colleagues with heavy clinical obligations are supported by US consultant. This also provides access to other sub-specialists in short supply in Kenya with potential reduction in ICU transfers from other hospitals and improved pre-transfer resuscitation and stabilization.

Cost, quality and stability of current internet connections, limitations of Skype[®], time-zone differences; and legal issues of data protection and patient confidentiality are some of the factors limiting the application of tele-ICU in our set up.

Tele-ICU has a great potential in Kenya. It may be used to improve ICU consultation and triage. Further research is needed to define optimal Tele-ICU configuration for a resource limited setting; and to elucidate regulatory and confidentiality issues regarding transmission of protected health information.

Introduction

Telemedicine is defined as the "the use of electronic information and communications technologies to provide and support health care when distance separates the participants". ¹ Critical care has long been regarded as an important arena to develop telemedicine capability. Enthusiasm for telemedicine consultation and management (tele-ICU) has been fueled by the evidence that intensive care patient care outcomes are improved by 24 hour per day presence of intensivists². The growth of tele-ICU in the United States has been driven by the increasing demand for real-time critical care services, and limitations in the supply of trained intensivists.

Beginning in the late 1960's advances in computerized monitoring of critically ill patients could be extended to include remote monitoring of physiological variables.³ This process was accelerated by the successes of NASA in monitoring astronauts' physiologic variables from space.⁴ In 1980's the first reports of telemedicine consultation of the ICU were published but the first large scale study comparing outcomes of a telemedicine consultation service to historical controls was published in 2000.⁵ In what was termed "an alternate paradigm" for ICU staffing, this 16 week study of 24 hour continuous remote ICU care demonstrated a 60% reduction in severity adjusted ICU mortality. The subsequent expansion of tele-ICU medicine was undoubtedly fueled by rapid progress in the development

of comprehensive computerized medical record systems and the growing consensus that continuous intensivist coverage of critical care units was associated with improved quality of care.6 Predictably, the introduction of multiple platforms, the commercialization of tele-ICU technologies and varied approaches to tele-ICU have complicated the landscape of clinical practice and research thereby complicating the task of understanding the true impact of tele-ICU.7 Since the introduction of telemedicine in the 1990's, improvements in technology and infrastructure have greatly outdistanced proof of benefit on patient outcomes in resource rich settings. This dichotomy is no more evident than in the area of tele-ICU. The precise magnitude of the benefit of tele-ICU is not known. While systematic reviews have shown that tele-ICU is associated with lower ICU and hospital mortality⁸ authors have warned that "the optimal telemedicine configuration and dose tailored to ICU organization and case mix remains unclear". A recent consensus statement outlines the agenda for high quality research in tele-ICU to address these questions.⁹

Ideally, Kenya would be the ideal venue to develop telemedicine applications. The obvious appeal of tele-ICU in Kenya lies in the ability to provide real-time consultant presence in small ICUs that would otherwise not be able to support this level of staffing. While Sub-Saharan Africa carries 24% of the burden of the world's disease, it is served by only 3% of the world's healthworkers.¹⁰ In Kenya there are 18 physicians/100,000 population compared to 254 physicians/100,000 in the United States $^{1\!1}$ These statistics compare unfavorably to countries such as India (60/100,000 and Brazil 170/100,000) ¹⁰ In view of this critical shortage, increased telemedicine efforts for Kenya are being promoted.¹² To date, most efforts have focused on the use of Short Message Services (SMS) and other mobile applications, to promote adherence to anti-retroviral therapy and assist rural clinical officers in protocoldriven care of chronic disease. Recently, the government of Kenya announced that by the end of May 2015 at least two hospitals in each of the 47 counties should have intensive care units at a cost for equipment of Sh3.3 billion.¹³ Tele-ICU consultation between these ICUs and the two national referral hospitals could be a key component of realizing this goal while improving triage, patient care and referral.

Results and Discussion

To our knowledge, tele-ICU has not been implemented in Kenya to link ICUs either within the country or to consultants abroad. Using a passive system model of tele-ICU consultation with readily available wireless internet connectivity and free social media, we report on the use of Tele-ICU to connect Kenyan and US colleagues. Since the initial experience, the Kenyan ICU staff has conducted regular tele-ICU rounds with an intensivist colleague in the United States. We have extended tele-ICU to include transmission of radiographic studies as well as consultations from other specialties including pediatrics, endocrinology, neurology and surgical subspecialties. The rounds also provide an opportunity for the US and Kenyan staff to continue collaboration on quality improvement initiatives.

Can the use of tele-ICU improve outcomes of critically ill patients in Kenya? The answer to this questions based on review of the literature and the authors' experience, is a qualified yes.^{8,14} Globally, a recent meta-analysis of available studies showed that tele-ICU lowered ICU and hospital mortality.⁸ This systematic review included ¹¹ before and after observation studies involving over 49,000 patients and found that tele-ICU telemedicine reduced ICU mortality (RR, 0.79; 95% CI, 0.65 to 0.96; P = 0.02) and hospital mortality (RR, 0.83; 95% CI, 0.73 to 0.94; P = 0.004). However, there is uncertainty to which interventions reduce as mortality, given the heterogeneity of methodologies included under the rubric of tele-ICU. High-intensity comprehensive monitoring and management (so-called active systems) were not superior to consultation-only passive systems. It is suggested that the mortality benefit was related to better adherence to protocols of "best practice" in ventilator management, infection control and sedation practice. Quality improvement interventions focused on adoption of "best practice" have been cited in several studies as being critical factors in improved outcomes in the arena of critical care where adherence to evidence based care guidelines has been suboptimal.15 In addition, better staff education and mentoring, increased attention to patient evaluation and treatment during the first hour of critical care and enhanced involvement of hospital leadership in quality improvement efforts in support of the ICU are postulated. Despite the existing uncertainties and limitations of previous studies, these potential "mechanisms of action" of tele-ICU are directly applicable to current and future needs of critical care in Kenya.

We can identify both already realized and potential benefits of real-time telemedicine consultation in Kenya. First, staff support for the initiative has been gratifying. Nurses, medical officers and other healthcare professionals in the ICU not only perceive benefit from the Tele-ICU rounds but enjoy the distance bidirectional interactions. Second, Kenyan consultants who must balance heavy teaching and clinical obligations benefit from even this modest support from US consultants to meet the need for 24/7 availability. They may more easily obtain opinions from a variety of subspecialties which are in short supply in Kenya. The two- way education process which proceeds in the background during weekly Tele-ICU sessions is of great importance to all participants and also serves as the resource for ongoing research collaboration.

In addition to the benefits already achieved, continuing to build capacity for Tele-ICU to link our institutions includes a long term process of moving cautiously from the passive system currently in use towards a more "realtime " active system. Improved image and data sharing is a critical next step in this development but is constrained by the challenges discussed below. On a bigger stage, a Tele-ICU consultation system supporting projected ICU capacity expansion in Kenya has the potential to improve the triage, initial resuscitation and stabilization of Kenyan patients who need critical care services. These factors are key drivers of improved patient outcomes along with optimal decision making regarding the need for and timing of transfer to a referral hospital. Given the projected physician shortages in Kenya which includes medical subspecialties vital to critical care, improved availability of these consultants could be a major impetus for Tele-ICU development.¹⁶

We can identify several limitations and barriers to the dissemination of tele-ICU in Kenya. These include both technical and medical concerns. The quality and stability of current internet connections associated with significant are reliability issues. Limitations of Skype® in supporting real-time transmission of radiographic images have also hampered Tele-ICU rounds. For Tele-ICU to be successful in Kenya it would ideally be "just-in-time," consistently "on-time" and readily available. While time-zone differences are certainly an impediment to "just-in-time" consultation between Kenya and the US, we have discovered inconsistent connectivity is that a far more serious concern. Even simple audio and video conferencing remains challenging and the more ambitious task of successful routine image transfer will require substantial upgrades. Fortunately, tele-sonography applications have been developed using low band-width applications.¹⁷ Similar systems for transmission of

digital imaging to support tele- radiology consultation in low-resource settings have been described.¹⁶ These reports suggest radiographic and ultrasound image transfer within and across the borders of Kenya should be feasible although the long term costs and sustainability of this component of an "active" tele-ICU system are not well understood. Finally, legal issues regarding data protection and patient confidentiality must be addressed. While Africa has the highest rate growth of tele-medicine in the developing world, most countries in this region, like the rest of the world, lack guiding policies and proper governance to address legal issues of what has been called "cross-border consultation". ¹⁹

Based on our experience and the opportunities for growth outlined above, we advocate strongly for a robust clinical and research program to define the value of tele-ICU consultation both within and outside the borders of Kenya. Future directions for tele-ICU in Kenya should include development of financial models which address the question whether a health care system weighing often competing priorities and possessing limited resources should build and sustain tele-ICU consultation and triage within Kenya. We believe the potential to link existing and planned ICUs to consultants in national referral hospitals will turn out to be a key variable in this decision process. Cross border consultation as we have described herein can certainly be a component of this structure moving forward. Furthermore, the modeling process should be grounded in the recognition that improvements in internet infrastructure allowing real-time sharing of data and images are critical deliverables. On the research side of the equation, studies are needed to define the optimal configuration of tele-ICU and assess the cost effectiveness of tele-ICU in Kenya. Rigorous scientific evaluation of telemedicine outcomes is needed to guide these policy decisions. Finally, next steps must include a clearer understanding and hopefully consensus regarding regulatory and confidentiality issues related to transmission of protected health information within Kenya and across its borders.

In the final analysis, the same uncertainties and unanswered questions regarding tele-ICU which characterize its status in the United States and Europe will color the discussion of its proper role in Sub-Saharan Africa and other resource limited settings. While this challenge must be acknowledged, it should not deter continued development of this important technology in Kenya.

Conflicts of Interest

None identified.

Disclaimer

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- Litten M: Ueber einige vom allegmein-klinischen Standpunkt aus interessante Augenveranderungen. Berl Klin Wochnschr 1881; 18: 23-27.
- Terson A: De l'hémorrhagie dans le corps vitre au cours de l'hémorrhagie cerebrale. Clin Ophthalmol 1900; 6: 309-312.
- Nowosielska A, Czarnecki W: Zespół Tersona. Klinika Oczna 2003; 105(1-2): 79-81.
- Medele RJ, Stummer W, Mueller AJ, et al. Terson's syndrome in subarachnoid hemorrhage and severe brain injury accompanied by acutely raised intracranial pressure. J Neurosurg 1998; 88:851– 4.
- McCarron MO, Alberts MJ, McCarron P. A systemic review of Terson's syndrome: Frequency and prognosis after subarachnoid hemorrhage. J Neurol Neurosurg Psychiatry. 2004 75:491–3.
- 6. Ness T, Janknecht P, Berghorn C. Frequency of ocular hemorrhages in patients with subarachnoidal hemorrhage. Graefes Arch Clin Exp Ophthalmol 2005; 243:859-62.
- Kuhn F, Morris R, Witherspoon CD, Mester V. Terson syndrome: results of vitrectomy and the significance of vitreous hemorrhage in patients with subarachnoid hemorrhage. Ophthalmology 1998; 105:472–7.
- Pfausler B, Belcl R, Metzler R, Mohsenipour I, Schmutzhard E. Terson syndrome in spontaneous subarachnoid hemorrhage, a prospective study in 60 consecutive patients. J Neurosurg 1996; 85: 392–394.
- Fountas KN, Kapsalaki EZ, Lee GP, et al. Terson hemorrhage in individuals suffering aneurysmal subarachnoid hemorrhage: predisposing factors and prognostic significance. J Neurosurg 2008; 109:439–44.
- Riddoch G, Goulden C. On the relationship between subarachnoid and intraocular hemorrhage. Br J Ophthalmol 1925; 9:209–33.
- 11. Castren JA. Pathogenesis and treatment of Terson syndrome. Acta Ophthalmol 1963; 41:430-4.
- 12. Ballantyne AJ. The ocular manifestations of spontaneous subarachnoid hemorrhage. Br J Ophthalmol 1943; 27:383- 414.
- Ogawa T, Kitaoaka T, Dake Y, et al. Terson syndrome: a case report suggesting the mechanism of vitreous hemorrhage. Ophthalmology 2001; 108:1654–6.
- 14. Gibran S, Mirza K, Kinsella F. Unilateral vitreous haemorrhage secondary to caudal epidural injection: A variant of Terson's syndrome. Br J Ophthalmol. 2002 86:350–62.
- Nogaki H, Tamaki N, Shirakuni T, et al: [Vitreous hemorrhage after ruptured intracerebral aneurysms (Terson's syndrome).] No To Shinkei 33:223–227, 1981 (Jpn).
- 16. Oyakawa RT, Michels RG, Blase WP: Vitrectomy for nondiabetic vitreous hemorrhage. Am J Ophthalmol 96:517–525, 1983.
- Roux FX, Panthier JN, Tanghe YM, et al: Complications intraoculaires dans les hémorrhagies méningées (26 cases). Neurochirurgie 37:106-108, 1991
- Shinoda J, Iwamura M, Iwai T, et al: Intraocular hemorrhage in ruptured intracranial aneurysm. Clinical study of 172 cases and reference to Terson's syndrome. Neurol Med Chir 23: 349–354, 1983.
- M O McCarron, M J Alberts, P McCarron; A systematic review of Terson's syndrome: frequency and prognosis after subarachnoid haemorrhage: J Neurol Neurosurg Psychiatry 2004; 75:491–493.
- Bamford J, Dennis M, Sandercock P, et al. The frequency, causes and timing of death within 30 days of a first stroke: the Oxfordshire Community Stroke Project. J Neurol Neurosurg Psychiatry 1990; 53:824–9.
- 21. By David J. Browning, MD, PhD; What You Should Know About Terson's Syndrome.

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