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Editorial

The patient experience in perioperative medicine is becoming a highly desired indicator in clinical practice: this is both a subjective patient assessment of met expectations as well as the clinician's objective evaluation of the desired outcome of each intervention in a timely, safe and economical manner with return of the patient to a productive life.

Obstetric anaesthetic practice, the most common intervention in our setting, has its success defined by the wellbeing of both mother and child with timely return of both to a productive role in their domicile. In this issue, we evaluate reasons for unplanned admissions to the obstetric critical care unit at the main teaching and referral institution with an intention of early identification and interventions to minimize these as well as plan appropriately for critical care facilities and support.

Regional anaesthesia in obstetrics-as epidural for analgesia and subarachnoid for caesarean section-is the preferred mode of management for surgical intervention: these methods have been extensively

studied and are fairly safe in practice but may on occasion result in complications that would certainly impact negatively on the patient experience. We highlight the management of persistent postdural puncture headache where use of the epidural blood patch is contraindicated and routine analgesics have not been successful.

Judicious perioperative fluid management in patients is a factor that may contribute to early enhanced recovery from anaesthetic and surgical intervention: the dangers of both hypervolemia and hypovolemia related to incorrect fluid therapy have been well studied and documented. This current issue carries findings and recommendations from a prospective study on the effects of intravenous fluid regimes on intestinal function after caesarean section under subarachnoid anaesthesia.

Lastly, we highlight the inherent danger found in consumption of a readily available fruit and its extracts with successful management of subsequent toxicity: cyanide poisoning from apple seeds.

Socio-Demographic and Clinical Predictors Of Admissions Of Post-Partum Patients In The Critical Care Unit, Kenyatta National Hospital: Case-Control Study, 2017-2018

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Key words: 'Critical Care Unit', near miss

Abstract

Background

World Health Organization (WHO) describes severe maternal morbidity as near death (near miss) but survived complications which occurred during pregnancy, childbirth or within 42 days of termination of pregnancy. Pregnancy is a physiological process but can complicate in the continuum of care resulting in severe maternal morbidity or mortality which can be tragic for herself, the newborn, family and community. Immediate transfer into critical care units without delay can be lifesaving.

Objective

To determine the socio-demographic and clinical predictors leading to admissions of critically ill obstetric patients after delivery from 24 weeks gestation to 6 weeks post-partum into the CCU.

Methods

This was a prospective case-control study among obstetric patients from 24 weeks gestation to 6 weeks post-partum admitted in the Critical Care Unit (CCU) after delivery. Cases were recruited in the CCU and the controls were obstetric patients after delivery admitted in the labour ward. Controls were unmatched with the cases and were chosen after the indexed case. The presence of risk factors is rare in the general population of obstetric patients in the labour ward. Data was extracted from the patient files and additional information obtained from the patients. Statistical analysis was done in SPSS version 21.0 and the risk factors associated with CCU admission determined using odds ratio with p values at 5% level of significance. We used logistic regression to analyze the predictors of CCU admission.

Results

The study showed that an increase in pre-pregnancy Body Mass Index was a contributing risk factor for admission into the CCU. An increase in gestational age was protective, hence reducing the chance of being admitted into the CCU. The two commonest indications for admission were eclampsia and obstetric hemorrhage.

Introduction

Near miss cases in the post-partum period lead to admission into the Critical Care Unit (CCU) of mothers with severe morbidity. Every day, globally approximately 830 women die from preventable causes related to pregnancy and childbirth⁽¹⁾. Maternal mortality is higher amongst vulnerable groups that include being in LMIC, adolescents and rural populace⁽²⁾. Maternal near misses and mortality are an indicator of quality of obstetric care⁽³⁾.

Pregnancy, childbirth and puerperium are associated with maternal physiological and organ changes. Various complications such as postpartum hemorrhage, eclampsia, septic shock and unsafe abortion during pregnancy and after childbirth can lead to significant pathophysiological alterations which may require management as a critically ill patient. Other factors that could lead to delay in diagnosis and treatment include poor social determinants and health seeking behavior; delay in both access to health facilities and timely provision of care at health institutions ⁽⁴⁾. It is, therefore, necessary for both the patient and the care givers to understand these changes and anticipate admission into a CCU⁽⁵⁾.

The aim of this study focused on the socio-demographic and clinical predictors leading to admissions of critically ill patients after delivery from 24 weeks gestation to 6 weeks post-partum into the CCU, KNH from October 2017- February 2018.

Methodology

Study Design

This study utilized un-matched case-control design to determine risk factors that predicted admission of obstetric patients post-delivery from 24 weeks gestation to 6 weeks post-partum in the CCU. The study was conducted from October 2017 to February 2018.

Study Site

This research was carried out at the Kenyatta National Hospital. KNH is the largest referral and teaching hospital in Kenya. The bed capacity in the hospital is approximately 1,800. The maternity unit has three antenatal and postnatal wards and a labor ward. Labour ward is manned 24 hours by consultant obstetricians and obstetric registrars (SHOs) providing comprehensive and emergency obstetric care. There is access to two maternity theatres that run 24 hours and are supported by auxiliary services such as blood transfusion unit, renal unit and laboratory services. The main Critical care unit (CCU) is a 21 bed capacity facility.

Other auxiliary CCUs include: Acute Room, Cardiothoracic CCU, Neurointensive CCU, NICU, PICU, renal ICU, laboratory and Medical Ward CCU. These units are mainly manned by consultant Anesthesiologists and Anesthesia registrars

(SHOs) with the help of other physicians. Management of critical patients requires a multidisciplinary team approach to care. Index patient evaluation is done with the maternal early obstetric warning signs and the WHO near miss criteria (Box 1).

Box 1: WHO Definition Of Near Miss

World Health Organization (WHO) describes severe maternal morbidity as near death (near miss) but survival from complications which occurred during pregnancy, childbirth or within 42 days of termination of pregnancy

WHO Critical Care Admission Criteria

Organ System	Clinical Indications
General/ Obstetric	Sepsis, severe pre-eclampsia, Eclampsia, HELLP syndrome, Amniotic fluid embolism, PPH
Pulmonary	Severe asthma, severe pneumonia, ARDS, respiratory failure, pulmonary oedema, pulmonary embolism
Cardiac	Valvular heart disease, rheumatic heart disease, CCF, severe hypertension, IHD, MI, peri-partum cardiomyopathy, dysrhythmias
Renal	Acute pyelonephritis, renal failure
Endocrine	Diabetic ketoacidosis, thyrotoxicosis, pancreatitis
Gastrointestinal	Hepatic failure, HELLP Syndrome
Hematological	Coagulation disorders, Anaemia
Surgical/Anaesthetic complications	Anaesthetic complications during surgery for caesarian section, surgical complication on table

Study Population

Cases

All obstetric patients admitted to the Critical Care Unit after delivery from 24weeks gestation to 6 weeks post-partum.

Controls

Unmatched sampling of post-partum mothers who deliver normally in KNH on the same day without admission to CCU.

Sample Size

Sample size was calculated using a Buderer et al formula(5) for comparing 2 proportions. A sample size (n) of 20 women in the CCU(cases) and 40 in the maternity ward (controls) with a ratio of 1:2 was used.

Data Collection

Sequential sampling of cases and controls was done in the CCU and maternity wards respectively after having met the inclusion criteria. Cases were recruited in the CCU and the controls were obstetric patients after delivery admitted in the postnatal ward. Controls were unmatched with the cases and were chosen after the indexed case. Data was extracted from the patient files and additional information obtained from the patients using a structured questionnaire. We obtained data on the socio-demographic factors such as age, parity, social class, booking status, level of education, smoking/drug use, alcohol intake history and also pre- pregnancy Body Mass Index (BMI) was evaluated (Box 2). Admission into the CCU admission of KNH adapted the WHO near miss criterion (Box 1).

Box 2

WHO BMI Classification
Underweight: <18.5
Normal weight: 18.5-24.9
Overweight: 25.0-29.9
Class 1 Obesity: 30.0-34.9
Class II Obesity: 35.0-39.9

Data Analysis

Data was entered and managed in pre-coded Microsoft Excel data entry sheet as data collection progressed. At the end of data entry and cleaning, data was exported to SPSS version 21.0 software for statistical analysis. The study population was described using demographic and clinical characteristics by summarizing categorical data into percentages and continuous data into means or medians. Indications for CCU admissions were presented as proportions. CCU parameters were presented as means and percentages as appropriate. Cases and controls were compared using Chi square test of associations for categorical data, independent t test compared means. The risk factors for CCU were based on odds ratios. Multiple logistic regression analysis determined factors predicting CCU admission among obstetric patients. Statistical test was interpreted at 5% level of significance.

Ethics

The study was approved by the Kenyatta National Hospital/ University of Nairobi Ethical and Research Committee and KNH administration (Protocol Number -P276/05/2017). An informed consent was obtained from each eligible subject.

Results

A total of seventy one patients were recruited to the study. Fifty subjects were selected into the control group and twenty one into the case group.

Table 1; The mean ages in the two groups were (28.3+/-6.4 versus 28.1+/- 6.4; $p = 0.944$) and mean parities (2.5+/-1.4 versus 2.0+/-1.0; $p = 0.204$) which was similar for both groups and was not statistically significant. The mean gestation at delivery in the two groups were 35.7 versus 38.9; $p = <0.001$. This was statistically significant.

The level of education in the two groups was almost evenly distributed in the two groups (Cases: 50% lower, 50% upper; Controls: lower 42.9%, upper 57.1%). Multiparity was noted in the two groups. However, these were not statistically significant. It was also noted that amongst the cases there was less antenatal clinic attendance in comparison to the controls (SD 3.0 versus SD 4.3; $p = 0.004$). In the case group, 4 patients had a history of alcohol consumption and none in the control group ($p = 0.006$).

Table 1: Socio-demographic characteristics and clinical characteristics of postpartum cases and controls admitted at CCU, Kenyatta National Hospital (October 2017-February 2018)

Variable	Cases n (%)	Controls n (%)	p-value
Mean age years (SD)	28.2 (6.4)	28.1 (6.4)	0.944
Marital status			
Married	19 (90.5)	46 (92.0)	0.833
Single	2 (9.5)	4 (8.0)	
Education level			
None	0	1 (2.0)	0.782
Primary	10 (50.0)	20 (40.8)	
Secondary	8 (40.0)	20 (40.8)	
Tertiary	2 (10.0)	8 (16.3)	
Mean gestation at delivery (SD)	35.7(4.4)	38.9(1.3)	<0.001
Mean ANC visits (SD)	3.0(1.0)	4.3(1.8)	0.004
Alcohol use			
Yes	4 (19.0)	0	
No	17 (81.0)	50 (100.0)	0.006
Mean parity (SD)	2.5 (1.4)	2.0 (1.0)	0.204
Mean BMI (SD)	33.7 (8.2)	27.8 (7.0)	0.003

*BMI: Body Mass Index

Analysis of the body mass index in the two groups was conducted. The mean Standard deviation in the cases and control was 33.7+/- 8.2 and 27.8+/- 7.0 respectively which were statistically significant. Further sub - analysis of the BMI shown in Figure 1 showed one subject in the control group was underweight (2%). For normal weight category: cases 2 (9.5%) and controls 15 (30%). In the overweight category; cases 5(23.8%) and controls 20 (40%). The majority of cases were in Class I and II of obesity showing Class I overweight category; cases: 8(38.1%) and controls: 9(18%) and Class II overweight; cases 6 (28.6%) and controls 5(10%) which was statistically significant with a p value of 0.003.

The number of antenatal clinic visits was statistically significant with the mean being; cases: 3.0+/- 1.0 and controls: 4.3+/-1.8 with a p value 0.004. The gestation by age was also statistically significant with averages of 35.7+/-4.4(cases) versus 38.8+/-1.3(controls); p value <0.001.

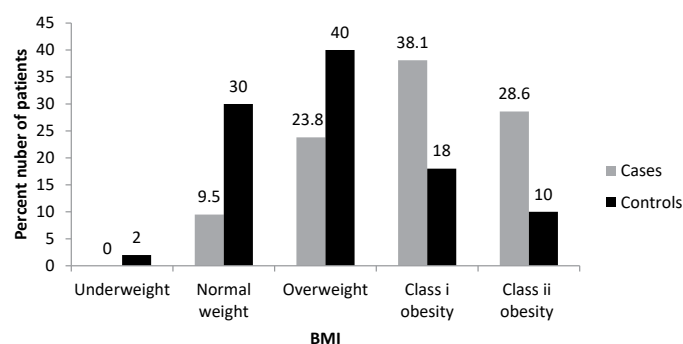


Figure 1: Comparison of Body mass Index of cases and controls at Kenyatta National Hospital of patients admitted into the CCU (October 2017- February 2018)

In the overall analyses using logistic regression as shown in Table 2, various factors were analyzed; age in years, BMI, alcohol use, ANC attendance and Gestational age at delivery. The statistically significant factors were BMI and Gestational age at delivery. A BMI odds ratio of 1.13 (CI 1.01-1.26) with a $p = 0.036$ meaning that an increase of one unit of the BMI would have a 13% risk of admission into the CCU. An odds ratio of 0.4(CI 0.2-0.7) for the gestational age at delivery with a $p = 0.005$, was protective thus meaning there was a 60% reduced risk of admission into the CCU with increased gestational age at delivery. The other factors; age, alcohol use, ANC attendance, mode of delivery, associated illness during pregnancy, previous obstetric complications and alarming signs during ANC were found not to be statistically significant.

Table 2: Logistic regression of predictors associated with CCU admission of postpartum women at the Kenyatta National Hospital(October 2017-February 2018)

Variable	OR (95% CI)	P value
Age in years	1.0 (0.9-1.2)	0.884
BMI	1.13 (1.01-1.26)	0.036
Alcohol use	-	0.999
ANC attendance	-	0.999
Gestational age at delivery	0.4 (0.2-0.7)	0.005

*BMI: Body Mass Index

*ANC: Antenatal Clinic

Discussion

In this study, amongst the socio-demographic and clinical predictors, the main findings were BMI and higher gestational age. The Body Mass Index- OR 1.13 (95 CI 1.01-1.26; $p = 0.036$) was found to be directly associated with admission into the CCU. A higher pre-partum BMI was shown to be associated with adverse obstetric outcome, therefore there was a 13% increase in CCU admission in those with high BMI. A gestational age of <36 weeks was a contributing factor to critical care admission.

The mean for the two groups was 35.7 and 38.9, OR: 0.4 (0.2-0.7; P= 0.005). This means that an increase in gestational age is 60% protective.

In the study, the commonest indications for admission into the CCU were eclampsia (38%) and obstetric hemorrhage (14.3%). It was thought that possibly eclampsia could have predisposed to pre-mature delivery and adverse outcomes of the mothers. The high Body Mass Index amongst the cases could have predisposed the patients to metabolic conditions like gestational diabetes which probably went undiagnosed leading to severe morbidity or pre-mature delivery. A lower gestation age posed a higher risk of admission into the CCU possibly due to the presence of the high risk obstetric complications or undiagnosed Non-Communicable Diseases (NCDs), which culminated in severe maternal morbidity.

KNH being the biggest referral facility in the country, most complicated cases were managed in the facility. The results of this study were comparable to a study by Abenhaim et al (6) which concluded that an increase in BMI leads to severity of the obstetric condition. The link between the high BMI and admission into the CCU could be attributed to the possibility of having missed out associated co-morbidities such as hypertension or diabetes during the pre-natal period. According to the Kenya Demographic Health Survey 2014, maternal mortality rate is at 362 deaths per 100,000 live births. Some of the direct causes of maternal morbidity / mortality were eclampsia and obstetric hemorrhage these findings were similar to this study. (7)

Firstly, this study used the STROBE Guidelines. Additionally gainful insights have been provided on the predictors of obstetric complications requiring admission into CCU. Limitation included the lack of follow up of the mothers who were admitted in the CCU. The maternal - infant outcome may have comprehensively given insight on the pregnancy outcomes, therefore future longitudinal studies should be conducted.

There is need to have critical care units in-cooperated in the Obstetric Department and a strengthened high risk evaluation of mothers throughout pregnancy, childbirth and puerperium.

Conclusion

This study therefore calls for routine screening of mothers due to high suspicion of Non-Communicable Diseases (NCDs); hypertension and diabetes especially in those with a high BMI and evaluation of lifestyle. Increased Antenatal contact with provision of comprehensive care, high risk detection and management reduces the risk of near misses in mothers and CCU admission. There is need to provide health providers with knowledge and skills for diagnosis and management of high risk pregnancies.

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References

1. Maternal mortality: who, when, where, and why. Lancet [Internet]. 2006 [cited 2016 Sep 6]; Available from: <http://www.sciencedirect.com/science/article/pii/S014067360669380X>.
2. Lataifeh I, Amarin Z, Zayed F. Indications and outcome for obstetric patients' admission to intensive care unit: a 7-year review. J Obstet [Internet]. 2010 [cited 2017 Apr 25]; Available from: <http://www.tandfonline.com/doi/abs/10.3109/01443611003646298>.
3. Faponle A, Adenekan A. Obstetric Admissions into the Intensive Care Unit in a Sub-urban University Teaching Hospital. Nepal J Obstet Gynaecol [Internet]. 2012 Sep 2 [cited 2016 Sep 6]; 6(2):33-6. Available from: <http://www.nepjol.info/index.php/NJOG/article/view/6754>.
4. Adeniran A, Bolaji B, Fawole A, Oyedepo O. Predictors of maternal mortality among critically ill obstetric patients. Malawi Med J [Internet]. 2015 [cited 2016 Sep 6]; Available from: <http://www.ajol.info/index.php/mmj/article/view/116230>.
5. Mungai K. Analysis of obstetric admission patterns and management in the critical care unit, Kenyatta national hospital. 2014 [cited 2016 Sep 6]; Available from: <http://erepository.uonbi.ac.ke/handle/11295/95368>.
6. Abenhaim HA, Kinch RA, Morin L, Benjamin A, Usher R. Effect of prepregnancy body mass index categories on obstetrical and neonatal outcomes. Arch Gynecol Obstet [Internet]. 2006 Nov 21 [cited 2018 Jun 6]; 275(1):39-43. Available from: <http://link.springer.com/10.1007/s00404-006-0219-y>.
7. Kenya 2014 Demographic and Health Survey. [cited 2017 May 18]; Available from: <https://dhsprogram.com/pubs/pdf/fr308/fr308.pdf>.

A Case Report and Review Of Literature.

Epidural Saline For Severe Postdural Puncture Headache In Nakuru County Referral Hospital, Kenya

Declaration

We the authors declare there was no funding, interests or conflict of interests when reporting this case summary.

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Abstract

Post Dural Puncture Headache (PDPH) has a high incidence of up to 50% and may be associated with serious complications such as subdural hematoma, seizures and sagittal sinus thrombosis. Although majority resolve spontaneously, it may cause severe discomfort for patients.

Other serious causes of headache should be ruled out before diagnosing postdural puncture headache.

Bed rest, adequate fluids and non-opioid analgesics are the first choice treatment. Noninvasive therapies like theophylline, caffeine, sumatriptan and ACTH can be an alternative but evidence-based recommendation is lacking.

The epidural blood patch remains the mainstay of severe post dural puncture headache therapy. Epidural saline patch has been demonstrated successfully in patients with postdural puncture headache who have contraindications for Epidural blood patches. Epidural saline appears to be of limited value for established PDPH. The successful use of epidural saline, administered as bolus or infusion, continues to be reported occasionally under exceptional circumstances.

Introduction

PDPH is a debilitating condition that is distressing to the patient as well as the Anesthetist. It presents in patients who have had neuroaxial blockade and is very characteristic. The patient will present from the 3rd day with a severe frontal occipital headache, which is worse when they sit up from a lying down position. It may be accompanied by neck stiffness, photophobia and nausea.

Case Presentation

We present Patient M.W.K. an antenatal mother with a history of one previous scar, who underwent an elective caesarian section uneventfully at a private hospital in Nakuru County. Spinal Anesthesia was done with a gauge 25 Spinal Needle and 10mg of Heavy Bupivacaine was administered. Only one attempt was made.

Surgery ended uneventfully. The patient was discharged on day 3 without any complaints.

Four days later, the patient was admitted to our hospital with complaints of severe headache accompanied with neck stiffness, not relieved by analgesics or fluid intake.

Investigations done included Full Blood Count, Head CT-Scan and the C-reactive protein (CRP) which was 39.40mg/l (normal upper limit is 10mg/l). The full hemogram and CT scan were normal.

With the patient in sitting position, at the level of L3-L4 space, the epidural space was identified through the Loss of Air resistance technique. However, free flowing CSF was found. While still seated, the needle was relocated to L2-L3 space. The epidural space was identified through the Loss of Saline resistance technique. Free flowing CSF was again found flowing. This confirmed there was too much CSF flow in the epidural space. With the patient now in lateral position, the last attempt was made at the L2-L3 space and Saline resistance technique used. Free flowing CSF was still present.

Epidural blood patch could NOT be done because:

- Too much CSF flow, so it was not clear if we were in the Subarachnoid or epidural space.
- Too much flow "may" have washed out the clot hence need for another patch.

What did we do? In consultation with a senior colleague, a decision was made to use Normal Saline 0.9% into the epidural space. We were to inject up to 30mls of normal saline or until the patient felt a sensation of severe back or head pressure.

After injecting 15mls, the patient reported an intense pressure headache at the occiput radiating to the lumbar region. Injection of saline was stopped at this moment, and patient was placed in supine position for 15 minutes. An attempt to sit up the patient was made. The patient reported no Post Dural puncture headache on assuming the sitting position.

Patient was discharged the following day on Betapyn 2 tablets twice a day for 2 days. She was booked for a review in one week's time. The patient reported no headache after one week.

Introduction

Neuroaxial regional blockade is a commonly used anesthesia technique for emergency caesarian section, due to associated reduced post-operative pain, reduced blood loss, advantage of an awake mother and increased safety and comfort for mother and fetus. Single dose subarachnoid block is preferred due to the ease of administration. It also provides rapid onset of surgical anesthesia with failure rates as low as 1%.^{1,2}

Using pencil-point spinal needles as opposed to cutting bevel needles has been shown to be beneficial for preventing PDPH in patients undergoing Cesarean section without increasing any potential adverse effects.³

The incidence of PDPH is estimated to be between 30-50% following diagnostic or therapeutic lumbar puncture, 0-5% following spinal anesthesia and up to 81% following accidental dural puncture during epidural insertion in the pregnant woman. PDPH may be associated with serious complications such as subdural hematoma, seizures and sagittal sinus thrombosis. Although majority resolve spontaneously, it may cause severe discomfort for patients.⁴

There are other serious causes of headache that should be ruled out before diagnosing postdural puncture headache. These include hypoglycemia, meningitis, encephalitis, dehydration, caffeine withdrawal, vascular migraine, cerebral vein thrombosis, cerebral infarction, subdural hematoma, subarachnoid/subdural hematoma, pre-eclampsia, tension headache, benign intracranial hypertension, pneumocephalus, lactation headache and space occupying lesions.⁴⁻⁵

A non-contrast head computer tomography (CT scan) was requested for our patient on admission to rule out majority of the other causes of headache.

Bed rest, adequate fluids and non-opioid analgesics are the first choice treatment⁶. Noninvasive therapies like theophylline, caffeine, sumatriptan and ACTH can be an alternative. However, an evidence-based recommendation is lacking⁷. Conservative measures including bedrest, caffeine,

rehydration and nonopioid analgesics were instituted on day three post subarachnoid block when the post dural puncture headache began but were not successful.

The epidural blood patch remains the mainstay of severe post dural puncture headache therapy.⁸ However, epidural blood patch carries some risks, such as subdural hematoma, pneumocephalus, exacerbation of postdural puncture headache and new dural puncture. Contraindications for Epidural blood patch include anticoagulation/coagulopathy, infection at the injection site, and patient refusal or lack of cooperation.

Our patient had none of the mentioned contraindications. However we experienced difficulty in locating the epidural space due to the high flows of cerebral spinal fluid. Once we achieved the loss of air or saline resistance, a continuous flow of cerebral spinal fluid was observed. Without ultrasound or imaging guidance, it was impossible to confirm if the needle was in a CSF flooded epidural space or in the subarachnoid space. An epidural blood patch could therefore not be done, and epidural saline injection was used instead.

Epidural saline patch has been demonstrated successfully in patients with postdural puncture headache who have contraindications for Epidural blood patches.^{9, 10, 11}

Epidural saline, as bolus and infusion, has a long history of use for treatment of PDPH. Bolus injections of epidural saline (usually 20-30 ml, repeated as necessary if a catheter is present) have been reported to produce prompt and virtually universal relief of PDPH, yet the practice is plagued by an extremely high rate of headache recurrence. This transient effect is not surprising as increases in epidural pressure following bolus administration of saline have been demonstrated to return to baseline within 10 minutes. Favorable results achieved with this approach have been speculated to represent the mechanical reapproximation of a dural flap (the "tin-lid" phenomenon). However, bolus administration of saline for treatment of PDPH has been convincingly shown to be inferior to the EBP, especially when headaches are secondary to large-bore needle punctures. Overall, epidural saline appears to be of limited value for established PDPH. Nevertheless, the successful use of epidural saline, administered as bolus or infusion, continues to be reported occasionally under exceptional circumstances.¹²

In our patient, one bolus injection of 15 mls of saline was given. Injection was stopped once patient developed severe pressure at the back referred up the spine to the neck, indicating a rise in epidural pressure. No further continuous infusion was required, and patient reported no post dural puncture headache 15 minutes later after sitting up from the supine position.

Conclusion

Epidural saline patch is a suitable alternative where epidural blood patch is contraindicated or cannot be used as in our case study.

References

1. Riley ET, Cohen SE, Macario A, et al. Spinal versus epidural anesthesia for cesarean section: a comparison of time efficiency, costs, charges, and complications. *Anesth Analg*. 1995; vol. 80 (pg. 709-12).
2. Fettes PD, Jansson JR, Wildsmith JA. Failed spinal anaesthesia : mechanisms, management, and prevention. *Br J Anaesth*. 2009; vol.102 (pg.739-48).
3. Lee SI¹, Sandhu S², Djulbegovic B³, Mhaskar RS³. Impact of spinal needle type on postdural puncture headache among women undergoing Cesarean section surgery under spinal anesthesia: A meta-analysis. *J Evid Based Med*. 2018 Aug; 11(3): (136-144).
4. Turnbull DK and Shepherd DB Post-dural puncture headache: pathogenesis, prevention and treatment. *Br. J. Anaesth*. 2003; 91 (5):718-729.
5. Landman UN, Jerome RM and Glass PS. Subdural hematoma after atraumatic spinal. *J. Clin. Anesthesia* 2005; 17: 379-81.
6. Kleine-Brüggeney M, Kranke P and Stamer UM. Prophylaxis and therapy of postdural puncture headache--a critical evaluation of treatment options. *Anesthesiol Intensivmed Notfallmed Schmerzther*. 2011 Jul; 46(7-8):516-24. Doi: 10.1055/s-0031-1284471.
7. Apfel CC, Saxena A, Cakmakaya OS, Gaiser R, George E and Radke O. Prevention of Postdural Puncture Headache after Accidental Dural Puncture: A Quantitative Systematic Review. *Br J Anaesth*. 2010; 105(3):255-263.
8. Boonmak P and Boonmak S. Epidural blood patching for preventing and treating post-dural puncture headache. *Cochrane Database Syst Rev*. 2010 Jan; 20(1):CD001791.
9. Liu SK1, Chen KB, Wu RS, Lin BC, Chang CS, Liu YC, Hung CJ Management of postdural puncture headache by epidural saline delivered with a patient-controlled pump--a case report. *Acta Anaesthesiol Taiwan*. 2006 Dec;44(4):227-30.
10. Kara I, Ciftci I, Apiliogullari S, Arun O, Duman A and Celik JB. Management of postdural puncture headache with epidural saline patch in a 10-year-old child after inguinal hernia repair: a case report. *J Pediatr Surg*. 2012 Oct;47(10):e55-e57.
11. Ravi Prakash1, Shefali Gautam1, S. Jafa3, Archana1, Asian Postdural puncture headache: Is epidural saline patch effective – A case report *Journal of Medical Sciences* | Oct-Dec 2014 | Vol 5 | Issue 4.
12. Brian E. Harrington and Miguel Angel Reina, Post dural puncture headache NYSORA continuous medical education, <https://www.nysora.com/foundations-of-regional-anesthesia/complications/postdural-puncture-headache/>.

ERRATUM

Kindly note that the article carried in the 5th KJACCM Edition - Vol 5 entitled “Inadvertent Placement of Central Venous Catheter in the Azygos Vein” had images 3 and 4 mislaid and erroneously labelled.

We highly apologise for that and below are the correct images and their correct labels.



Figure-3: Chest X ray PA-Right IJV Central Venous Catheter, abrupt bend of Catheter tip within the SVC



Figure-4: Chest X ray PA-Right IJV Central Venous Catheter Tip within the SVC, after repositioning

Effect Of Peri-Operative Intravenous Fluids On Recovery Of Intestinal Function After Caeserean Delivery Under Spinal Anaesthesia At Kenyatta National Hospital

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Abstract

Background

Postoperative Ileus refers to the delay of the resumption of regular bowel movement following intra-abdominal surgery lasting 2 to 3 days. It is a major problem after abdomino-pelvic operations that delays recovery. Several studies on adults have investigated the effect of "Liberal" versus "Restrictive" peri-operative fluid regimes on ileus after abdominal surgery in non-obstetric population. Restrictive fluid regimen (<2700 mls) intraoperatively was found to be associated with enhanced intestinal recovery although data remains scanty in cesarean delivery.

During caesarean delivery under spinal anaesthesia different regimes of intravenous fluids are used perioperatively. Literature review revealed no studies conducted on their effect on intestinal recovery or data on the incidence of post-operative ileus in the obstetric population. A study was necessary to establish incidence ileus after surgery and assist in developing a protocol for peri-operative intravenous fluids administration during caesarean delivery.

Objective

The aim of this study was to establish the effect of perioperative intravenous fluid volume on recovery of intestinal function after caesarean delivery under spinal anaesthesia. We also sought to find out the incidence of post-operative ileus in this population.

Materials and Methods

This was a prospective observational study conducted after getting ethical approval from The Kenyatta National Hospital/ University of Nairobi Ethics and Research committee. 150 patients who delivered through caesarean section under spinal anaesthesia at KNH between March 2016 and May 2016 were recruited using consecutive sampling technique and followed up from admission into theatre for up to 3 days post-operatively in the postnatal wards. Intravenous fluid volumes administered preoperatively, intraoperatively and post-operatively were documented and their effect on intestinal recovery evaluated. The outcomes of interest for recovery of intestinal function were time to first normal bowel sounds, time to tolerance of the first solid food, time to first flatus and time to defecation.

Collected data was analysed using SPSS version 20. Descriptive statistics was used to determine prevalence of ileus after surgery. Regression analysis was undertaken to establish how various independent variables influenced development of post-operative ileus. Paired t-test, Pearson correlation test and analysis of variance (ANOVA) were run to determine association between perioperative intravenous fluids and development of postoperative ileus.

Results

Data from 150 patients was collected and analysed. Majority of patients (50%) were between 24 and 32 years, had BMI range of 26 to 32 and average parity was 3 to 5. Prevalence of ileus after caesarian delivery was 5.6%. The results of Pearson correlation test and ANOVA showed that preoperative intravenous fluids (normal saline 501 – 1000 mls) significantly influenced duration to first flatus ($P=0.011$), time to normal bowel rounds ($P=0.029$) and time to first oral feed($P=0.045$).

Conclusion

Prevalence of postoperative ileus in obstetric population was noted to be similar to that of other abdominal surgery patients in studies performed elsewhere. There was a significant association between preoperative intravenous fluid administration and postoperative recovery of intestinal function. It was noted in this study, that 501-1000mls of normal saline preoperatively led to enhanced recovery of intestinal function compared to other fluid volumes. Other intraoperative fluid volumes were found to have no significant effect on gut recovery after operation. Since this was an observational study and hence difficult to draw conclusions from, a further randomized controlled study should be considered to better understand the role of perioperative fluid volumes on the recovery of intestinal functions.

Introduction

Caesarean delivery involves extraction of a baby through an incision in the abdomen and uterus. Gastrointestinal manipulation during the procedure predisposes the patient to post-operative ileus. Post-operative ileus is defined by at least two of the following 5 signs on or after the third postoperative day:

- i) Nausea and vomiting
- ii) Delayed tolerance of oral diet
- iii) No gas or stool passed during the preceding 24hrs
- iv) Abdominal swelling
- v) Evidence of ileus on abdominal X-Ray (5,6)

Physiologic ileus resolves within 2-3 days (small intestines and stomach resume function within hours and 1-2 days respectively whereas large gut regains activity within 3-5 days) after sigmoid motility returns to normal. Morbidity due to ileus is characterized by discomfort and pain, atelectasis/hypostatic pneumonia, abdominal distension, nausea and vomiting (4, 5, 6, 7)

Both Spinal and general anesthesia may be used for caesarian delivery. They both have different risks intra-operatively and post-operatively. General anesthesia is associated with increased blood loss intra-operatively due to halogenated volatile agents, thromboembolic complications, pulmonary infection, impaired gastrointestinal motility and prolonged length of hospital stay compared to spinal anesthesia (8, 9).

Literature Review

The post-operative state is the most common cause of ileus (4). Pathogenesis of ileus involves the autonomic and central nervous systems as well as local and regional substances which lead to ileus (10).

Pathophysiology Of Ileus

Post-operative ileus develops in phases. First phase involves the sympathetic nervous system, while the second phase involves hormonal and inflammatory mechanisms. The final phase involves para-sympathetic nervous system stimulation and plays a critical role in the resolution of ileus (4).

Neurological phase: This phase involves the enteric nervous system (ENS) and the sympathetic nerves. Glial cell dysfunction of the ENS could lead to interruption of the intestinal mucosal barrier. Alpha-2 adrenergic receptors in the inflamed muscularis mucosae are then stimulated worsening ileus by increasing synthesis of messenger RNA of the inducible nitric oxide synthetase (i-NOS mRNA) with the release of nitrogen monoxide (NO) which leads to ileus (4).

Hormonal and Inflammatory phase: As the neurological phase declines, there is increased inflammation of intestinal mucosa that involves monocytes, macrophages and mast cells

that produce pro-inflammatory molecules and auto-regulate themselves. Intestinal manipulation leads to inflammation by activating dendritic cells which produce interleukin-12 (IL-12). IL-12 activates T1 helper lymphocytes (T1H) which migrate to regions that have not been manipulated causing inflammation by production of alpha interferon (IFN alpha) through recruitment of microphages. This is called the "field effect". During this second phase, intestinal mucosal barrier permeability is raised which increases inflammation (4).

Phase of ileus resolution and vagal activation: Increased vagal tone reduces manipulation-induced intestinal inflammation. The mediators include nicotinic alpha 7 acetylcholine receptors (alpha 7 and ACHR) and 5-hydroxytryptamine 4 receptors (5-HT4R). Stimulation of 5-HT4R increases acetylcholine release from myenteric cholinergic neurons. This activates alpha-7 - nAChR on monocytes and macrophages hence reducing the inflammatory response. Vagal system mediates the phase of resolution (4).

Other causes of post-operative ileus include:

- i) Drugs (general anaesthesia, opioids, anticholinergics, amitriptyline).
- ii) Trauma (fractured ribs, fractured spine).
- iii) Pneumonia/peritonitis/Sepsis.
- iv) Endocrine and metabolic disorders (Diabetes mellitus, Addison's disease, myxoedema coma and low potassium levels).
- v) Biliary and renal colic.
- vi) Cardiopulmonary insufficiency (e.g. myocardial infarction).
- vii) Head injury and trauma to spinal cord.
- viii) Retroperitoneal pathologies (e.g. hematomas) (11).

Perioperative fasting causes intravascular hypovolemia which, compounded with intraoperative hemodynamic instability and post-operative fluid deficit, can cause delayed intestinal recovery due to a compromised splanchnic circulation (8).

European Society of Anaesthesiology guidelines recommend that adults should take clear fluids and solid food up to 2 hours and 6 hours before surgery respectively (12). Contrary to these guidelines, patients in our hospital are maintained nil per oral for long periods before surgery.

The common indications for intravenous fluid administration in elective surgery are: -

- a) Correction of preoperative fluid deficits for maintenance of CVP.
- b) Control of intra-operative and post-operative hemodynamics.
- c) Avoidance of blood transfusion and post-operative renal failure.
- d) Substitution for non-enteral nutrition post-operatively.
- e) Prevention of hypotension during regional anesthesia/analgesia (10).

Effective interventions targeting the different phases of ileus that have been studied include Alvimopan (an opioid receptor antagonist) and Lidocaine which act on neurological phase of ileus by antagonizing the effects of opioids and reducing pain respectively (13, 14). Prokinetic agents such as intravenous magnesium sulfate (40mg/kg bolus to 10mg/kg infusion during operating period) and metoclopramide decrease the interval of return of transit (15, 16).

Mastication of gum mimics dietary intake hence stimulates vagal nerves, which has an anti-inflammatory effect. This leads to cephalo-caudal stimulation of digestion by increasing the activity of neural and humoral factors on multiple parts of the gastrointestinal tract. This increases serum concentration of peptide hormones as well (17, 18).

Reduction of intravenous fluids (by early resumption of diet) and coffee intake post-operatively reduces the incidence of ileus (19, 20, 21).

Perioperative Fluid Management and Intestinal Recovery

Studies in surgery other than caesarean delivery have demonstrated that peri-operative fluid management influences intestinal recovery post-operatively. However, the effect of peri-operative fluid management on post-operative intestinal recovery remains imprecise (10).

A cohort study done to establish the causes of ileus after abdominal surgery demonstrated statistically significant correlation with volume of blood loss during operation, duration of surgery and total dose of opioids (22)

Goal-directed fluid therapy (GDT) effect on recovery of internal function post-operatively has been investigated in randomized controlled trials. Goal-directed therapy involves use of cardiac output, pulse rate, venous (CVP) and arterial (MAP) pressure to guide intravenous fluid therapy. Studies done involved patients of varying fitness undergoing colorectal surgery (as determined by cardiopulmonary exercise testing - CPET) (23), general surgery patients randomized to GDT (with standard fluid management) versus standard fluid management groups (vascular, upper gastrointestinal and hepatobiliary surgeries) (24) and general urologic and gynecologic patients randomized to GDT and standard fluid management control groups. (25)

Standard fluid management aimed to optimize total blood volume by administering 10mls/kg of crystalloid fluid followed by 8ml/kg/hour in the duration of surgery.

The outcomes of interest included readiness for discharge, duration of hospital stay, complications after operation, and period to toleration of first solid feed and antiemetic requirements (23, 24, 25).

Goal-directed therapy (with standard fluid management) was compared to standard fluid management it was shown

to prolong time to readiness for discharge and length of hospital stay in fit patients, but it was not significant in unfit patients (23). When GDT (without standard fluid therapy) was compared against standard fluid management, it was shown to reduce duration of hospital stay, time to tolerance of first oral feed, nausea and vomiting after surgery, antiemetic requirements and complications postoperatively (Wound sepsis, Pneumonia, Urinary Tract Infection, Pulmonary emboli and Arrhythmias) (24,25).

The outcome of interest and surgeries were different in each study hence the study population was not homogenous (23, 24, 25).

Stratified Meta-analyses of randomized controlled trials of various methods of fluid therapy that have been done involve comparisons of restricted (<1.75 liters/d), liberal (>2.75 liters/d) and fluid balance therapy (1.75-2.75 liters/d) (27); perioperative liberal fluid therapy (LVR) versus goal directed therapy (GDT) or restricted fluid regime (34) and standard, restrictive and supplemental fluid regimes (28).

The outcomes of interest were post-operative complications (Wound dehiscence, anastomotic leak, wound sepsis, pneumonia, arrhythmias, and urinary tract infection), duration to first flatus, normal bowel movements, (27, 28) and total length of hospital stay (26, 27, 28).

Analysis revealed statistically significant reduction in duration to first flatus, normal bowel movements, complications postoperatively and total period of hospital stay for fluid restricted patients compared to liberal and goal directed fluid therapy patients (27, 28).

This benefit was also found in patients maintained in a state of fluid balance compared to patients who were maintained on fluid restriction and liberal fluid therapy (26).

Restrictive versus liberal fluid therapy studies have been done with patients being randomized in surgeries varying from laparoscopic cholecystectomy (29) and elective colorectal surgery (30, 33, and 34) to gastric resection and pancreaticoduodenectomy (31, 32).

The outcomes of interest included post-operative pulmonary function, exercise capacity (30), duration to first flatus, first bowel motion (30, 31, 33), post-operative morbidity and complications (31, 32, 34).The duration of hospital stay was also included in the studies (29, 30, 31, 32, 34).

Pulmonary function and exercise capacity were shown to be better in the liberal group compared to restricted group (29).

Sodium and fluid restriction did not shorten duration to first flatus and bowel movements (30). Fluid restriction (without sodium restriction) shortened the period to first flatus and defecation (31). It also significantly reduced post-operative complications (vomiting, wound dehiscence, wound sepsis,

peritonitis, pneumonia and atelectasis) and the length of hospital stay (31, 32, 34)

The study outcomes varied because of differences in sample sizes, extent of different surgeries and volumes of fluid and electrolytes administered in restrictive and liberal groups (29, 30, 31, 32, 33, 34).

Post-operative nausea and vomiting was evaluated as a primary outcome in laparoscopic cholecystectomy and gynecological surgery with patients randomized to restrictive and liberal fluid therapy groups. Restrictive fluid therapy significantly reduced the incidence of nausea and vomiting post-operatively (35).

Other studies have been conducted with the objective of minimizing intraoperative variation of pulse pressure by randomizing gastrectomy and colectomy patients into restrictive Ringer's lactate group (R-RL), goal-directed Ringer's lactate group (GD-RL) and a colloid (hydroxyethyl starch) goal-directed group (GD-C) (36) vis-a-vis maximization of intraoperative stroke volume guided by Esophageal Doppler monitoring, with (colorectal surgery) patients being randomized into esophageal Doppler (guided maximal stroke volume Doppler group, D-group) and normal body weight with zero balance group (Zero balance, Z-group)(37).

The outcomes of interest were duration to passage of flatus, length of hospital stay and postoperative complications (wound sepsis, wound dehiscence, anastomotic leak, pneumonia, arrhythmias) (36, 37)

Goal-directed hydroxyethyl starch therapy was found to be superior to goal directed lactated Ringers therapy and restrictive lactated Ringer's therapy in shortening time to passage of flatus and length of hospital stay (36) while goal directed fluid therapy was found to add no benefit compared to zero balance therapy (and normal body weight) in reducing post-operative complications or duration of hospital stay (37)

Overall, restrictive management fluid regimes were found to have better abdominal surgery outcomes compared to liberal and goal directed fluid therapy regimes.

Caesarean Delivery and Ileus

Caesarean delivery is a common obstetric surgery which triggers postoperative changes in autonomic nervous system leading to decreased bowel movements. The ileus that ensues leads to pain after surgery, abdominal distention, reduced tolerance to feeds and delayed recovery (38).

The indications for caesarean delivery include previous classical caesarean delivery, placenta previa grade 4 and placental abruption, breech presentation or transverse lie, genital herpes/warts in the mother, severe pre-eclampsia, uterine malformations (bicornuate uterus), cervical dystocia, ovarian and cervical malignancies, fetal distress and maternal request (38).

The type of uterine incision during caesarean delivery is associated with different degrees of maternal morbidity (puerperal infection, delayed wound healing, blood transfusion, hysterectomy). A systematic meta-analysis revealed that maternal morbidity was significantly higher in classical and inverted "T" incisions compared to low transverse caesarean incisions (39)

In pregnancy, the cardiovascular system undergoes physiological changes in order to ensure adequate oxygen and nutrient supply to the fetus. The plasma and red blood cell volume expands by 1500-1600mls which increases the stroke volume and the cardiac output. Placental auto-transfusion (300-500mls) increases blood volume. The increase helps in mitigating the blood loss of delivery but placental auto-transfusion in addition to fluid loading and infusion can lead to a liberal intra-operative fluid administration state (40).

Intravenous Fluid Therapy And Caesarean Delivery

During caesarean deliveries, patients are predisposed to intraoperative hypotension due to neuraxial sympatholytic effect, aorto-caval compression and preoperative fasting. Fluid co-loading with colloids and crystalloids (10-20 mls/kg) is more effective than the use of crystalloids only. It has also been shown to be superior to pre-loading. Vasopressor drugs (phenylephrine and ephedrine) are also used intra-operatively during post-spinal hypotension and their use is reduced in co-loaded rather than pre-loaded patients (41).

The volume of fluid used intra-operatively in cesarean delivery may exceed the restrictive intra-operative fluid regime that has been studied in general surgery due to fluid loading and administration during episodes of hypotension (3). Intra-operative drugs and mode of anaesthesia are also contributing factors to post-operative morbidity. Spinal anesthesia (compared to general anesthesia) for cesarean delivery has been shown to reduce the need for analgesia after surgery, lowers incidence of nausea and vomiting postoperatively, promotes early mobilization due to better analgesia and enhances faster tolerance to oral feeds (because fewer medications are used intra-operatively that predispose to ileus) (42). Opioids and general anesthetics reduce gastro-intestinal motility. Prokinetics such as metoclopramide enhance gut motility reducing the extent of post-operative ileus (43).

In some obstetric conditions/emergencies, it is challenging to assess the effect of fluid status on intestinal recovery post-operatively. Pre-eclampsia is associated with organ dysfunction and electrolyte imbalance. Fluid restriction is essential to avoid fluid overload (44). Post-partum hemorrhage patients require high volume of fluids for resuscitation and blood transfusion while emergency caesarian delivery patients' volume status cannot easily be ascertained if they are referral patients (45). Therefore, it is difficult to ascertain the effect of perioperative fluid management on recovery of intestinal function after surgery in these patients.

Study Justification

The actual data on post-operative ileus incidence in Africa is not available (14).

Fasting for too long before caesarean delivery at Kenyatta National Hospital leads to fluid deficits whose impact on post-operative ileus has not been established.

During caesarean delivery under spinal anaesthesia different regimes of intravenous fluids are used and their effect on intestinal recovery has not been established. Published data on the incidence of ileus after surgery in obstetric population is lacking and a protocol for peri-operative intravenous fluid use during caesarean delivery has not been established. No study is available locally that demonstrates the optimal obstetric peri-operative intravenous fluid management for enhanced recovery of gut function after surgery. This study was to determine the prevalence of post-operative ileus and assist in developing a protocol for peri-operative intravenous fluid administration during caesarean delivery.

Ileus is common after abdominal operations and is a cause of prolonged hospital stay. There has been no study that compares the economic impact of post-operative analgesic needs and anti-emetic requirements between standard post-operative care and peri-operative intravenous fluid administration.

By demonstrating the effects of peri-operative intravenous crystalloids in the local population, this study was expected to improve the standard peri-operative fluid management. Lack of local peri-operative intravenous fluid administration data at KNH necessitated a study that could justify early feeding.

Study Design

This was a prospective observational study involving patients undergoing caesarean delivery under spinal anaesthesia at the Kenyatta National Hospital. Follow-up was for 3 days post-operatively because this is the time most patients are discharged from KNH after caesarean delivery. Patients not discharged by the third post-operative day (without complications) were followed up until the fifth day. Standard intra-operative and post-operative care was maintained in all patients.

All intravenous fluids administered to patients from theatre up to 3 days post-operatively including intraoperative estimated blood loss (EBL) were documented and the effect on recovery of intestinal function noted.

Patients participating in the study were assessed every hour (from post-anaesthesia care unit) for 4 hours, then 4 hourly until all of the following could be noted: Time to first flatus, time to normal bowel sounds, period of time to first defecation and time taken to tolerance of first solid food. Post-operative antiemetic requirement was also assessed. The study provided evidence for the effects of peri-operative intravenous fluids to post-operative recovery of intestinal function.

Patients under study still received additional treatment if required, and it was certified to be safe for participants since it did not have any interventional component.

Sampling Technique

The sampling was done using consecutive convenience sampling method of all the women undergoing caesarean delivery in labour ward and meeting the inclusion criteria at Kenyatta National Hospital.

The Sampling frame for this study was the list of all women undergoing caesarean delivery under spinal anaesthesia during the study period.

Results

150 patients scheduled for caesarian delivery through spinal anaesthesia were recruited in the study. 2.7% (4 patients) had elective caesarian deliveries while 97.3% (146 patients) underwent emergency caesarian deliveries.

Data was collected from all study subjects and analyzed for effect of perioperative intravenous fluid infusion on recovery of gastro-intestinal function after caesarian delivery under spinal anaesthesia.

Most patients were between 24 and 32 years (50%), with an average BMI of 29 and parity of 3 previous deliveries. Commonest indications for surgery included non-reassuring foetal status (13%), breech presentation 12.2% and Hypertension in pregnancy (9.3%). Difficult foetal extraction in 1.3% of patients (2 patients) was encountered as the only intraoperative complication.

The average duration of surgery was 1.3 hours (range of 1.0 – 1.5 hours) with 68.9% of patients having Estimated Blood Loss (EBL) of 251 mls – 500 mls in comparison with 29.7% patients who had an EBL of 501 mls-1000 mls (Fig 2). Consultants performed most of the surgeries (69.3%) followed by Registrar part II (26.0%) and Registrar part I (4.7%).

Table 1: EBL of Patients

Estimated Blood Loss	N	%
1-250ml	2	.7
251-500ml	102	68.9
501-1000	44	29.7
>1000	2	.7

Intraoperatively, analgesics administered included Diclofenac (69.3%), Paracetamol (55.3%) and Tramadol (41.3%).

Table 2: Intra-operative analgesics

	N	Mean dose
i. Fentanyl	141	24
ii. Tramadol	62	100
iii. Paracetamol	83	15
iv. Diclofenac	104	133
v. Others	27	60

The volume of intravenous fluids administered preoperatively, intraoperatively and postoperatively varied. Preoperatively, 66% of patients (99 patients) received restrictive fluid regime of normal saline (mean = 909 mls), 32% (49 patients) received ringer's lactate (mean = 536 mls) and 1.3% (2 patients) received colloids (mean = 500 mls) and whole blood (mean = 450 mls). During surgery, 67.3% of patients (101 patients) received normal saline (mean 1089 mls), 9.3% (14 patients) ringer's lactate (mean 723 ml), 17.3% (26 patients) normal saline and ringer's lactate (mean 841 ml), 3.3% (5 patients) colloids (mean = 600 ml) and 2.6% (4 patients) received blood transfusion (mean = 463 ml) (Table 3).

Table 3: Intravenous fluids administered preoperatively and intraoperatively

	Preoperatively		Intra-operatively	
	N	Mean	N	Mean
i. Crystalloids a Normal saline 0.9 mls	99	909	101	1089
i. Crystalloids b Ringers lactate mls	49	536	14	723
i. Crystalloids c N/saline + R/lactate			26	841
ii. Colloids mls	1	500	5	600
iii. Blood mls	1	450	4	463
iv. Blood products mls	0	.	0	.

In the first postoperative day, 62% of patients (93 patients) received restrictive fluid regime of normal saline (mean = 1219 ml), 18.7% ringer's lactate (mean = 760 ml), 18.7% ringer's lactate and normal saline and 3.3% colloids (mean = 900 ml). Crystalloids were not administered in the second and third postoperative day but 2% of patients on both days received colloids (mean = 833 ml and 667 ml respectively). Blood was transfused in the second postoperative day to 1 patient (0.6%) (Mean = 500 ml).

Table 4: Fluids administered postoperatively

	Day 1		Day 2		Day 3	
	N	Mean	N	Mean	N	Mean
Post-operative Colloids	5	900	3	833	3	667
Post-operative Normal saline	93	1219	0	.	0	.
Post-operative Ringer's lactate	28	760	0	.	0	.
N/saline + R/lactate	28	672	1	500	0	.
Post-operative Blood transfusion	0	.	0	.	0	.
Other post-operative blood products	0	.	0	.		

Most patients received between 500 – 1000 mls of normal saline and <500 mls of ringer's lactate perioperatively as shown in the table below.

Table 5: Fluids administered perioperatively

	Amount	Preopera-tively		Intra-opera-tively		Post-opera-tively	
		N	Mean	N	Mean	N	Mean
Normal Saline	<500ml	41	41.4	20	17.9	12	10.8
	500-1000ml	39	39.4	59	52.7	58	52.3
	>1000ml	19	19.2	33	29.5	41	36.9
Ringer Lactate	<500ml	45	92.9	16	61.5	28	62.5
	500-1000ml	4	7.1	8	30.8	13	29.2
	>1000ml	0	0	2	7.6	3	8.3
Normal Saline + Ringer Lactate	<500ml			7	26.9	9	32.1
	500-1000ml			18	69.2	16	57.1
	>1000ml			1	3.8	3	10.7

The average time to occurrence of outcomes of interest from surgical incision time include: period of time to first flatus (7 hours), duration of time to normal bowel sounds (10 hours), the time taken to first rescue analgesia (11 hours), period of time to first defecation (32 hours) and time taken to tolerance of first solid food (14 hours) (Table 6).

In the Holte et al study of Liberal versus restrictive fluid regimen for colonic surgery patients, those on restrictive fluid regime took longer to pass flatus (24 hours) and to pass stool (48 hours) (14). This could be due to the invasiveness of surgeries that involved right/transverse/left colon resection compared to minimal gut manipulation during obstetric surgery.

Table 6: Number of hours elapsed from the time of surgical incision

	Mean	Minimum	Maximum
Time to first flatus (hrs)	7	1	36
Time to normal bowel sounds (hrs)	10	1	54
Time to first defecation (hrs)	32	4	85
Time to first rescue analgesia (hrs)	11	1	52
Time to tolerance of first oral feed (hrs)	14	4	36

27.3% (41 patients) of patients developed nausea postoperatively reducing to 18.6% (28 patients) and 14% (21 patients) on second and third day respectively. Anti-emetics were administered to all patients who developed nausea. 14.6% (22 patients) vomited in the first postoperative day which reduced to 8.6% (13 patients) by the third postoperative day. Rescue analgesia was administered to 12.6% (19 patients) on the first postoperative day compared to 8% on the third day after surgery (Table 7). Post-operative opioid (morphine) rescue analgesia could have contributed to post-operative nausea and vomiting as an adverse effect of the drug. This is demonstrated in table 7 whereby patients who experienced post-operative nausea and vomiting exceeded the number of patients who received rescue analgesia.

Table 7: Occurrence of nausea and vomiting versus anti-emetics and rescue analgesia administration post operatively

		Day 1	Day 2	Day 3
No. Of times of anti-emetics in 24 hours	Valid N	42	28	24
	Mean	2	2	4
	Minimum	0	0	0
	Maximum	4	4	5
No. Of times of nausea in 24hrs.	Valid N	41	28	21
	Mean	2	2	2
	Minimum	0	0	0
	Maximum	5	3	4
No. Of times of vomiting in 24 hours	Valid N	22	15	13
	Mean	2	1	1
	Minimum	0	0	0
	Maximum	3	2	2
No. Of times of rescue analgesia in 24 hrs	Valid N	19	15	12
	Mean	1	1	1
	Minimum	0	0	0
	Maximum	3	2	1

Results of paired t-test carried out to find association between age and BMI on recovery of intestinal function after surgery revealed that they did not significantly influence ileus (outcomes of interest) post-operatively (Table 8).

Table 8: Age and BMI versus Ileus

		Time to flatus	Time to bowel movement	Time to defecation	Time to first rescue analgesia	Time to oral feeding
		Mean	Mean	Mean	Mean	Mean
Age	Up to 1 day	28	28	29	29	28
	More than 1 day	30	31	28	30	39
p-value		0.106	0.804	0.567	0.207	0.314
BMI	Up to 1 day	29	29	30	30	29
	More than 1 day	28	30	29	31	32
p-value		0.314	0.743	0.518	0.473	0.565

As shown in the table below, longer duration of surgery was associated with higher EBL, but through Pearson correlation test, there was no significant statistical difference noted.

Duration of Surgery (Hrs)	EBL (Mean in mls)	P-Value
1.0 - 1.20	475	0.672
1.21 - 1.40	525	0.861
1.41 - 1.50	650	0.954

The effect of drugs given intraoperatively on recovery of intestinal function postoperatively was evaluated using paired t-test. Fentanyl significantly ($p < 0.0001$) reduced time to rescue analgesia postoperatively but did not have significant effect on recovery of gut after surgery. Other drugs also had no significant effect on intestinal recovery after surgery (Table 9).

Table 9: Effect of intraoperative drugs on Ileus

		Time to flatus	Time to bowel movement	Time to defecation	Time to first rescue analgesia	Time to oral feeding
		Mean	Mean	Mean	Mean	Mean
i. Fentanyl Dose	Up to 1 day	24	24	24	25	24
	More than 1 day	25	25	24	19	25
p-value		0.569	0.533	0.895	<0.0001	0.661
ii. Tramadol Dose	Up to 1 day	100	100	88	77	100
	More than 1 day	100	100	101	100	88
p-value		0.997	0.997	0.801	0.484	0.890
iii. Paracetamol Dose	Up to 1 day	14	14	12	19	12
	More than 1 day	7	7	14	1	1
ii. Tramadol Dose p-value		0.703	0.708	0.811	0.500	0.721
iv. Diclofenac Dose	Up to 1 day	137	137	100	131	136
	More than 1 day	100	100	141	100	100
p-value		0.689	0.655	0.397	0.751	0.781

Patients operated on by consultants significantly ($P < 0.001$) tolerated first oral feeds earlier compared to patients operated on by registrars; but the latter cadre achieved faster onset to first defecation ($P = 0.016$) compared to consultants. Achievement of shorter time to oral feeds in patients operated on by consultants could have been due to strict follow-up of postoperative instructions.

Table 10:

Outcome	Service Provider	Mean	Std. Deviation	p-value
Time to first flatus (hours)	Consultant	7.61	7.708	24
	Registrar Part II	7.36	6.058	25
	Registrar Part I	5.20	2.775	0.661
Time to normal bowel sounds (hours)	Consultant	10.69	8.757	100
	Registrar Part II	9.69	6.528	88
	Registrar Part I	7.80	5.020	0.890
Time to first defecation (hours)	Consultant	33.50	9.429	12
	Registrar Part II	28.31	8.301	1
	Registrar Part I	31.40	9.762	0.721

Time to first rescue analgesia (hours)	Consultant	12.47	12.409	136
	Registrar Part II	7.00	3.490	100
	Registrar Part I	16.00	.	0.781
Time to tolerance of first oral feed (hours)	Consultant	12.78	5.321	
	Registrar part II	17.06	5.477	
	Registrar part I	19.40	2.510	

Discussion

Prevalence of ileus after 3 days post caesarian delivery (5.6%)

As in the study by Tevis et al, on abdominal surgery patients in which the prevalence of postoperative ileus was 5.3 to 24% (37), the prevalence of ileus in this study was 5.6%. Time to first defecation was considered as the clinical hall mark that best reflected recovery of gut after surgery which had been validated by Vanbree et al in a multicenter trial of segmental colectomy patients (39).

Predictive Factors associated with Postoperative Ileus after Caesarean Delivery.

The age and BMI of patients did not influence recovery of gut after surgery. A randomized controlled trial by Kathrine, H et al on post-operative intestinal recovery in cholecystectomy patients (13), yielded the same result.

Preoperative intravenous fluids (normal saline 501 – 1000 mls) significantly influenced duration of time to first flatus, time to normal bowel sounds and time to first oral feeds. This suggests that preoperative hydration status of the patient influences intestinal recovery postoperatively.

In this study, restrictive intravenous crystalloids had no significant effect on postoperative intestinal function. This is dissimilar to the study by Nisanevich, V et al on laparotomy patients (colectomy, gastrectomy and cholecystectomy) which revealed that intraoperative restrictive fluid regime significantly shortened time to first flatus and time to defecation compared to liberal administration of fluids (29). Similar results on intraoperative fluid restriction with improved gastrointestinal recovery in comparison with liberal fluid administration were demonstrated by Varadhan et al using systematic meta-analysis on 801 patients who underwent open abdominal surgery (40).

The research methodology utilized here varied from other studies because all patients received fluid volumes of the restrictive regimen. Fluid administration was not goal guided by vital signs (central venous pressure) or invasive monitoring (stroke volume oesophageal Doppler monitoring) and patients were not randomized in comparison to studies by Pearse et al and Rahbari et al which included goal directed therapy and the use of different fluid regimes with patient randomization (31,33). Restrictive fluid regimes (especially with colloid) were demonstrated to be superior to liberal fluid regime in

enhancing gut recovery after abdominal surgeries (colon resection, cholecystectomy, hepatectomy and gastrectomy).

Level of surgical competence was shown to influence recovery of intestinal function. Patients operated by consultants had significantly reduced time to tolerance of first oral feeds compared to patients operated by registrars. Since time to first flatus, time to normal bowel sounds and time to defecation was not significantly different in all surgical cadres, the consultants' instructions to feeding of patients could have influenced the time to first oral feed (considering they operated most of the patients).

Statistically Significant Association between Perioperative Intravenous Fluids and Recovery of Intestinal Function.

Pearson correlation test carried out to find any association between perioperative intravenous fluids and recovery of gut function after caesarean delivery showed statistically significant association between shortened time to tolerance of first oral feeds and administration of normal saline preoperatively ($P=0.045$) with a significantly lengthened time to first rescue analgesia in the first postoperative day ($P=0.048$) when 500 – 1000 mls was administered. Analysis of variance also revealed association between preoperative crystalloids administration (500-1000 mls) and shortened time to normal bowel sounds ($P=0.029$) and time to first flatus (0.011). This was most likely due to improved hydration status preoperatively.

In conclusion, the prevalence of postoperative ileus in obstetric population is like that seen in abdominal surgery patients. There is a significant association between preoperative intravenous fluid administration and recovery of intestinal function postoperatively. 500-1000mls of normal saline preoperatively leads to enhanced recovery of intestinal function compared to other fluid volumes. The type of surgical practice has a positive influence on recovery of intestinal function postoperatively. Most consultant obstetricians in KNH repair the uterus without exteriorization. This may have contributed to enhanced gut recovery post-operatively.

References

1. Artinyan, A. Nunoo-Mensah, J. W., Balasubramaniam, S., Gauderman, J., Essani, R., Gonzalez-Ruiz, C. Beart, R. W. Prolonged postoperative ileus - Definition, risk factors, and predictors after surgery. *World Journal of Surgery*, 2008;32(7), 1495-1500.
2. Ali, S. Z., Taguchi, A., Holtmann, B. and Kurz, A. Effect of supplemental pre-operative fluid on postoperative nausea and vomiting Anaesthesia, *Journal of the Association of Anaesthetists of Great Britain and Ireland*, 2003;58: 780-784.
3. Behm B, Stollman N. Postoperative Ileus: Etiologies and Interventions. *Clinical Gastroenterology and Hepatology Journal*, 2003;1: 71-80.
4. Venara, A., Neunlist, M., Slim, K., Barbieux, J., Colas, P. A., Hamy, A., & Meurette, G. Postoperative ileus: Pathophysiology, incidence, and prevention. *Journal of Visceral Surgery*, 2016;153(6), 439-446.

5. Bragg, D., El-Sharkawy, A. M., Psaltis, E., Maxwell-Armstrong, C. A., & Lobo, D. N. Postoperative ileus: Recent developments in pathophysiology and management. National Institute for Health Research Biomedical Research Unit, Clinical Nutrition, 2015;34(3), 367-376.
6. Bundgaard-Nielsen, M., Secher, N. H., & Kehlet, H. "Liberal" vs. "restrictive" perioperative fluid therapy a critical assessment of the evidence. Acta Anaesthesiologica Scandinavica, 2009;53(7), 843-51.
7. C. Challand, R. Struthers, J. R. Sneyd, P. D. Erasmus, N. Mellor, K. B. Hosie, G. Minto. Randomized controlled trial of intraoperative goal-directed fluid therapy in aerobically fit and unfit patients having major colorectal surgery. British Journal of Anaesthesia 2012;108 (1): 53-62.
8. Chang J. Physiologic Adaptations to Pregnancy. Maternal-Fetal Medicine Neurol Clinic Journal NA, 2012;30(3):781-9.
9. Corcoran, T., Emma, J., Rhodes, J., Hons, M., Clarke, S., Hons, M., & Myles, P. S. Perioperative Fluid Management Strategies in Major Surgery: A Stratified Meta-Analysis, International Anesthesia Research Society, 2012;114(3), 640-651.
10. Dahlke JD, Mendez-figueroa H, Maggio L, Alisse K, Sperling J, Chauhan SP. Prevention and management of postpartum hemorrhage: A comparison of 4 national guidelines. American Journal of Obstetrics and Gynaecology, 2015;2:23-24.
11. Gan, T. J., Soppitt, A., Sc, B., & Maroof, M. Goal-directed Intraoperative Fluid Administration Reduces Length of Hospital Stay after Major Surgery, American Society of Anesthesiologists 2017;97 (4): 820-6 (4).
12. Hansen, B., Møller, D. R., Brandstrup, B., Svendsen, P. E., Rasmussen, M., Belhage, B. ... Simonsen, L. Which goal for fluid therapy during colorectal surgery is followed by the best outcome: near-maximal stroke volume or zero fluid balance , British Journal of Anaesthesia, 2012;109:191-199.
13. Holte, K., Klarskov, B., Christensen, D. S., Lund, C., Nielsen, K. G., Bie, P., & Kehlet, H. Liberal versus restrictive fluid administration to improve recovery after laparoscopic cholecystectomy: a randomized, double-blind study. Annals of Surgery, 2004;240(5), 892-9.
14. Holte, K., Foss, N. B., Andersen, J., Valentiner, L., Lund, C., Bie, P., & Kehlet, H. Liberal or restrictive fluid administration in fast-track colonic surgery : a randomized, double-blind study †, Annals of Surgery, 2007;99(4), 500-508.
15. Holte, K. Pathophysiology and clinical implications of perioperative fluid management in elective surgery. Danish Medical Bulletin, 2012;57(7), B4156.
16. Jacob M, Chappell D. Best Practice & Research Clinical Anaesthesiology Effects of perioperative fasting on haemodynamics and intravascular volumes. Best Pract Res Clin Anaesthesiol 2012;26(4):5421-30.
17. Jernigan, A. M., Chen, C. C. G., & Sewell, C. A randomized trial of chewing gum to prevent postoperative ileus after laparotomy for benign gynecologic surgery. International Journal of Gynecology and Obstetrics, 2014;127(3), 279-282.
18. Kehlet H. Effect of postoperative analgesia on surgical outcome. British Journal of Anaesthesiology 2001;87 (1):62-72.
19. Kranke, P., Jokinen, J., Ni, P., Schnabel, A., Mw, H., Hahnenkamp, K., ... Hahnenkamp, K. Continuous intravenous perioperative lidocaine infusion for postoperative pain and recovery (Review) Continuous intravenous perioperative lidocaine infusion for postoperative pain and recovery, The Cochrane Library, 2015;2 (7).
20. Kumaravadivel TD, Azian N, Z A. Survey on Maternal Satisfaction in Receiving Spinal Anaesthesia for Caesarean Section. Malaysian Journal of Medical Sciences, 2011;51-4.
21. Lee, C. T., Chang, S. S., Kamat, A. M., Amiel, G., Beard, T. L., Fergany, A ... Koch, M. Alvimopan accelerates gastrointestinal recovery after radical cystectomy: A multicenter randomized placebo-controlled trial. European Urology, 2014;66(2), 265-272.
22. Levy DM. Emergency Caesarean section: best practice. Anaesthesia 'update' lectures at the Winter Scientific Meeting of the Association of Anaesthetists of Great Britain and Ireland, 2006;61(8) 786-91.
23. Lobo, D. N., Bostock, K. A., Neal, K. R., Perkins, A. C., Rowlands, B. J., & Allison, S. P. Effect of salt and water balance on recovery of gastrointestinal function after elective colonic resection : a randomised controlled trial, The Lancet, 2002;359:1812-1818.
24. Livingston, E., & Passaro, E. J. Postoperative ileus. Digestive Diseases and Sciences, 1990;35(1), 121-32.
25. Luckey, A., Livingston, E., & Taché, Y. Mechanisms and treatment of postoperative ileus. Archives of Surgery, 2003;138(2), 206-214.
26. Mackay, G., Fearon, K., Mcconnachie, A., Serpell, M. G., Molloy, R. G., & Dwyer, P. J. O. Randomized clinical trial of the effect of postoperative intravenous fluid restriction on recovery after elective colorectal surgery, British Journal of Surgery, 2006;93: 1469-1474.
27. Moharari, R. S., Motalebi, M., Najafi, A., Zamani, M. M., Imani, F., Etezadi, F., ... Khajavi, M. R. Magnesium can decrease postoperative physiological ileus and postoperative pain in major non laparoscopic gastrointestinal surgeries: a randomized controlled trial. Anesthesiology and Pain Medicine, 2013;4(1), 1-7.
28. Müller, S. A., Rahbari, N. N., Schneider, F., Warschkow, R., Simon, T., Von Frankenberg, M. ... Büchler, M. W. Randomized clinical trial on the effect of coffee on postoperative ileus following elective colectomy. British Journal of Surgery, 2012;99(11), 1530-1538.
29. Nisanevich, V., Felsenstein, I., Almog, G., Weissman, C., Einav, S., & Matot, I. Effect of Intraoperative Fluid Management on Outcome after Intraabdominal Surgery, American Society of Anesthesiologists, 2017;103 (1), 25-32.
30. Patterson LS, Connell CMO, Basket TF Maternal and Perinatal Morbidity Associated With Classic and Inverted T Cesarean Incisions. ; The American College of Obstetricians and Gynecologists, 2002;100(4):633-7.
31. Pearse, R., Dawson, D., Fawcett, J., Rhodes, A., & Grounds, R. M. Early goal-directed therapy after major surgery reduces complications and duration of hospital stay. A randomised controlled trial, St George's Hospital Critical care Journal, 2005;9(6).
32. Penn Z. Indications for caesarean section. Best Practice and Research Clinical Obstetrics and Gynaecology, 2001;15(1):1-15.
33. Rahbari, N. N., Zimmermann, J. B., Schmidt, T., Koch, M., Weigand, M. A., & Weitz, J. Meta-analysis of standard, restrictive and supplemental fluid administration in colorectal surgery, British Journal of Surgery, 2009;96: 331-341.
34. Seta, M., & Kale-Pradhan, P. (2001). Efficacy of metoclopramide in postoperative ileus after exploratory laparotomy. Department of Pharmacy Practice, Wayne State University Pharmacotherapy, 2001;21(10), 1181-6.

35. Silva, R. M. Clinical Benefits After the Implementation of a Protocol of Restricted Perioperative Intravenous Crystalloid Fluids in Major Abdominal Operations, *World Journal of Surgery* 2009;33:925-930.
36. Teoh WHL, Consultant S, Westphal M, Medical C, Kampmeier TG. Best Practice & Research Clinical Anaesthesiology Update on volume therapy in obstetrics. *Best Practice & Research Clinical Anaesthesiology*, 2014;28(3):297-303.
37. Tevis, S. E., Carchman, E. H., Foley, E. F., Harms, B. A., Heise, C. P., & Kennedy, G. D. Postoperative Ileus???More than Just Prolonged Length of Stay? *Journal of Gastrointestinal Surgery*, 2015;19(9), 1684-1690.
38. Udo A, Ekott M. Delivery techniques at elective caesarean section for the singleton term transverse-lying fetus in a Nigerian teaching hospital. *Transnational Journal of Science and Technology* 2013;3(5):32-41.
39. Van Bree, S. H. W., Bemelman, W. A., Hollmann, M. W., Zwinderman, A. H., Matteoli, G., El Temna, Boeckstaens s. G. E. E. Identification of clinical outcome measures for recovery of gastrointestinal motility in postoperative ileus. *Annals of Surgery*, 2014;259(4), 708-14.
40. Varadhan, K. K., & Lobo, D. N. Conference on "Malnutrition matters" Symposium 3 : Death by drowning A meta-analysis of randomised controlled trials of intravenous fluid therapy in major elective open abdominal surgery : getting the balance right, *Proceedings of the Nutrition Society*, 2009;488-498.
41. Boelens, P. G., Heesakkers, F. F. B. M., Luyer, M. D. P., van Barneveld, K. W. Y., de Hingh, I. H. J. T., Nieuwenhuijzen, G. A. P., ... Rutten, H. J. T. Reduction of postoperative ileus by early enteral nutrition in patients undergoing major rectal surgery: prospective, randomized, controlled trial. *Annals of Surgery*, 2014;259(4), 649-655.
42. Vather, R., Trivedi, S., & Bissett, I. Defining Postoperative Ileus: Results of a Systematic Review and Global Survey. *Journal of Gastrointestinal Surgery*, 2013; 17(5), 962-972.
43. Walker JJ. Pre-eclampsia. *Best Practice & Research Clinical Anaesthesiology* 2000; 356:1260-5.
44. Walsh, R. M. Current therapies to shorten postoperative ileus, *Cleveland Clinic Journal of Medicine*, v, 2009; 641-648.
45. Wu, Z., Boersema, G. S. A., & Lange, J. F. Nicotine gum chewing: A novel strategy to shorten duration of postoperative ileus via vagus nerve activation. *Erasmus University Medical Center journal of surgery, Medical Hypotheses*, 2014; 83(3), 352-354.
46. Zhang, J., Qiao, H., He, Z., Wang, Y., Che, X., & Liang, W. Intraoperative fluid management in open gastro-intestinal surgery: goal-directed versus restrictive, *Fudan University Anesthesiology Clinics Journal*, 2012; 67(10), 1149-1155.

TENTATIVE KENYA SOCIETY OF ANAESTHESIOLOGISTS 2019 CME CALENDAR

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14 Mar	Sepsis	Dr. David Odaba	Sandoz	Crowne Plaza
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04 May	Medicolegal Symposium	Multiple	Several	Swiss Lenana Mount Hotel
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A Case Report and Review Of Literature.

Cyanide Poisoning Following Ingestion Of Apple Seeds

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Introduction

Cyanogenic glycosides are found in many plants in nature. These plants include bitter almond, peach, cherry, apricot and apple¹. Such plants have high concentrations of the cyanogenic glycoside amygdalin in their seeds. Intoxication could result from contamination of food with these seeds during processing or intentional ingestion². Of the two scenarios, intentional ingestion occurs less frequently¹. The intentional ingestion of apple seeds for self-harm, especially in the African region is unreported. We report the case of a nineteen-year-old male patient who ingested a blended mixture of approximately 150 apple seeds.

Case Report

The patient, a 19-year-old male was admitted for management of reduced consciousness and seizures. The history revealed that he had blended seeds from 30 apples apparent self-harm having learnt from the internet about the cyanide content in apple seeds. The patient consequently lost consciousness at home and had 3 episodes of generalized tonic-clonic convulsions each lasting 2 minutes. Subsequently, he remained unconscious. At the time of presentation to hospital and at initial assessment, his comma scale was 8/15 (opening eyes to pain, incomprehensible and flexing to pain), pupils were 2mm equal and reacting to light, the neck was supple and Kernig's sign was negative. His blood pressure was 90/58, pulse was 128, respiration rate was 28 and pulse oximetry was 100% off oxygen. The rest of his neurological and systemic examination was unremarkable. Neuroimaging was also unremarkable. Initial laboratory workup showed metabolic acidosis (pH 7.215, HCO₃ 12.8, PO₂ 115, PCO₂ 18 and lactic acid 16.8 mmol/L). The ECG showed sinus tachycardia. Blood chemistry was normal (BUN 2.77mmol/L, serum creatinine 54.4Qmol/L, K 3.81mmol/L, Na 141.5mmol/L, Phosphorous 0.85 mmol/L, Ca 2.05mmol/L). Blood counts and liver function tests were also within reference ranges. Blood samples were taken for toxicological analysis of cyanide levels. These levels were 0.45 mg/L, albeit reported 5 days after the patient had been admitted.

Initially, the patient was managed with anticonvulsant medication (phenytoin 750 mg IV loading dose and 250 mg IV BID). On clinical suspicion of cyanide intoxication, 5g of hydroxocobalamin was administered intravenously. The arterial blood gas 12 hours following administration of hydroxocobalamin had significant improvement of acidosis (pH 7.35, PCO₂ 23, PO₂ 139, Lactate 2.3 mmol/L). Thereafter,

the patient did not have any further seizures and his consciousness improved to a coma scale of between 12/15 and 14/15. However, the patient became febrile on the third day and had a seizure lasting 1 minute with a subsequent reduction in consciousness. Sodium valproate 500 mg IV BID was added to the anticonvulsant regimen. Cerebrospinal fluid analysis was positive for Human Herpesvirus 6 infection wherewith Acyclovir 750 mg IV TID was added to the treatment. Subsequently, the patient had steady clinical improvement and was discharged home after 16 days of hospitalization having been weaned off anticonvulsant medication. He was stable when reviewed after 3 months and 6 months and had no residual neurological symptoms.

Discussion

Cyanide has been used as a poison since antiquity even prior to its characterization and identification¹. Cyanide poisoning commonly occurs in burn victims as a result of the combustion of synthetic polymers such as nylon which elaborated hydrogen cyanide³. Iatrogenic exposure to cyanide may result from the administration of nitroprusside which can release cyanide moieties that can accumulate to toxic levels⁴. Cyanide can also be ingested in its naturally occurring form found in plants. In this form, cyanide exists as a glucoside, amygdalin. It is found in low concentrations in the seeds, fruit pits and bark of more than 1000 plant species including apples^{1,2}. Since it exists in low concentrations where it occurs in these fruits, it rarely causes poisoning. When ingested, the sugar moieties in amygdalin are broken down by digestive enzymes in the gut^{1,6,7}. The remaining compound, containing hydrogen cyanide, can accumulate to toxic concentrations. When absorbed detoxification occurs in the liver via the enzyme rhodanase which catalyzes the conversion of cyanide to thiocyanate⁶. Further, 3- mercaptopyruvate sulfurtransferase, thiosulfate reductase (in the liver), and cystathionase can also convert cyanide to thiocyanate⁷. In this regard, approximately 0.017 mg of cyanide/ kilogram/minute can be detoxified¹. This rate is however insufficient to detoxify the high concentrations of cyanide that result from poisoning. Such poisoning has been reported in Kenya mainly following ingestion of cassava⁵.

Amygdalin in apple seeds is found in concentrations of up to 3mg/g⁸. Eating the seeds whole rarely causes poisoning but ingesting large amounts of seeds or blending them into "shakes" can result in the release of toxic levels of cyanide¹⁸. The patient in our case who blended apple seeds no doubt ingested a high concentration of amygdalin. Once absorbed,

cyanide is rapidly distributed ubiquitously in the circulation to all organs. Its toxicity majorly results from binding the ferric iron in cytochrome oxidase a3 resulting in uncoupling of oxidative phosphorylation⁴. Consequently, multi-organ system effects are experienced. Anaerobic respiration that results from cyanide toxicity leads to accumulation of lactic acid causing a high anion-gap acidosis. In fact, lactate levels have been proposed as a measure of severity of cyanide poisoning. Since tissue utilization of oxygen is blocked, the oxygen concentration in the venous circulation rises resulting in a narrowing of the arterio-venous oxygen gradient to <10mmHg^{4,7}. Cyanide can also bind the ferric ion in haemoglobin consequently embarrassing the oxygen carrying capacity of blood².

Clinically, inhibited oxygen extraction causes multi-systemic symptoms. These symptoms are apparent within a minute of intoxication. In the nervous system, acute manifestations include increased respiratory drive causing hyperpnoea and hyperventilation due to stimulation of peripheral chemoreceptors⁷. This is a transient response that may be followed by hypoventilation and apnea^{4,7}. Since anaerobic metabolism predominates, a disruption in the brains calcium homeostasis alters neurotransmission. Tremors, convulsions, coma and death can result. Moreover, haemodynamic compromise resulting from vasodilation, arrhythmias and reduction in the heart's inotropic capacity can occur. This may end in heart block and cardiac arrest⁷.

Serum concentrations of cyanide greater than 0.5 mg/L are typically associated with acute cyanide poisoning¹. However, since cyanide tests aren't readily available and often require several days for turn around as happened in the current case, their use in the acute treatment of cyanide poisoning is impractical⁴. Their use is used only to confirm exposure and management of the patient should be guided by clinical suspicion.

Acute management of cyanide exposure involves decontamination⁶. In cases of ingestion such as this case report, gastrointestinal decontamination measures may not be useful if the time lag from exposure to presentation is prolonged since cyanide has a high potency and rapid onset of toxicity. Should presentation happen sooner, it is reasonable to consider performing an orogastric lavage and administering activated charcoal which binds to cyanide as one gram for 35 grams of cyanide^{4,6}. Administration of oxygen and addressing acidosis, seizures and hemodynamic compromise additionally is useful. Definitively, antidotal remediation of poisoning is warranted. For this purpose, several therapies are available. Hydroxocobalamin 5g over 15 minutes can be used. The detoxifying capacity of hydroxocobalamin results from its ability to bind cyanide to form non-toxic vitamin B12 (cyanocobalamin) which is easily excreted through the kidney.

An antidote kit containing amyl nitrite, sodium nitrite, and sodium thiosulfate can also be administered as an alternative remedy^{4,9}. Their administration induces production of methemoglobin whose ferric iron preferentially binds cyanide reducing the amount of cyanide available to bind to cytochrome a3. Amyl nitrite, which is inhaled, generates about 5% methemoglobin while sodium nitrite administered intravenously increases methemoglobin by about 8-20%^{9,10}.

In conclusion, this case highlights the need for a high index of suspicion as well as vigilance in the clinical diagnosis of suspected intoxication. The nature of the poisoning agent used highlights the increasing danger that availability of such knowledge on the internet poses for patients who may be intent on self-harm. Prompt diagnosis and administration of antidotal treatment are key in assuaging toxicity of ingested cyanide.

References

1. Agency for Toxic Substances and Disease Registry. Cyanide Toxicity. 1991;(15):1-29.
2. Akyildiz BN, Kurto lu S, Kondolot M, Tunç A. Cyanide poisoning caused by ingestion of apricot seeds. *Ann Trop Paediatr*. 2010;30(1):39-43. doi:10.1179/146532810x12637745451951.
3. Barillo DJ. Diagnosis and treatment of cyanide toxicity. *J Burn Care Res*. 2009;30(1):148-152. doi:10.1097/BCR.0b013e3181923b91.
4. Hamel J. A review of acute cyanide poisoning with a treatment update. *Crit Care Nurse*. 2011;31(1):72-82. doi:10.4037/ccn2011799.
5. Maitai CK MN. Human Poisoning with Plants In Kenya. *East Cent African J Pharm Stud*. 2006;8(1):10-13.
6. Gracia R, Shepherd G. Cyanide poisoning and its treatment. *Pharmacotherapy*. 2004;24(10 II):1358-1365. doi:10.1592/phco.24.14.1358.43149.
7. Beasley DMG, Glass WI. Cyanide poisoning: Pathophysiology and treatment recommendations. *Occup Med (Chic Ill)*. 1998;48(7):427-431. doi:10.1093/occmed/48.7.427.
8. Bolarinwa IF, Orfila C, Morgan MRA. Determination of amygdalin in apple seeds, fresh apples and processed apple juices. *Food Chem*. 2015;170:437-442. doi:10.1016/j.foodchem.2014.08.083.
9. Mégarbane B, Delahaye A, Goldgran-Tolédano D, Baud FJ. Antidotal treatment of cyanide poisoning. *J Chin Med Assoc*. 2003;66(4):193-203. <http://www.ncbi.nlm.nih.gov/pubmed/12854870>.
10. Cummings TF. The treatment of cyanide poisoning. *Occup Med (Chic Ill)*. 2004;54(2):82-85. doi:10.1093/occmed/kqh020.



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- ➔ Cardiac Anaesthesia
- ➔ Neuro Anaesthesia
- ➔ Emergency/Trauma Anaesthesia
- ➔ Sedation and Monitored Anaesthesia Care
- ➔ Research
- ➔ Pain Management

**CONFERENCE
 REGISTRATION
 DETAILS**

There will be **3 Full Day Workshops** (limited slots) at **Kshs. 4,000** that will run in parallel; ➔

1. Focus Assessed Transthoracic Echocardiography (FATE) Course
2. Mechanical Ventilation
3. Managing Emergencies in Paediatric Anaesthesia (MEPA) Course

Designation	Delegates Fee Local/ International On or Before 31st March Early Registration	Delegates Fee Local/ International From 1st April Normal Registration	Full Day (Parallel) Workshops - FATE Course - Mechanical Ventilation - MEPA Course
Physicians/ Consultants	KSH 15,000/USD 170	KSH 20,000/USD 220	KES 4000/USD 50
NPAs	KSH 10,000/USD 120		
GATs	KSH 7,000/USD 90		

KEY: NPAs - Non- Physician Anaesthetists **GAT** - Graduate Anaesthetists in Training

For more information, contact:

KMA Centre, 4th Flr, Suite 406, Wing C,
 Mara Road, Upper Hill
 Tel: +254 716 303 868, +254 733 747 299
 E-mail: admin@anaesthesiakenya.co.ke
 Website: www.anaesthesiakenya.co.ke

For reservations, contact:

Lake Naivasha Resort (conference venue)
 Samson Kariuki (0725 211 985)
 Email: reservations@lakenaivasharesort.co.ke
 (Please inform the Hotel that you are a KSA/CCSK 2019 delegate, kindly ensure that you have registered for the conference to enjoy discounted rates.)

Gala Dinner open to all conference delegates;
accompanying persons to pay a supplement of KES 2,000.

Cezol®-Cefazolin

*Narrow Angle,
Targeted Precision
Prophylaxis.*

New Frontier In Preoperative Care

CEZOL® - Cefazolin

Parenteral first-generation cephalosporin. Compared to other first-generation cephalosporins cefazolin has greater gram-positive coverage, requires less frequent dosing, and achieves higher blood levels after IM/IV administration.

Dosage Guidelines: - Adults

2 g IM or IV as a single dose within 30 to 60 minutes prior to the surgical incision.

Operations longer than 2 hours, additional doses of 0.5 to 1 g IM or IV may be given during the procedure and postoperatively every 6 to 8 hours for 24 hours.

High risk procedures - (e.g. open-heart surgery, prosthetic arthroplasty), prophylaxis should be continued for up to 3 to 5 days. **Clinical practice guidelines** suggest 2 g IV, and for patients weighing 120 kg or more, 3 g IV. For gynecologic procedures, the **American College of Obstetricians and Gynecologists (ACOG)** recommends 1 g IV, and for women with a body mass index (BMI) more than 35 or weight more than 100 kg, 2 g IV. Intraoperative re-dosing 4 hours from the first preoperative dose and duration of prophylaxis less than 24 hours for most procedures is suggested by **clinical practice guidelines**.

Cefazolin is FDA-approved for contaminated or potentially contaminated procedures, including vaginal hysterectomy and cholecystectomy in high-risk patients, as well as in surgical patients in whom infection at the operative site would present a serious risk (e.g. open-heart surgery, prosthetic arthroplasty). **Clinical practice guidelines** recommend cefazolin monotherapy for cardiothoracic, gastrointestinal, biliary tract, hernia repair, clean head and neck with prosthesis, neurosurgical, uro-gynecology, orthopedic, vascular, certain transplantation, and plastic surgery procedures.

Cefazolin is recommended as part of combination therapy for appendectomy, obstructed GI, colorectal, clean-contaminated head and neck, and urologic with prosthesis or clean-contaminated procedures.

Pediatric Dosing:

30 mg/kg IM or IV as a single dose (Max: 2 g/dose) within 60 minutes prior to the surgical incision. Repeat dose intraoperatively 4 hours after preoperative dose if surgery still in progress. Depending on the procedure, 30 mg/kg/dose (Max: 2 g/dose) IV may be given postoperatively every 8 hours for 24 hours.



Manufactured By: //



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