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Obstetrics fistulae: A review and risk factors

- Diabetic Ketoacidosis in Adults: Pathogenesis, diagnosis and management
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FRONT COVER: Dr Koma Akim performing an obstetric fistula repair at Juba Teaching Hospital during a fistula camp and training in 2021. (Photo credit: Koma Akim)

Obstetric fistulae in South Sudan: a paradigm shift in repairs is required

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Obstetric fistula (OF) is an abnormal communication between the vagina and the bladder/urethra and/or the rectum, which results in the passage of urine and/or faeces through the vagina. This occurs because during obstructed labour, the bladder, the vagina, and the rectum are compressed between the baby's head and the pelvis leading to ischaemic necrosis of the areas involved thus leading to either vesicovaginal fistula (VVF) or rectovaginal fistula (RVF) or both.[1] It is the severest complication of prolonged obstructed labour.[2] The World Health Organisation estimates that approximately two million women suffer from OF. About 200-500 cases of OF occur per 100,000 deliveries, with an overall estimate of 0.8 per 1,000 birth.[3] The OF case fatality rate in South Sudan is 3.5% and it is high in at least three States mainly Lakes States, Unity State, and Jonglei State.[4]

Obstetric fistulae repairs in South Sudan

The Ministry of Health estimates that there are about 60,000 women living with OF in South Sudan and only about 1,000 of them have had surgical repairs and treatment. Almost all of these surgical repairs of OF were performed during OF camps in various States. These OF camps are sporadic and cannot keep pace with the huge backlog of cases and the ever-increasing numbers of new patients. The OF repairs were mainly done by expatriate fistula surgeons and none by South Sudanese surgeons. The overall cost of one fistula management is about $1,500 [5] which is expensive and an unsustainable.

> "About 200-500 cases of Obstetrics Fistula occur per 100,000 deliveries, with an overall estimate of 0.8 per 1,000 birth."

Paradigm shift in the treatment of obstetric fistulae

Obstetric fistulae repairs and treatment need to be done routinely in ALL Hospitals in South Sudan. It is essential to train LOCAL specialist physicians to treat women with OF. Concurrently, there should be a DELIBERATE effort in building and equipping Operating Rooms in the hospitals dedicated to OF management. Additionally, incentives need to be put in place to motivate the OF teams. Finally, a centre of excellence especially dedicated to the management of patients with OF needs to be built in South Sudan.

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Audit of in-hospital mortality by age and time-of-day among patients presenting to a low-resource Ugandan hospital

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ABSTRACT

Introduction: Admission to hospital outside of normal working hours is consistently associated with poorer patient outcomes. Our aim was to determine the association of patients' age and time of presentation to a low-resource Ugandan hospital with admission rate and in-hospital mortality.

Method: Prospective observational non-interventional audit in the emergency and outpatient departments of Kitovu Hospital in Uganda, a low-resource sub-Saharan hospital. Data on age, sex, time of admission was collected from all non-pregnant patients during 2020 and 2021, and outcomes analysed.

Results: Out of 17,133 patients who presented to the hospital 189 died in hospital (1.1% of all presentations and 7.9% of all admissions); 46 (24.3%) patients died within 24 hours of arrival (0.3% of all presentations and 1.9% of all admissions). Deaths within 24 hours of arrival in hospital were more likely in the very young and the old, and in those who presented at night and on the weekend.

Conclusion: As many in-hospital deaths occur shortly after arrival, resuscitation skills are needed even in low-resource settings for as much of the 24-hour day as possible.

Key words: Out-of-hours presentation, hospital mortality, hospitalization

INTRODUCTION

Providing 24-hour care seven days a week is especially challenging for low-resource hospitals in sub-Saharan Africa. Admission to hospital on weekends and/or outside of normal working hours is consistently associated with poorer patient outcomes.[1-4] This audit compared the patients admitted to a low-resource Ugandan hospital and their mortality according to their age, sex, and time of presentation

The objective of the study is to determine the association of patients' age and time of presentation to a low-resource Ugandan hospital with admission rate and patient mortality

METHOD

Study design and Setting

This prospective observational study, which is part of an ongoing audit process, was performed in the emergency and outpatient department of Kitovu Hospital, which has 248 beds (50 medical and 35 surgical) and is located near Masaka, Uganda, 140km from the capital city of Kampala. It is a private not-for-profit (PNFP) hospital, accredited by the Uganda Catholic Medical Bureau.

Most emergency medical care is provided by recently qualified doctors (i.e.,

within 3 years of graduation) assisted by clinical officers (i.e., non-physician clinicians). The emergency (ED) and outpatient departments (OPD), which care for all patients attending the hospital except those attending the obstetric department, are located beside each other, sharing a common entrance. Clinical staff move between them as needed. The ED is open 24 hours a day, and the OPD from 9 am to 5 pm. During the day the combined departments are staffed by at least two clinical officers and a doctor, and at night one doctor is first on-call and supported by two others who are second and third on-call.

Participants and study process

Participants were all non-pregnant patients who presented between 23rd November 2020 and 31st October 2021. During the day, a dedicated researcher entered patients' age, sex, date, and time of arrival into an Excel database (Version 2102, Microsoft Corp., Redmond, WA). At night, this information was recorded by the nurse on duty and entered in the database the following morning. The subsequent immediate disposition of each patient was also recorded (i.e., admitted, discharged, or died while in the emergency department), and hospital records were then reviewed to identify patients who died while in hospital.

Statistical methods and data analysis

The admission and mortality rates according to patient age, time of day, day of week, and month of presentation were graphed, and the ages and times with the lowest admission rates were identified by visual inspection. Two different denominators were used to assess mortality: for death occurring on the day of presentation (i.e., within 24-hours) all patient presentations to the hospital were used as the denominator, whereas for deaths after 24-hours the denominator was the number of patients admitted. Numeric variables were compared using Student's t-test and the unadjusted odds ratios of categorical variables with admission rate and mortality were compared using chi squared analysis with Yates' continuity correction; calculations were performed using Epi-Info version 6.0 (Centre for Disease Control and Prevention, USA). The p-value for statistical significance was 0.05. Adjustment of odds ratios was performed using Logistic software.[5]

Ethics

Ethics Ethical approval of the study was obtained from the Scientific Committee Kitovu Hospital. The study conforms to the principles outlined in the Declaration of Helsinki.

Limitations

This study was performed in a single centre and there was no follow-up of patients after hospital discharge. We did not record the number of patients who attended repeatedly or who had chronic conditions.

RESULTS

During the study period of 341 days 17,133 patients (50.2 per day) presented to the hospital's OPD or ED; their mean age was 38.0 (SD 23.4) years, 14% were younger than 10 years, 8% 10 to 19 years old, 35% 20 to 39 years, and 43% over 40 years of age). About 7,010 (40.9%) were male, and 2,400 patients (14%) were admitted to hospital.

One hundred and eighty-nine patients died (1.1% of all presentations and 7.9% of all admissions); 46 patients died within 24 hours of arrival (0.3% of all presentations and 1.9% of all admissions) and 143 patients died more than 24 hours after admission to hospital (0.8% of all presentations and 6.0% of all admissions).

Patients who were admitted were only slightly older than those not admitted [39.0 (SD 28.4) versus 37.8 (SD 22.4) years, p-value 0.02). Men were more likely to be admitted than women (odds ratio 1.75, 95% CI 1.60 – 1.91 p-value <0.0001), even though men who were admitted were significantly younger than women admitted [36.7 (SD 27.9) versus 41.7 (SD 28.8) years, p-value <0.0001). Of the 2,338 children below 10 years of age who presented to the hospital 563 (24.1%) were admitted, after 40 years of age the chance of admission increased from 10% to >30% for patients ≥ 90-year-olds (Figure 1).

Most patients (82.6%) presented between 9 am and 5 pm. The lowest chances of admission were observed in patients between 20 and 40 years of age (7.9%) (Figure 1), those presenting between 8 am and 12 noon (6.3%) (Figure 2), and those presenting from Monday to Thursday (11.3%). As there was a surge in admissions attributable to COVID-19 before February 2021 and between May and July, 2021 the lowest admission rates were observed from February to April 2021 and from August to October 2021. Patients admitted between these age groups and

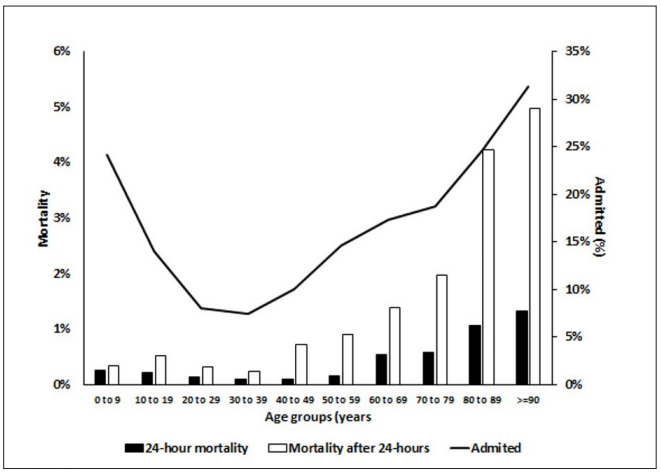

Figure 1. Mortality and admission rates by age at presentation

Table 1. Patients admitted, died within 24 hours of presentation, and died more than 24 hours after hospital admission

		Of 17,133 patients presenting n (%)	Admitted to hospital n (%)	Died in hospital within 24 hours n (%)
Sex	Female	10123 (59.1)	1133 (11.2)	20 (0.2)
	Male	7010 (40.9)	1267 (18.1)	26 (0.4)
Age	20-40 years	6287 (36.7)	495 (7.9)	8 (0.1)
	<20 or >40 years	10846 (63.3)	1905 (17.6)	38 (0.4)
Time	8:00 am to 12:00 noon	11331 (66.1)	718 (6.3)	11 (0.1)
	12:01 pm to 7:59 am	5802 (33.9)	1682 (29.0)	35 (0.6)
Day	Monday to Thursday	12763 (74.5)	1448 (11.3)	25 (0.2)
	Friday to Sunday	4370 (25.5)	952 (21.8)	21 (0.5)
Month	February to April or August to October	9414 (55.0)	1094 (11.6)	13 (0.1)
	November to February or May to July	7719 (45.0)	1306 (16.9)	33 (0.4)
		Of 2,400 patients admitted n (%)	**Died in hospital after 24 hours n (%)**	
Sex	Female	1133 (47.2)	90 (7.9)	
	Male	1267 (52.8)	53 (4.2)	
Age	20-40 years	495 (20.6)	19 (3.8)	
	<20 or >40 years	1905 (79.4)	124 (6.5)	
Time	8:00 am to 12:00 noon	718 (29.9)	37 (5.2)	
	12:01 pm to 7:59 am	1682 (70.1)	106 (6.3)	
Day	Monday to Thursday	1448 (60.3)	84 (5.8)	
	Friday to Sunday	952 (39.7)	59 (6.2)	
Month	February to April or August to October	1094 (45.6)	44 (4.0)	
	November to February or May to July	1306 (54.4)	99 (7.6)	

time periods also had a lower risk of death within 24 hours of presentation and, for those admitted, a lower chance of death more than 24 hours after hospital admission (Table 1).

After adjustment for age, time of presentation and sex, the chance of death within 24 hours increased six-fold for presentation between 8 am and 12 noon and nearly two-fold for presentation between Friday and Sunday. However, after adjustment, neither time of day nor day of the week were associated with mortality 24 hours after hospital admission. After adjustment men were not more likely to die within 24 hours, but women were after 24 hours (Table 2).

The deaths within 24 hours accounted for 24% of all in-hospital deaths, the rate of which subsequently fell exponentially with the last death recorded 31 days after admission. Although only 412 patients (2.4%) presented between midnight and 7am, 280 (68%) were admitted and these patients had the highest chance of dying (4.1%) within 24 hours of arrival; indeed, 17 of their 29 deaths (58.6%) occurred within 24 hours and accounted for 34.8% of all deaths within 24 hours (Figure 2).

Patients who died were older [56.6 (SD 26.5) versus 37.8 (SD 23.3) years, p-value (<0.0001)], regardless of whether they died within 24 hours of presentation [52.1 (SD 29.1) versus 38.0 (SD 23.3) years, p-value <0.0001], or more than 24 hours after admission [58.0 (SD 25.6) versus 37.8 (SD 28.2) years, p-value <0.0001]. There was no significant difference between the ages of men and women who died [52.7 (SD 26.7) versus 59.4 (SD 26.1), p-value 0.0900].

DISCUSSION

Main findings

Nearly a quarter of the patients who died in hospital did so within 24-hours of arrival to the hospital. These deaths were more likely in the very young and old, and in

Table 2. Crude (cOR) and adjusted odds ratio (aOR) of admission, death within 24 hours of presentation and death more than 24 hours after admission to hospital.

Hospital admission after presentation (N=17,133)					
		cOR	**(95% CI)**	**aOR**	**(95% CI)**
Sex	Female	(reference)			
	Male	1.75	(1.60 - 1.91)	1.61	(1.46 - 1.76)
Age	20-40 years	(reference)			
	<20 or >40 years	2.49	(2.24 - 2.77)	2.80	(2.51 - 3.13)
Time	8:00 am to 12:00 noon	(reference)			
	12:01 pm to 7:59 am	6.03	(5.48 - 6.65)	5.96	(5.41 - 6.57)
Day	Monday to Thursday	(reference)			
	Friday to Sunday	2.18	(1.98 - 2.39)	1.79	(1.62 - 1.98)
Month	February to April or August to October	(reference)			
	November to February or May to July	1.55	(1.42 - 1.69)	1.41	(1.28 - 1.55)
Death within 24 hours of presentation (N=17,133)					
		cOR	**(95% CI)**	**aOR**	**(95% CI)**
Sex	Female	(reference)			
	Male	1.88	(1.01 - 3.52)	1.63*	(0.91 - 2.93)
Age	20-40 years	(reference)			
	<20 or >40 years	2.76	(1.23 - 6.45)	2.73	(1.27 - 5.88)
Time	8:00 am to 12:00 noon	(reference)			
	12:01 pm to 7:59 am	6.25	(3.03 - 13.14)	5.39	(2.71 - 10.71)
Day	Monday to Thursday	(reference)			
	Friday to Sunday	2.46	(1.32 - 4.58)	1.85	(1.03 - 3.33)
Month	February to April or August to October	(reference)			
	November to February or May to July	3.10	(1.57 - 6.25)	2.70	(1.42 - 5.14)
Death after 24 hours in 2,400 patients admitted to hospital					
		cOR	**(95% CI)**	**aOR**	**(95% CI)**
Sex	Female	(reference)			
	Male	0.51	(0.35 - 0.73)	0.52	(0.37 - 0.74)
Age	20-40 years	(reference)			
	<20 or >40 years	1.74	(1.04 - 2.97)	1.69	(1.03 - 2.78)
Time	8:00 am to 12:00 noon	(reference)			
	12:01 pm to 7:59 am	1.24	(0.83 - 1.86)	1.22*	(0.82 - 1.79)
Day	Monday to Thursday	(reference)			
	Friday to Sunday	1.07	(0.75 - 1.54)	1.09*	(0.77 - 1.53)
Month	February to April or August to October	(reference)			
	November to February or May to July	1.96	(1.33 -2.88)	1.92	(1.33 - 2.77)

** = not statistically significant*

95% CI = 95% confidence interval

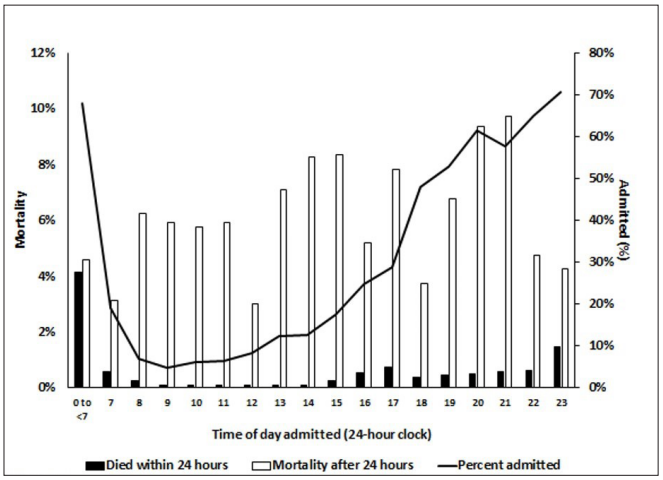

Figure 2. Mortality and admission rates by time of day of presentation.

those who presented to the hospital at night and on the weekend. However, after adjustment for age, sex and all times of presentation, in-hospital mortality 24 hours after admission to hospital was not associated with the time of day or day of the week of presentation but was associated with months of presentation, age, and female sex.

Interpretation

Unsurprisingly, very young and older patients were more likely to die, and patients arriving to the hospital were probably sicker during the two peaks of the COVID-19 pandemic before February and between May and July 2021. The precise number of patients who had COVID-19 during these periods can only be estimated, because the number of antigen tests available was limited. Others have reported that men are more likely to present to hospital out-of-hours than women [3] and that, after adjustment for age and sex, weekend admissions were associated with early but not late in-hospital deaths.[6] However, these increases in mortality for out-of-hours and weekend admissions are small and not the two to six-fold increases we observed.

Clinical significance

It is debateable if out-of-hours mortality we observed was higher because the patients were sicker or the quality of care provided at these times was lower.[1,4] Anyone can become acutely ill at any time, and the efficacy of most treatments for acute life-threatening illnesses are time dependent and must be delivered within minutes. Many deaths can be prevented if the causes of poor perfusion and oxygenation are identified and promptly corrected. Our results highlight the need for these resuscitation skills [7] to be available, even in a low resource setting, for as much of the 24-hour day as possible.

CONCLUSION

As many in-hospital deaths occur shortly after arrival, resuscitation skills are needed even in low-resource settings for as much of the 24-hour day as possible.

Funding and Conflict of interest statement

All costs were borne by the authors. John Kellett is a major shareholder of Tapa Healthcare DAC. The other authors have no potential conflicts of interest.

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Cochlear implantation and outcomes in a resource–limited setting: experience from Tanzania

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ABSTRACT

Introduction: Cochlear Implant is a small medical electronic device that is surgically inserted partially in the cochlear (inner ear) to restore some hearing in patients with severe to profound hearing loss. Cochlear implantation is considered a rehabilitative measure of choice that positively impacts on the quality of life of patients.

Objective: The objective was to describe the clinico-demographic characteristics of cochlear implantees and the outcomes of the intervention among the implantees at Muhimbili National Hospital (MNH) in Tanzania.

Method: This was a hospital based cross-sectional study which involved a total of 39 patients who underwent cochlear implantation from July 2017 to May 2021 at MNH. Clinico-demographic characteristics and outcomes of the intervention among the implantees were collected using structured questionnaires and data were analysed using Statistical Package for Social Sciences Version 20. Results were then presented in frequency tables and figures.

Results: This study recruited 39 patients with bilateral hearing loss with their ages ranging from 2 to 55 years. Their mean age was 4.7 years and median of 3 years. More than half, 24(61.5%) of implantees aged 2-3 years. Males predominated with male to female ratio of 1.2:1. Majority 37(94.9%) had pre lingual hearing loss and 36 (92.3 %) had bilateral profound sensorineural hearing loss. Ototoxicity was the commonest cause of hearing loss among the implantees contributing 16(41%) followed by birth asphyxia, 8(20.5%). A total of 37(94.9%) of these patients were implanted with a single cochlear device due to the high cost associated with this type of intervention.

Conclusion: Cochlear implantation in limited resource settings is possible and cost effective if there is enough support from the government and other charitable organisations. The availability of rehabilitative services remains key for better outcome after cochlear implantation.

Keywords: Cochlear implantation, resource limited, Tanzania.

INTRODUCTION

The World Health Organisation (WHO) estimated that about 466 million people in the world have hearing loss of greater than 35dB in the better hearing ear of which 432 million are adults and 34 million are children. Up to 80% of them live in low- and middle-income countries (LMICS).[1] There is also high prevalence of adult and childhood onset of hearing impairment in low-income regions, especially in sub-Saharan Africa and in South and Southeast Asia.[2] The causes of hearing loss can be congenital or acquired, however almost 60% of the causes are preventable in children.[1]

Hearing loss whether conductive or sensorineural requires treatment/rehabilitation since it has socio-economic impacts in the life of an individual. Hearing loss in

children results in a communication barrier and delayed language development which is later associated with poor academic performance hence reduction in employment opportunity later in life. On the other hand, adults with hearing loss have significant social and emotional stress leading to loneliness, isolation, frustration and early onset of dementia.[1, 3]

Introduction of multichannel cochlear implants in 1984, has changed lives of individuals with severe to profound hearing loss through restoring their hearing ability and therefore improved speech reception threshold. [4] Cochlear implantation is seen to be an expensive treatment option for patients in the LMICs compared to high income countries due to device related expenses, lack of rehabilitation services, and trained personnel.[5]

Tanzania is among one of the sub-Saharan countries referring patients abroad for cochlear implantation, especially those with prelingual onset hearing loss. Such referrals were done to those patients who did not benefit from hearing aid devices. This practice of referrals existed until June 2017 when the cochlear implantation program was established in the country. Initiation and support of the cochlear implantation program by the government of Tanzania has made this program cost effective as compared to patient being referred abroad. All services from hearing screening, candidacy evaluation, cochlear implantation, post implant care and rehabilitation are now available in the country at Muhimbili National Hospital.

This study describes the clinico-demographic characteristics of cochlear implantees and the outcomes of the intervention among the implantees at Muhimbili National Hospital (MNH) in Tanzania

METHOD

This was a hospital based descriptive cross-sectional study conducted at MNH, the main National referral hospital in Tanzania. Ethical clearance was obtained from the Research and Publications Committee of MNH. The department of otorhinolaryngology (ORL) receives patients from all the regional referral hospitals and is the only one performing cochlear implant surgeries in the country since July 2017.

A total of 39 patients who underwent cochlear implant surgery from July 2017 to May 2021 were included in this study. Data were collected using structured questionnaires. The information collected included: age, sex, age of onset and severity of hearing loss, causes of hearing loss and laterality of cochlear implantation. Pure tone audiometry (PTA), otoacoustic emissions (OAE), auditory brainstem response (ABR), computed tomography (CT) scan of the temporal bone and the magnetic resonance imaging for the evaluation of inner ear malformation and surgical planning were the hearing assessments and imaging modalities employed for evaluation and eligibility of candidacy selection for cochlear implantation. Data were analysed using the Statistical Package for Social Sciences (SPSS) version 20 for descriptive analysis and results were presented in frequency tables and figures.

RESULTS

This study involved 39 patients with bilateral hearing loss and their ages ranged from 2 to 55 years. Their mean age was 4.7 years and median of 3 years. More than half, 24(61.5%) of implantees were in the age group of 2-3 years. Males predominated with male to female ratio of 1.2:1. Majority of them 37(94.9%) had pre lingual hearing loss. (Table 1).

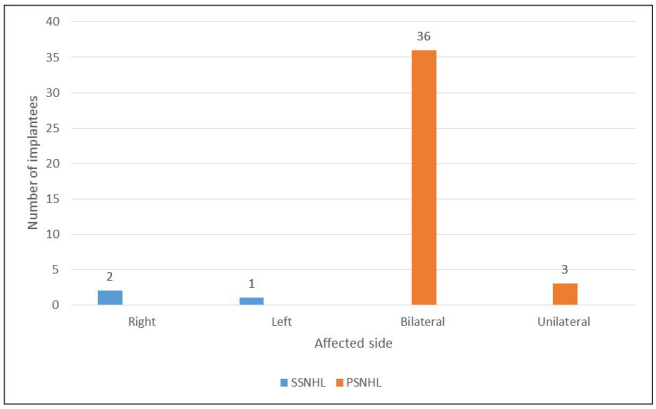
Characterization of severity of hearing loss of cochlear implantees by laterality.

The majority of cochlear implantees, 36(92.3%) had bilateral profound sensorineural hearing loss (PSNHL), (Figure 1).

Distribution of the causes of hearing loss among cochlear implantees.

Table 1. Clinico-demographic characteristics of cochlear implantees (N=39).

Variable		n (%)
Age group(years)	2-3	24 (61.5)
	4-5	13 (33.3)
	>5	2 (5.1)
Sex	Male	21 (53.8)
	Female	18 (46.2)
Age at onset of hearing loss	Pre lingual	37 (94.9)
	Post lingual	2(5.1)



KEY: SSNHL- Severe sensorineural hearing loss, PSNHL-Profound sensorineural hearing loss

Figure 1. Severity of hearing loss of the cochlear implantees by the affected side.

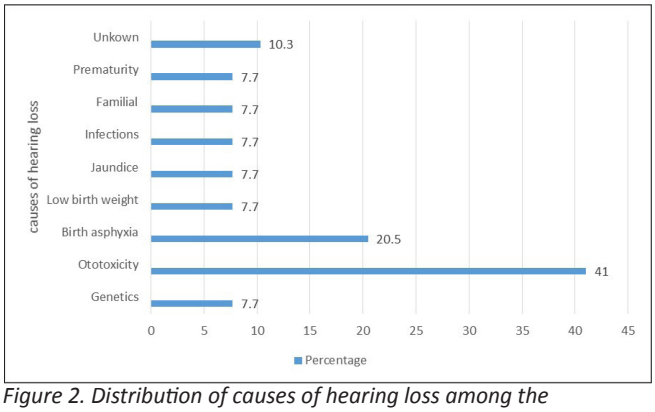


Figure 2. Distribution of causes of hearing loss among the implantees

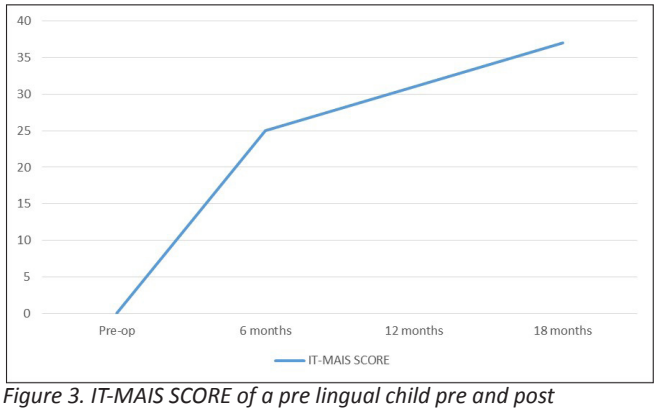


Figure 3. IT-MAIS SCORE of a pre lingual child pre and post implantation

Ototoxicity was the commonest cause of hearing loss among the implantees contributing 16(41%) followed by birth asphyxia, 8(20.5%). (Figure 2).

Lateralization of cochlear implantation among cochlear implantees.

The study has found that the majority of the implantees, 37(94.9%) to have been implanted with one device.

Complications post cochlear implantation among the implantees.

Facial weakness, 3 (7.7%) was the most encountered complication among the implantees.

Post-operative auditory and speech performance of one of the pre lingual after cochlear implantation.

The Infant-Toddler Meaningful Auditory Integration Scale (IT-MAIS) was used to follow up some patients, and assess their auditory development. MAIS scores for one such patient demonstrates the benefit observed post-operatively. This child was implanted at the age of two years and six months, and followed up over eighteen months. The questionnaire was scored out of 40 points, and a great improvement from 0/40 pre-operatively to 36/40 eighteen months post-operatively has been observed (Figure 3).

DISCUSSION

The study aimed to describe the clinico-demographic characteristics of the cochlear implantees and the outcomes of the intervention in our setting as the first novel findings since the establishment of in - country cochlear implantation programme. Majority of the implantees had pre lingual hearing loss and were in the age group of 2 -3 years. This could be due to the fact that, majority of parents realise that their children could not talk as their peers at this age and this is attributed to lack of neonatal screening in most of our settings as compared to high income countries where such children with hearing loss are detected early in life and prompt intervention.[6]

In our cochlear implantation programme, which is supported by the government, the age limit has been set at 5 years of age and this led almost 95% of our implantees being those with pre lingual hearing loss. Bilateral severe to profound hearing loss has been another criterion for cochlear implantation in our setting and thus more than 90 % of the implantees had bilateral profound hearing loss and such patients had been on hearing aid machines for at least 3 months without improvement.[7]

The most common causes of hearing loss in this study were due to ototoxicity (gentamicin, quinine and streptomycin) and birth asphyxia. According to WHO these causes of hearing loss can be prevented in 60% of the population. Several workshops have been conducted with gynaecologists and paediatricians in our setting to address these challenges and design policies on judicious use of antibiotics and other drugs. Our study on the most common causes of hearing loss concurs with similar studies in literature.[8-12] Other causes included genetics, familial, jaundice, infections and unknown aetiologies. Among the candidates implanted one was a case of Waardenburg and the other incomplete partition of cochlear type 3 (IPT3) which suggested genetic mutations.

The Tanzanian government pledged to support each of the candidates with one cochlear implant, except in two cases where the implantees received two devices. This was because of the cost implications related to the cochlear implantation device. In the developed countries bilateral cochlear implantation is an encouraged policy to ensure binaural hearing and continuous nerve stimulation. However in some cases with unilateral cochlear implantation, hearing aid device can be used in the other ear though studies are ongoing to ascertain its benefit as compared to the former (bilateral cochlear implantation).[13, 14]

Complications that arose post cochlear implantation included facial weakness, which was the commonest,

followed by skin infection at the surgical site which led to extrusion of the device. Other complications such as meningitis, was not reported and this could be attributed to the provision of pneumococcal polysaccharide vaccine (pneumovax 23) and Haemophilus influenzae vaccines prior surgery.

Currently, some of the children who underwent surgery in 2017 to 2019 are attending mainstream schools alongside with their hearing peers and some are bilingual.

CONCLUSION

Cochlear implantation in resource – limited settings is possible and cost effective if there is a great support from the government, device manufacturers and mentor surgeons from other countries who are advanced in cochlear implantation. Availability of rehabilitative services is very important for the better outcome of cochlear implantation. There is a great need for all the stakeholders in the developing countries to work together and address hearing loss, since 80% of the cases with hearing loss are preventable.

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Risk factors for vesicovaginal and rectovaginal fistulae in women treated at Juba Teaching Hospital in 2020-2021: A retrospective study

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ABSTRACT

Introduction: Vesicovaginal fistulae (VVF) and rectovaginal fistulae (RVF) are major public health concerns especially in sub-Saharan Africa. Our hypothesis was that prolonged obstructed labour, teenage marriages, inadequate emergency obstetric care and poverty are responsible for a high prevalence of VVF and RVF in South Sudan.

Objective: The objective of the study is to determine the risk factors for vesicovaginal and rectovaginal fistulae in women treated at Juba Teaching Hospital in South Sudan.

Method: Data were obtained from the files of all the 40 women who were operated on for VVF and RVF in Juba Teaching Hospital (JTH) during the 2020 and 2021 fistula campaigns; three women were interviewed.

Results: Thirty-six women had VVF, two had RVF and two had both. The main cause was obstructed labour. Spontaneous vaginal delivery accounted for 22 cases, Caesarean Section for 13 while five had a forceps delivery. Four women were aged under 18 years; the age of marriage was below 18 years for 22 women and between 18-25 years for 18 women. Of these 40 women 21 were para 1 & 2,14 were para 3 to 5 and five were para 6 and above; 17 had delivered at home and 23 in hospital; 18 had been attended by midwives / doctor and 22 by Traditional Birth Attendants (TBAs) in the villages; 22 were in labour for more than three days. Thirty-two deliveries had resulted in stillbirths. Most women had not been to school, and all were classified as 'poor'. Almost all (38) had not attended an ante-natal clinic (ANC) during the pregnancy in which they developed fistula. Ten women were operated on more than three times, 10 twice and 20 once. Nine women were divorced after developing fistula and 13 were abandoned. The psychosocial consequences included childlessness, stigmatization, depression, divorce, and abandonment.

Conclusion: The main risk factor for developing obstetric fistula was prolonged obstructed labour. Secondary risk factors were delivering at home, lack of obstetric care facilities, deliveries attended by unskilled health workers and TBAs, poor ANC attendance during pregnancy, cultural factors that encourage early marriage, low socioeconomic status, and lack of education.

Keywords: Vesicovaginal fistula, rectovaginal fistula, risk factors, fistula campaigns, South Sudan.

INTRODUCTION

Vesicovaginal fistulae (VVF) and rectovaginal fistulae (RVF) are major public health concerns globally and especially in sub-Saharan Africa.

Obstetric complications are the leading cause of fistulae in sub-Saharan Africa in a review of articles published from 1987-2008.[1] The high rates of VVF and /or RVF or both in the region reflects the poor quality and the level of perinatal care provided by the local health systems.[2]

Approximately 50-80 women/ year attend fistula campaigns in South Sudan with around half having a fistula and receiving a repair.[3] It was estimated in 2013 that at least 30 women out of 100,000 deliveries have obstetric fistulae either VVF or RVF or both despite the efforts being made by both health partners and the Ministry of Health.[3]

These fistulae are a serious health burden on the women of South Sudan which has the highest maternal mortality rate in the world at 2,054 per 100,000 live births,[4] 90% of deliveries occur in rural areas with only 10% attended by skilled midwives.[5]

In South Sudan obstetric fistulae are treated at the Teaching Hospitals in Juba (the main referral hospital where most obstetric fistulae are treated in the Dr Festo Fistula Centre) and in Wau and Malakal.

The objective of the study is to determine the risk factors for vesicovaginal and rectovaginal fistulae in women treated at Juba Teaching Hospital in South Sudan.[6]

METHOD

Data from the files of all 40 women and girls of reproductive age (12-49 years) who were operated on for VVF and RVF in the 2020 and 2021 fistula campaigns in JTH were analysed. The collected variables included cause of fistula; age at marriage; psychosocial consequences; accessibility to treatment and maternal health care and coping mechanisms. Interviews (after obtaining informed consent) were conducted with three of these women who were still in the ward. Both qualitative and quantitative methods were used to analyse the data. Frequency tables are used to present the data here.

RESULTS

Forty women were treated at JTH during the fistula campaigns of December 2020 and July 2021: 29 in 2020 and 11 in 2021. Over the two campaigns 36 women had VVF, 2 had RVF and 2 had both.

Table 1 shows the patient characteristics in terms of age, age of marriage, education, and parity.

Sixteen women came from Central Equatoria State, followed by 11 from Eastern Equatorial State, four from Western Bahr El Ghazal State, three each from Warrap State and Jonglei State, two from Lakes State and one from Upper Nile State; there were no cases from Western Equatorial, Northern Bahr El Ghazal and Unity States.

The characteristics of the women according to the mode and place of delivery, attendant at delivery, and foetal outcome are shown in table 2.

Of the 40 women who were operated on, 10 had been

Table 1. Characteristics: age, age of marriage, education and parity (N = 40)

Variable		n (%)
Age (years)	<15	0
	15-18	4 (10.0)
	19-25	17 (42.5)
	>25	19 (47.5)
Age of marriage (years)	<18	22 (55.0)
	18-25	18 (45.0)
	>25	0
	Total	40 (100.0)
Education level	P1-P4	1 (2.5)
	P5-P8	5 (12.5)
	None	34 (85.0)
Parity when fistula developed	Nulliparous	10 (25.0)
	Para 1-2	11 (27.5)
	Para 3-5	14 (35.0)
	Para 6 and above	5 (12.5)

Table 2. Mode and place of delivery, attendant at delivery, and foetal outcome (N=40)

Variable		n (%)
Mode of delivery	SVD	22 (55.0)
	Assisted by vacuum or forceps	5 (12.5)
	Caesarean Section	13 (32,5)
Place of delivery	Home	17 (42.5)
	Hospital	23 (57.5)
	PHCC	0 (0.0)
	Total	40 (100)
Delivery attended by	TBA	18 (45.0)
	MCH worker	0 (0)
	Skilled midwife/ doctor	22 (55.0)
Fetal outcome	Still birth	32 (80.0)
	Live	8 (20.0)
	Para 3-5	14 (35.0)
	Para 6 and above	5 (12.5)

Table 3. Marital status after fistula, social relationships, age first fistula, age first delivery (N=40)

Variable		n (%)
Marital status after fistula	Married	31 (77.5)
	Divorced	9 (22.5)
Social Relationship	Abandoned by husband/friends	13 (32.5)
	Not abandoned by husband/friends	27 (67.5)
Developed fistula at first delivery	Yes	27 (67.5)
	No	13 (32.5)
Age at first delivery (years)	12-17	17 (42.5)
	18-25	22(55.0)
	>25	1 (2.5)
	Skilled midwife/doctor	22 (55.0)
Fetal outcome	Still birth	32 (80.0)
	Live	8 (20.0)
	Para 6 and above	5 (12.5)

previously operated on 3 or more times, 10 twice and 20 once. Most women (34) were referred directly to JTH, 26 during 2020 and 8 during 2021. Wau and Malakal teaching hospitals referred 5 cases and a state hospital referred one case over the two campaigns.

More than half of the women (22) had developed a fistula after a labour that had lasted three or more days, 10 developed one after two days and 8 on the first day of labour.

Table 3 shows the marital status after fistula, social relationships, age first fistula, age first delivery

Twenty-three women had children and 17 were childless. None of the women had undergone FGM, which is not usually widely practiced in South Sudan.

Stories from the women interviewed

Here we summarise three quotes from the interviews with the women operated on at JTH:

> "I have lived with this satanic disease for more than ten years now. Before, I used to work hard in my farm and grow sorghum, sesame, groundnut but now, am just in miserable life of washing my beddings and once I have finished the bed sheet that I have been washing, the urine has already flown in the other one, I cannot manage to get food by my toil and my husband and relatives have already forgotten me, I beg to live".

> "I hate myself with this disease, I don't know why God has made me this way, many friends, and relatives have now abandoned me, I stay alone, I am better treated here now in the hospital than when am staying at home"

> "I feel being well cared and happier and comfortable when I am in the church and in the hospital like this, but at home, am not well treated but my people have hated me due to smell of urine for all these years"

DISCUSSION

The most significant risk factor for the development of obstetric fistula in this study was prolonged obstructed labour as has been found elsewhere.[7] More than half the women had developed a fistula after a labour that had lasted three or more days. About half our women had delivered at home and half had been delivered by a TBA. None had attended ANCs.

These data, and the fact that ten women had been operated on for their fistulae three or more times, together with the 'stories' of the women interviewed suggest that lack of skilled obstetric care, untrained health workers, and cultural restrictions on women attending ANCs contributed to women in our study developing obstetric fistulae.

Gynaecological procedures and radiotherapy accounted for no cases in our study. This does not mean that there were no cases but may be due to poor recording, or inaccessibility or unaffordability of transport to Juba.

Most of those (34/85%) who developed fistula had had no schooling. Educated girls are more likely to know the risks associated with early pregnancy, and the importance of healthy behaviours. Education raises the status of women and enhances their confidence making it more likely they attend health services. We do not know why most of the women in our study did not attend school but a literature review [6] suggests that it may be related to cultural influences, lack of schools, and money to pay fees for school.[8] There is need to increase access to free universal primary education and, more especially, empowerment of girl child education in the far-to-reach areas of South Sudan.

Over half the girls in our study had been married, and nearly half had had their first delivery before the age of 18 years. Early marriage is common in some communities in South Sudan which look at girls as a source of wealth when they are married with dowries. Thus, increasing the risk that a girl becomes pregnant before the pelvis is adequately matured.

A consequence of developing an obstetric fistula is a stillbirth and most of the deliveries in our study resulted in a stillbirth. South Sudan has the highest neonatal mortality rate in Africa and there appears to be still no

increase in deliveries conducted in hospital by skilled birth attendants.[4] Only two of the women had attended focussed ANC. Many deliveries were at home, and nearly half by a TBA.

Other effects are abandonment, stigmatization, loss of employment and social support, psychological pain and infertility, worsening of pre-existing psychological, psychiatric (including suicidal thoughts), and medical illnesses. All three women who were interviewed said that they would commit suicide if they did not heal soon. The psychosocial consequences were found to be consistent with the previous studies in South Sudan and other countries such as Nigeria and Ethiopia.[8,9,10]

The rate of divorce and abandonment for the women in this study was low compared to other studies, for example in Nigeria.[10] The majority remained in marriage after developing a fistula and few were rejected by the community and relatives. We postulate that this is probably because South Sudanese have specific cultural and customary traditions and attitudes that frown on divorce, whatever the reason. More studies need to be done to understand.

CONCLUSION

The main risk factor for developing obstetric fistula was prolonged obstructed labour. Secondary risk factors were delivering at home, lack of obstetric care facilities, deliveries attended by unskilled health workers and TBAs, poor ANC attendance during pregnancy, cultural practices that encourage early marriage, low socioeconomic status, and lack of education.

Recommendations

We recommend that development of the health system in South Sudan must include improved training and motivation of midwives and doctors and focussed ANC services, and the establishment of fistula centres. Provision of free basic education, especially for girls in the far-to-reach areas and discouraging early marriage, should also be among the top priorities of government.

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Obstetric fistulae: a review

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ABSTRACT

Obstetric fistula is one of the most feared complications of labour, leaving a woman with a life of ostracism and shame due to her complete incontinence. Many women see this as a curse for something they did and many end up trying to take their life. Obstetric fistula patients present with a complicated array of physical and psychological needs which should all be addressed by the medical team caring for her. In the majority of cases, obstetric fistula is curable as long as the doctor is trained in and skilled in the right surgical techniques. However, the most important message is that obstetric fistula is preventable. Ensuring that all women receive proper, affordable and timely medical care in labour will ensure that women will not need to fear such dreadful sequalae of trying to have a baby.

Key words: Obstetric fistula, vesicovaginal, rectovaginal, South Sudan, symptoms, treatment.

INTRODUCTION

Obstetric fistulae have been occurring ever since women first started to deliver babies. However, it was not until recently, only around 350 years ago, that the first recorded case of an obstetric fistula was cured in Europe. James Marion Sims, the father of modern gynaecology developed flap splitting surgical techniques for fistula repair and is credited as the first surgeon to more reliably cure fistula patients. His work dates back to the 19th century and took place largely in New York where obstetric fistula were common at the time.

PREVALENCE AND EPIDEMIOLOGY

Obstetric fistulae have been almost eradicated where women have access to safe and affordable emergency obstetric care including Caesarean Section. However, wherever women do not have ready access obstetric fistulae occur.

No one knows how many fistula patients are still waiting for treatment or how many are added to that number each year. One large hospital-based study estimated 0.35% of deliveries in areas without access to safe and affordable obstetric care.[1] Extrapolating from this, there may be two million women living with obstetric fistulae and 50-100,000 new cases each year.[2] A meta-analysis from 2013 showed that these estimates are probably exaggerated and suggested that the prevalence is 0.29 per 1000 women of reproductive age meaning that there are probably around one million women living with this condition.[3]

There is geographical variation. In countries with a more accessible health services there will be lower maternal deaths and lower incidence of obstetric fistulae. In countries like South Sudan, with a high maternal mortality rate, obstetric fistula sufferers will be numerous and the Ministry of Health of South Sudan has estimated that there are some 60,000 women suffering from obstetric fistulae in South Sudan alone.

It is clear from numerous studies that obstetric fistulae occur simply because of a long, unrelieved obstructed labour.[4,5,6,7,8,9,10,11] Labour should last well below one day The active phase of labour in a primiparous woman averages 12 hours

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and for a multiparous, eight hours. However, women who develop an obstetric fistula have been in labour on average for 3.9 days.[5] During the long labour, the presenting part is impacted against the bony pelvis of the mother, crushing the intervening tissues of the urinary and genital tracts, and in around 5-10% of cases the tissues of the rectum and anus and vagina as well. The necrosed tissues ultimately slough away and the fistula can occur between the ureter, bladder and / or urethra and uterus, cervix and or vagina and in 5-10% of cases, between the rectum and / or anus and vagina as well. Total incontinence, shame, divorce, isolation and suicide are commonplace.

What is more controversial is the claim that obstetric fistula is associated with early marriage and early pregnancy and this is often quoted as a proven fact. It is true that about 50% of women who develop fistulae do so in their first delivery, but equally, around 50% of women get their fistula on their subsequent delivery. This claim makes common sense as the female pelvis does not fully mature until some two years after the menarche, meaning a pregnancy in this young age could lead to obstruction and hence fistula. However, a study from Tanzania found that there was no association with an early pregnancy and fistula and although not statistically significant, there was less obstructed labour and fistula formation in those women getting pregnant very young.[12]

It is often argued that there are over 500,000 teenage pregnancies in the USA each year and no obstetric fistula due to obstructed labour because all women have access to a Caesarean Section if needed. Fistulae occur in areas of poor access to Caesarean Sections. These populations are poorly resourced, educated and get married and pregnant early. The fact that getting pregnant young is the norm and is related to poverty does not prove that an early pregnancy is the cause of a fistula. Fistula will only be eradicated when all women can deliver safely with appropriate obstetric care.

A more worrying trend is the rise of the iatrogenic fistula around Africa and South East Asia. It is estimated that around 25% of all women suffering obstetric fistulae in Eastern Africa have done the right thing and come to hospital to deliver their babies, but received a fistula at the hand of the doctor during a Caesarean Section. The definition of this outcome is a Caesarean Section carried out after less than a day of labour and delivering a live child; that is, her labour has not yet damaged the tissues.

Hence the only possible cause of the fistula is faulty surgical technique.[13,14] It has become common practice not to properly mobilise the bladder and ureters during a Caesarean Section making it very easy to incorporate the bladder into the repair of the uterus leading to fistula formation. Ureteric fistulae are almost always iatrogenic, the ureters being sutured into the lower segment especially if the angle of the uterine incision has torn laterally.

The only way to prevent obstetric fistulae is for all women to gain access to an operative delivery to relieve the obstructed labour if it is needed. However, there are many barriers to this: lack of awareness to get help in labour, needing permission from the mother-in-law or husband to go to hospital, lack of transport or funds to get to the hospital, a reluctance for fear of abuse physically or verbally at hospital. There is also a common belief (often correct) that they will receive poor quality care as the local health facilities are under-resourced and staff unmotivated for the lack of pay for months. The fear of having to pay or inability to pay hospital fees also keeps patients at home.[15,16,17,18]

SYMPTOMS

It is wrong to think that a woman who has an obstetric fistula just has a hole in her bladder making her incontinent. The ischaemic process that causes the fistula affects all the tissues in her pelvis, the muscles, nerves, bones as well as reproductive, urinary and alimentary tracts. It is a 'field injury' affecting a wide variety of structures and the term 'the obstructed labour injury complex' is used to describe it.[19]

Thus, the symptoms include:

1. **Urinary incontinence** caused by a fistula anywhere from the ureter to urethra and communicating with the cervix and/ or vagina. The fistula can be small, less than one centimetre in diameter, or large, having destroyed the whole anterior vagina and almost all of the bladder and urethra. Despite the size, the incontinence is complete, leaking urine every minute of every day.
2. **Faecal incontinence** can involve the rectum and or anus and posterior vaginal wall.
3. **Bone damage.** 32% will have some bony abnormality in the pelvis, more commonly separation or obliteration of the symphysis pubis.
4. **Hydroureters and hydronephrosis.** Scar forms in the pelvis after the necrotic tissue comes away causing obstructive uropathies in 49% of patients.
5. **Levator muscle damage.** Some women have their whole levator plate sloughed away.
6. **Nerves.** Some 20% will have damage to the lumbosacral plexus or the common peroneal nerve as it traverses the head of the fibula causing some degree of foot drop.

Then because of their incontinence they suffer later, secondary conditions occur.

1. **Urine dermatitis** from the urine irritating the skin.
2. **Lower limb contractures** from long periods of

immobility, usually associated with foot drop.

3. **Malnutrition and anaemia,** especially if they have been divorced and ostracised with no one to care for them.
4. **Mental health issues.** Up to 100% of women will screen positive for some form of mental illness most notably depression. Up to 40% have thought about or attempted suicide.

TREATMENT

The mainstay of treatment is surgery, although some fistula can be cured by prolonged catheterisation soon after delivery. If the fistula is small, less than two centimetres, and doesn't involve the pelvic side walls, then inserting a catheter to drain the urine away from the fistula can result in spontaneous closure of the defect, as long as the catheter is inserted within one to two weeks of the delivery. However, it is essential to make sure the catheter has not been passed through the fistula and into the vagina. This of course will keep the fistula open.

Most surgeons wait until three months has passed since the obstructed labour that caused the fistula. This gives the tissues time to recover from the ischaemic insult. Some senior fistula surgeons will operate earlier than this, but the tissues are much more difficult to handle and the sutures often tear out. It is best to wait until the tissues have healed.

The most important thing about fistula surgery is to only operate if you have had specific training in the relevant surgical techniques. Unfortunately, there are many surgeons who try fistula surgery and are sometimes paid to try, but they don't know the steps and they usually leave a high number of failed fistula operations. This is devastating for the patient who might give up hope that they can ever be cured. A subsequent operation will be much more difficult as the first operation has just resulted in more tissue damage.

From my personal series of fistula operations, about 60% of the cases I see in South Sudan have been operated on before, sometimes five or six times with no benefit. This makes a successful cure from the next operation unlikely. Every surgeon will have failures, but any skilled surgeon, with appropriate training, should be able to successfully close 90% of all fistulae at the first operation.

To achieve this these five basic principles should be followed:

1. **Good exposure,** with good specula, retractors and assistants.
2. **Protect the ureters.** It is easy to cut or ligate a ureter during a fistula operation. You must identify and catheterise the ureters in all fistula defects that lie close to the uretero-vesical junction.
3. **Mobilise the urinary tract away from the genital tract**. This must be done slowly and meticulously without tearing at the tissues. The tissues are often thin and fragile. Tearing and rough operating just makes the damage even worse.
4. **Tension free closure.** Mobilise the tissues adequately to get a tension free closure of the urinary tract. Any tension on the tissues will cause the healing union of the tissues to fail.
5. **Check you have it closed by doing a dye test.** Always perform a dye test by inserting at least 60ml of dilute dye into the bladder to ensure you have a water tight closure. If there is any leak, the operation with fail.

By adhering to these basic principles at least 90% of obstetric fistulae will be closed at the first operation. However, this doesn't mean that the patient will be continent. Up to 33-45% of all fistula patients will have some degree of ongoing urinary incontinence despite a closed fistula. This is because the structures and muscles that keep a woman continent have been affected by the ischaemic process. More recently there has been three more principles added to the above five that will not only increase the successful closure of the fistula but reduce the rate of ongoing incontinence. These principles aim at restoring normal anatomy.

1. **Restore the normal length and width of the urethra**. Around one third of women will have their urethras affected by the fistula injury and unless the urethra is reconstructed to a normal length and width out of bladder tissue, then the chance of ongoing incontinence is unacceptably high.
2. **Support the urethra** with a sling to reconstruct the pubourethral ligament. This can be made from levator complex or rectus sheath.
3. **Repair the vagina with no tension.** Remember there is also tissue loss to the vagina and pulling it together pulls tension on the urethra just pulling it open and making the patient incontinent. If there is loss of the vaginal tissue you need to use specialised flaps to restore normal vaginal anatomy.

Training in these steps and following them should not only get more than 90% of fistulae closed, but the ongoing incontinence rate after repair should lower and approach 15%.

CONCLUSION

Obstetric fistula is a devastating condition to those who suffer from it. It is treatable but the surgeons should only attempt a repair after having received adequate training without which they run the risk of doing more harm than good.

More importantly, obstetric fistulae are entirely preventable. We shouldn't see women suffering from a long unrelieved obstructed labour which runs the risk of them dying and if they survive, of getting a fistula. This requires an enormous investment into health care which will take time and commitment, but it is achievable.

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Diabetic Ketoacidosis in Adults: Part 1. Pathogenesis and Diagnosis

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ABSTRACT

The metabolic derangements that lead to Diabetic Ketoacidosis (DKA) are described. Understanding the pathogenesis is the key to rapid and accurate diagnosis and hence successful management. DKA may often be prevented by clear advice to patients about how to manage their type 1 or ketosis-prone type 2 diabetes during periods of intercurrent illness. DKA must be considered in the differential diagnosis of metabolic acidosis even where other diseases that may present similarly, such as malaria, are highly prevalent.

Key words: Diabetic ketoacidosis, pathogenesis, diagnosis, emergency, prevention.

INTRODUCTION

Diabetic Ketoacidosis (DKA) is a life-threatening medical emergency characterized by high anion gap metabolic acidosis, hyperglycaemia, ketone accumulation and volume depletion. Although traditionally considered a complication of type 1 diabetes, DKA is now also commonly encountered as a complication of 'ketosis-prone' type 2 diabetes. Diagnosis may be delayed, particularly in those presenting with a new diagnosis of diabetes, as its presentation closely mimics other medical emergencies caused by a range of conditions such as infectious diseases or the 'acute abdomen' (e.g. appendicitis, diverticulitis, bowel perforation).[1] Although mortality rates have fallen to <1% in some countries over the past 20 years, they have been reported to remain as high as 29% in others.[1] The presentation of malaria, common in tropical Africa and many other parts of the world, with confusion, diarrhoea and vomiting, malaise, metabolic acidosis and renal impairment closely mimics the presentation of DKA and can sometimes lead to delayed diagnosis of DKA. Timely recognition of DKA is important to minimize complications and death. Many individuals with new onset diabetes, including children, remain undiagnosed until they present with DKA.[2]

This review is presented in three separate articles in the same issue of the journal. In this, the first part, we discuss the pathogenesis and diagnosis of DKA. In part two we focus on the clinical management of DKA and in part 3 we consider some of the pitfalls that may be encountered, as well as the management of DKA in some special situations, such as renal failure and pregnancy.

Management of children with DKA is beyond the scope of this article and the reader is referred to detailed published guidelines for the management of DKA in children.[3]

PATHOGENESIS OF DKA

Clear understanding of the pathogenesis of DKA is helpful in planning its management. Figure 1 summarizes this pathogenesis which is explained in detail below.

DKA develops either due to absolute insulin deficiency or to relative insulin

deficiency associated with an increase in counter-regulatory hormones (especially glucagon, catecholamines and cortisol).

During conditions of reduced insulin/glucagon ratio, hyperglycaemia exceeding the threshold for renal tubular reabsorption of glucose develops causing a brisk osmotic diuresis with water and electrolyte losses which lead to dehydration and eventually extracellular fluid volume depletion. The ensuing plasma volume contraction causes compensatory release of more counter-regulatory hormones (particularly cortisol and catecholamines) which lead to ever worsening hyperglycaemia and volume depletion in a positive feedback loop. At the same time, decreased renal plasma flow causes secondary hyperaldosteronism and additional potassium losses.

Individuals with DKA are potassium depleted, often with a total body K^+ deficit of 400 mmol or more (3–5 mmol/kg). As most K^+ ions are intracellular, and in the presence of acidosis and insulin deficiency, blood tests often give a poor estimate of the severity of the deficit and can show low, normal or even high serum [K^+] concentrations.[4] The positive feedback loops caused by volume depletion thus worsen hypokalaemia and hyperglycaemia and are important in the development of DKA.[5]

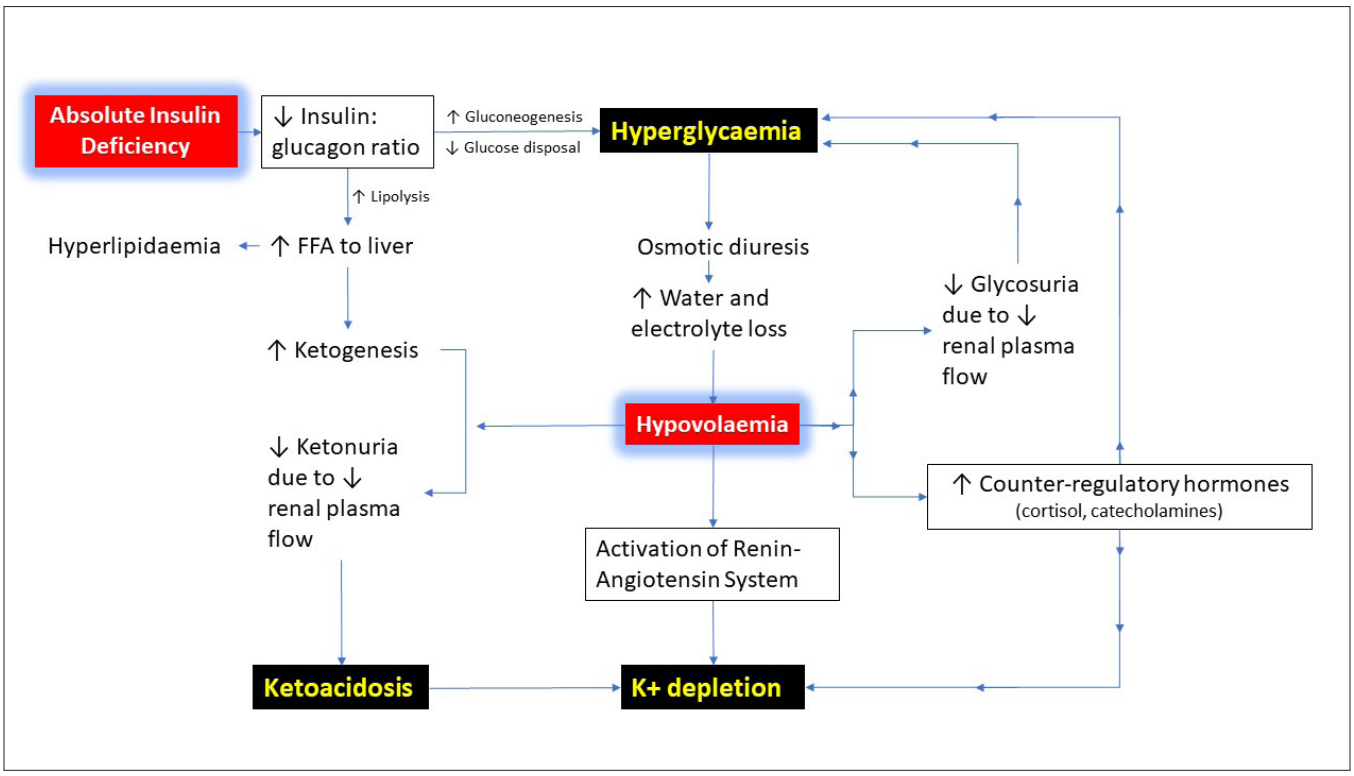
Normally, hyperglycaemia should suppress glucagon via insulin release within pancreatic islets. In DKA, glucagon levels may be markedly elevated despite profound hyperglycaemia. This occurs due to sympathetic stimulation of islet alpha cells secondary to the volume depletion that develops during DKA, and also due to severe loss of beta-cell mass (or function) that removes tonic inhibition of the alpha cell by insulin. Elevated glucagon levels increase both hyperglycaemia and ketone production.[6]

With insulin deficiency and high glucagon levels, the low insulin to glucagon ratio promotes breakdown of triglycerides through activation of hormone sensitive lipase in adipose tissue releasing free fatty acids (FFA) and glycerol. FFAs generate acetyl-CoA in the liver (through beta-oxidation) which then enter the Krebs cycle to generate ATP. As acetyl-CoA is produced in large quantities under these conditions, the capacity of the Krebs cycle becomes saturated and acetyl-CoA diverts into the ketogenic pathway resulting in the production of ketone bodies (acetoacetate, beta-hydroxybutyrate and acetone).[7]

Hyperglycaemia, increased glucagon and increased levels of other counter-regulatory hormones such as adrenaline and cortisol all contribute to insulin resistance and decreased glucose uptake in skeletal muscles.[8]

PREVENTION OF DKA- 'SICK DAY RULES'

DKA will be slow to develop in a well hydrated individual even in the presence of significant insulin deficiency.



FFA (Free Fatty acids), Gluconeogenesis (glucose synthesis)

Figure 1. Pathogenesis of DKA (Credit: Ali Azkoul, Sing Sim and Victor Lawrence)

Attention to hydration, together with continuing insulin administration, forms the basis of the 'sick day rules' which should be explained to all patients at risk of DKA to equip them to avoid it. In the UK, the patient group 'Diabetes UK' has a website with a wealth of information (available in many languages) for both patients and medical professionals, including information on 'sick day rules', the use of which may help prevent the development of DKA in patients during an intercurrent illness https://www.diabetes.org.uk/. These guidelines are primarily designed for those who manage their diabetes with basal-bolus insulin regimes or insulin pumps. It is much more difficult to adjust insulin doses in twice daily mixed insulin regimes and no universally accepted guidance exists on this although the principles are as follows.

In general, it is vitally important, in order to avoid DKA, that people with type 1 diabetes never stop their insulin and consider 10-20% dose increases particularly if blood glucose levels exceed 10-14 mmol/l and/or in the presence of increased blood ketone levels (hydroxybutyrate >0.6 mmol/l and particularly when >1.5 mmol/l) or more than trace urine ketones. It is advisable to test blood glucose levels, and, where possible, urine or (ideally) blood ketone levels at 4-6 hourly intervals day and night during acute intercurrent illness. Testing at 2-hour intervals is recommended if blood glucose levels exceed 14 mmol/l and/or capillary blood beta-hydroxybutyrate levels exceed 1.5 mmol/l. It is important to emphasise that insulin should not be stopped even if vomiting. In this situation, carbohydrate-rich fluids should be sipped if less than about 50g carbohydrate can be taken in as food in a 4–6-hour period unless blood glucose levels are increasing or exceed 10-14 mmol/l in which case carbohydrate-free liquids should be taken instead. People with diabetes can be reassured that even if they continue to vomit, a significant proportion of the fluid and carbohydrate may still be absorbed. The principle here is to maintain the blood glucose levels high enough for normal/slightly increased insulin doses to be given safely (without inducing hypoglycaemia) in association with abundant fluid intake to prevent a spiral into DKA.

It is important for individuals with diabetes to seek urgent medical care if they cannot maintain the necessary fluid intake (ideally at least 100-200 mls/h in small regular amounts), become dehydrated or experience rising blood glucose and/or ketone levels (>1.5 mmol/l that do not reduce rapidly or >3.0 mmol/l at any time). Urgent medical care is also needed if symptoms of DKA develop (rapid breathing, abdominal pain, drowsiness, vomiting where this is not part of the intercurrent illness). Women with diabetes who are pregnant, unwell and have elevated ketones should seek urgent medical care, usually in hospital, immediately.

Certain medications should be temporarily discontinued during intercurrent illness to avoid side effects driven by any associated dehydration. These include, but are not limited to, ACE inhibitors/Angiotensin Receptor Blockers, diuretics, metformin, and non-steroidal anti-inflammatory drugs (NSAIDS). Sodium-glucose Cotransporter-2 (SGLT2) inhibitors should not generally be used in patients at risk of DKA (type 1 diabetes or ketosis-prone type 2 diabetes) and should certainly be stopped during an intercurrent illness.

Education for 'sick days' and ideally provision of ketone meters and testing strips with instructions about how and when to test (e.g. on sick days, where blood glucose levels exceed 14 mmol/l on 2 consecutive tests or if exercise is planned after one test or on any occasion that blood glucose is >18 mmol/l) should be an integral part of diabetes care.

PRECIPITATING FACTORS FOR DKA

- New diagnosis of type 1 diabetes.
- Limited adherence to agreed insulin treatment plans (e.g., because of conflicting health priorities or beliefs or lack of availability or affordability of insulin) or inadequate insulin doses (e.g., equipment failure, lack of dose titration or attempts to control weight by use of inadequate insulin doses, an increasingly recognised form of self-harm particularly where recurrent).
- Insulin denaturing due to storage at temperatures over 25-30^{0}C (depending on the specific insulin brand) or inadvertent freezing during storage close to the freezer compartment in a refrigerator. Unopened insulin should ideally be kept refrigerated at 2-8^{0}C during storage. The pen/vial in current use can generally be stored at 'room temperature' depending on the manufacturer's instructions as long as it is out of direct sunlight and assuming 'room temperature' is less than 25-30^{0}C). When damaged by heat, 'clear' insulin may become cloudy and 'cloudy' insulin may appear 'grainy' and stick to the side of the glass. Insulin that has been exposed to bright sunlight sometimes has a brownish colour. If there is a risk of heat-damage, insulin in use should also be stored in the refrigerator or in a 'cool bag' as long as it does not freeze. Insulin which may have been damaged by temperature should never be used.
- Intercurrent illness – e.g., acute coronary syndrome, acute infectious diseases, pancreatitis, cerebrovascular accident, diarrhoea and vomiting, Covid-19 infection without attention to 'sick day rules' (see 'sick day rules' above).
- Drugs – SGLT2 inhibitors, steroids, atypical antipsychotics, tocolytics in pregnancy.
- Ketosis prone type 2 diabetes (often in African/Caribbean populations).

Dehydration	→	Dry tongue, loss of skin turgor, tachycardia and hypotension
Hyperglycaemia	→	Polyuria, polydipsia, fatigue, headache, abdominal pain (impaired gastrointestinal motility)
Ketoacidosis	→	Deep breathing (Kussmaul's sign), sweet smell of mouth breath (acetone)
Hypokalaemia	→	Not usually clinically apparent unless exceptionally severe

DIAGNOSIS OF DKA

The cardinal clinical features of DKA are shown in the box above.

All of these acting together may contribute to drowsiness. Although the term 'diabetic coma' is sometimes used, true coma, as opposed to a degree of drowsiness, is very unusual and other causes should be considered where a patient in DKA presents with true coma (e.g., central nervous system infection, stroke).

The Joint British Diabetes Society for Inpatient Care (JBDS-IP) guidelines for DKA diagnosis.[9]

Reference to this excellent, freely available, and comprehensive guideline for detailed algorithms for the diagnosis and hour by hour management of DKA in adults is recommended. Unless specified to the contrary, Part 2 of these reviews[10] - giving practical management guidance- has been taken and adapted from this guideline.

All the following must be present to make the diagnosis of DKA:

1. The **'D'** – a blood glucose concentration of >11.0 mmol/L (200 mg/dL) or pre-existing diabetes mellitus
2. The **'K'** – a capillary or blood ketone concentration of >3.0 mmol/L (measures beta-hydroxybutyrate) or significant ketonuria (2+ or more on standard urine sticks which measure acetoacetate). Blood beta-hydroxybutyrate levels >6.0 mmol/l indicate 'severe' DKA.
3. The **'A'** – a bicarbonate concentration of <15.0 mmol/L and/or venous pH <7.3. Arterial blood gasses are generally un-necessary in DKA unless there is evidence of hypoxaemia.

DKA causes a high anion gap metabolic acidosis. The American Diabetes Association (ADA) recommends calculating the anion gap and this may be particularly useful if direct ketone measurement is not possible.[6] Anion Gap = $[Na^+] + [K^+] - [Cl^-] - [HCO_3^-]$ Normal range 10-14.

A raised anion gap implies the presence of unmeasured anions which in this context are assumed to be ketones. This can be recalculated during treatment to assess progress if direct ketone measurement is not available.

INVESTIGATIONS

Further investigations depend on the context but may include any or all of the following tests depending on availability and degree of clinical suspicion

Blood tests:

- Venous blood gas (pH, HCO_3, electrolytes, lactate, glucose)
- Ketones (capillary/ urine as above)
- Glucose (blood and/or capillary)
- Serum urea and electrolytes
- Full Blood Count
- C-reactive protein
- Blood culture
- Amylase, cardiac enzymes etc.
- Tests for malaria/ other infections if suspected

Urine tests:

- Dipstick (ketones, signs of urinary tract infection, proteinuria etc.)
- Microscopy, Culture and Sensitivity.
- Pregnancy test in women of child-bearing age (DKA during pregnancy carries a high risk of foetal loss, see section on DKA in Pregnancy.[11]

Other:

- Electrocardiogram (ECG).
- Chest X-ray and/or other imaging.
- Lumbar puncture if central nervous system infection is suspected.

SUMMARY

In this, the first of three articles on DKA, we have discussed the sequence of metabolic derangements that culminate in DKA. Understanding the pathogenesis is the key to rapid and accurate diagnosis as well as to successful management of this condition. Furthermore, it will be seen that DKA may often be prevented by clear advice to patients about how to manage their type 1 diabetes during periods of intercurrent illness and by sound understanding of how to break or reverse the vicious cycles that all too often culminate in DKA.

We stress that DKA should be considered in the differential diagnosis of metabolic acidosis even in settings where other diseases that may present somewhat similarly, such as malaria, may much more commonly be encountered.

In part 2 of this review, we will discuss the management of DKA based on the Joint British Diabetes Society for Inpatient Care (JBDS-IP) guidelines for DKA.[9]

By these means, we hope that the goal of significantly reducing mortality from DKA, which is almost uniformly lethal if not recognised early and treated appropriately, may be realised.

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Diabetic Ketoacidosis in Adults: Part 2. Management

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ABSTRACT

The priorities for the management of Diabetic Ketoacidosis (DKA) are to assess severity and establish intravenous (i.v.) 0.9% NaCl rehydration with the careful addition of potassium ($[K^+]$). Ideally, a fixed rate insulin infusion should be used initially and addition of 10% glucose infusion when the blood glucose level has fallen to below 14 mmol/l. Regular clinical and laboratory monitoring, particularly of the rate of fall of blood ketones (beta-hydroxybutyrate) and of serum $[K^+]$ and glucose is essential to guide fluid and insulin infusion rates. When the criteria for resolution of DKA are met, the patient may be switched to subcutaneous (s.c.) insulin if eating or variable rate insulin infusion if not yet able to eat and drink. Basal insulin should be continued (or started) where possible alongside infused insulin during the treatment of DKA. If fixed rate insulin infusion is not possible, then intermittent s.c. insulin injections may be used instead.

Key words: Diabetic ketoacidosis, management, insulin, potassium.

INTRODUCTION

This is the second article in a three-part series on Diabetic Ketoacidosis (DKA) in adults in this issue of the Journal. This article will focus mainly on the practical management of DKA in adults.

The advice given throughout this review is based on the Joint British Diabetes Society for Inpatient Care (JBDS-IP) guidelines for DKA[1] adapted for situations where certain medical devices or blood tests, such as infusion pumps or blood ketone measurement, may not always be readily available. Emphasis is placed throughout on pathophysiological principles so that the diagnosis and management of this condition may be optimised using whatever resources are available at the point of care.

The priorities in DKA are, in approximate order of priority:

1. To make the diagnosis and identify, and if necessary, treat the precipitant.
2. To assess the severity and resuscitate (Airway, Breathing, Circulation) as necessary.
3. To correct volume depletion and restore renal plasma flow.
4. To replace potassium (K^+).
5. To administer insulin ideally by fixed rate i.v. insulin infusion (FRII).
6. To monitor the progress of treatment.
7. To determine when DKA has resolved, institute on-going diabetes treatment and give advice to prevent recurrence.

ASSESSMENT OF SEVERITY

Severe DKA is present if any one or more of the following features are present:

- Serum beta-hydroxybutyrate > 6.0 mmol/l
- Bicarbonate level below 5.0 mmol/L
- Venous/arterial pH below 7.0
- Hypokalaemia on admission (under 3.5 mmol/L)
- Glasgow Coma Scale score (GCS) below 12
- Oxygen saturation below 92% on air (assuming normal baseline respiratory function)
- Systolic BP below 90 mmHg
- Pulse over 100 or below 60 bpm
- Anion gap above 16.

The presence of one or more of the features listed above indicates 'severe DKA'. Of these, low BP particularly when not responding to administration of a fluid bolus, severely depressed conscious level, pH<7.0 with the possibility of impending cardiovascular collapse and hypokalaemia are ominous signs requiring rapid identification and correction.

RESUSCITATION

As with any medical emergency, assessment of Airway patency, adequate Breathing and Circulation (the ABC approach) is essential. If Systolic Blood Pressure (SBP)< 90 mmHg, give 500 ml N/Saline over 10-15 min and reassess/repeat. If not responding, continue treatment and consider requesting Intensive Care assistance if available.

IV Fluids

Fluids are central in the management of DKA bearing in mind that fluid depletion is a key part of the pathogenesis. If serum potassium is <3.3 mmol/L, this should be corrected by potassium infusion before starting insulin as insulin will drive K^+ into cells thereby further, and potentially dangerously, lowering the levels. It is important to remember that insulin only inhibits new ketone formation and does not directly reduce ketone concentrations which rely on metabolism and excretion in the urine and breath. Insulin should not be commenced at the risk of provoking dangerous hypokalaemia.

Start IV fluids with 0.9% NaCl (also known as Normal Saline, N/Saline).

The aim is to expand intravascular volume, restore renal perfusion, reduce counter-regulatory hormone production and reduce secondary hyper-aldosteronism. Excretion of ketones and glucose will increase as a result of volume correction.

A typical fluid deficit is of the order of 100ml/kg (10%) in severe DKA and a typical regime for replacement is shown in the table below.

0.9% Saline Administration Regime for Adults in DKA

Consider the need for one or more fluid boluses if SBP<90 mmHg (see 'Resuscitation' above). Following this, or if bolus not considered necessary, infuse:

- 1L 0.9% Saline over 1h i.v.
- 1L 0.9% Saline over 2h i.v.
- 1L 0.9% Saline over 2h i.v.
- 1L 0.9% Saline over 4h i.v.
- 1L 0.9% Saline over 4h i.v.
- 1L 0.9% Saline over 6h i.v.
- Note: K+ replacement is essential and see below for details.

Take a more cautious approach in the elderly, those with heart or renal failure, in pregnancy and in young people due to the increased risk of cerebral oedema. Consider the need for infusing glucose (5%, 10% or more concentrated depending on circumstances and availability) at the same time if blood glucose levels are not sufficient to permit adequate insulin infusion without hypoglycaemia (see 'Fixed Rate Insulin Infusion' below).

Potassium replacement

Most DKA patients will be K^+ depleted. A further drop in [K^+] may be anticipated with insulin infusion. Do not initiate insulin if potassium is <3.3mmol/l to prevent cardiac dysrhythmias and respiratory muscle weakness. Replace potassium intravenously and in general do not exceed a rate of 20 mmol/h by peripheral infusion (higher concentrations and rates may be given under Intensive Care type monitoring via a central line). Be prepared to adapt this regimen in patients with established renal failure and exercise great caution in potassium infusion until it is clear that there is a good urine output. Aim to maintain serum potassium levels between 4 and 5 mmol/l.[2]

Serum [K^+]	Amount of K^+ to add to each litre of 0.9% Saline
>5.5	Nil
3.5-5.5	20-40 mmol depending on [K^+] and fluid infusion rate
< 3.5	Seek senior advice, consider infusion via central venous catheter, do not start insulin until above 3.3 mmol/l

Fixed Rate Insulin Infusion (FRII)

- Add 50 Units of human soluble insulin (e.g., Actrapid, Humulin S) to 50 ml 0.9% NaCl in an infusion pump and start the fixed rate insulin infusion at 0.1 unit/kg/h. For example, in a 70 kg individual this gives 7 units insulin/hour (7 mls of this infusate per hour).
- If hourly blood ketones are not dropping based on target rates of decline (≥0.5 mmol/L/h), then increase insulin infusion rate by 1 unit/h and reassess after one hour.
- Where blood ketone measurements are not easily available, use venous [HCO_3^-] instead (target rise is ≥3.0 mmol/L/h). Plasma glucose can be used as a last resort (target fall is ≥3.0 mmol/L/h) although this is not reflective of the resolution of acidosis.

In the past, DKA was often treated using 'Variable Rate Insulin Infusions' (VRII, sometimes known as 'sliding scale insulin infusions'). These vary the number of units of insulin infused per hour according to the prevailing blood glucose levels and will usually result in successful treatment eventually. However, this approach has now widely been superseded by the 'Fixed Rate Insulin Infusion' in which a constant, weight-based dose of insulin is infused hourly and if necessary, glucose is infused at the same time alongside 0.9% Saline to prevent hypoglycaemia.

The drawback of VRII is that as the levels of blood glucose fall (mostly through expansion of the extracellular fluid compartment, glucose losses in the urine and increased glucose uptake, metabolism and storage in insulin sensitive tissues), the rate of insulin infusion may be decreased to a level that is no longer sufficient to suppress ketogenesis. In other words, adjusting insulin infusion based on glucose level and using it as a marker will lead to reduction of infused insulin whilst the patient is still in ketoacidosis and may prolong the duration of the ketoacidotic state.

By infusing a weight-based hourly amount of insulin that is known to suppress ketogenesis, even in the insulin resistant conditions of DKA, a more rapid correction of DKA is achieved. VRII can give the misleading impression that the target for correction in DKA is the blood glucose level when in fact, it is the volume and potassium depletion, acidosis and ketone accumulation that form the therapeutic goals. The main disadvantage of FRII is the risk of developing hypoglycaemia, hence the need to co-infuse glucose as blood glucose levels fall.

In addition to the FRII, the patient's usual long-acting basal insulin should be continued from the outset (e.g., Degludec (Tresiba), Glargine (Lantus/Toujeo), Detemir (Levemir) or Human Isophane Insulin (Humulin I, Insulatard). In a newly diagnosed patient with type 1 diabetes not previously on a basal insulin, basal insulin may be given s.c. once daily at a dose of 0.25 Units/Kg. (e.g., for a 70 kg individual = 18 units). This ensures the presence of insulin in case of interruption in the insulin infusion and allows a smooth transition to the usual subcutaneous insulin regimen of the patient when he or she is able to eat and drink after ketoacidosis has resolved. Early administration of long-acting insulin glargine has shown to protect from rebound hyperglycaemia[3] without increasing the risk of hypoglycaemia and the average time required for recovery is reduced.[4]

The JBDS-IP guidelines recommend considering reduction of the Fixed Rate Insulin Infusion to 0.05 U/kg/hour once glucose is <14 mmol/l.

- 10% Glucose (or equivalent) must be infused (typically starting at a rate of 125 mls/h and titrating to response) when the blood glucose level falls to below 14 mmol/l to prevent hypoglycaemia until the conditions for resolution of DKA are met (see below) and the FRII is stopped.
- Once resolution criteria are met, variable rate insulin infusions (together with infusion of 5% Glucose and K^+) may then be administered unless or until the patient is able to eat and drink and resume (or start, if newly diagnosed) subcutaneous insulin treatment.

Monitoring intervals

Monitoring intervals will depend on initial severity of the DKA, progress in response to treatment and available resources. However, as a guide, the following are useful starting points.

- Capillary glucose and ketones: Hourly until resolution
- Venous blood gas (HCO_3^- & K^+): 2h, 4h, 8h, 12h and 24h
- Plasma electrolytes: Every 4 hours until resolution

In addition to this, close monitoring of conscious level, clinical improvement and fluid sufficiency (especially recommended at 12 hours) should be carried out.

Criteria for resolution of DKA and transfer to subcutaneous insulin

By 24 hours DKA should have resolved in the majority of patients- in practice, many will have fully recovered within 6-12 hours of optimal treatment. Criteria for resolution of DKA and transfer to VRII (if still unable to eat and drink) or s.c. insulin (if eating and drinking) are as set out below.

Criteria for resolution of DKA

- Blood ketones <0.6 mmol/l AND
- Venous pH >7.3
- (Venous HCO_3^->15 mmol/l can be used but may be

delayed by hyper-chloraemic acidosis after the DKA has in fact fully resolved- see 'pitfalls' in Part 3 of this series.[5]

If the patient is eating and drinking, transfer to subcutaneous insulin given with food. Give the fast-acting insulin (e.g., Humulin S, Actrapid, Novorapid, Humalog, Apidra) with a meal and stop the i.v. insulin infusion 30-60 minutes thereafter to allow some time for the subcutaneous insulin to be absorbed.

If the patient was not previously known to have diabetes, a reasonable starting point for s.c. insulin is to give 0.4-0.5 units/kg/day of insulin in total, half of which long acting and half of which rapid acting insulin, the latter half shared between 3 meal time doses. For example, in a 70 kg individual, 14-18 units of long acting or 'basal' insulin once daily together with 5-6 units of rapid acting insulin with each meal.

These doses are only starting points and would be titrated to blood glucose readings in the fasting (for basal insulin) or 2-hour post-prandial (for soluble or rapid acting insulin) states bearing in mind that patients are usually insulin resistant when recovering from ketoacidosis so doses should be conservative to allow for this. It is important to avoid a hypoglycaemic episode shortly after starting insulin which may reduce their confidence in both insulin treatment and also in the prescribing physician.

If the patient has met the criteria for DKA resolution but is not yet eating and drinking, transfer to variable rate insulin infusion (VRII) until such time as they are.

SUMMARY

The priorities in DKA management following diagnosis are to assess severity, consider the need for involvement of critical care physicians if available and establish i.v. 0.9% NaCl rehydration with the addition of K^+ as described.

Ideally, a fixed rate insulin infusion should be used initially with consideration of insulin dose reduction (halving) and addition of 10% Glucose infusion when the blood glucose level has fallen to below 14 mmol/l. Regular clinical and laboratory monitoring, particularly of the rate of fall of blood ketones (hydroxybutyrate) and of serum [K^+] and glucose is essential to guide fluid and insulin infusion rates.

Once the criteria for resolution of DKA are met, the patient may be switched to s.c. insulin if eating or variable rate insulin infusion if not yet able to eat and drink.

Basal insulin should be continued (or started) where possible alongside infused insulin during the treatment of DKA.

Where facilities are not available for fixed rate insulin infusion, intermittent s.c. insulin injections may be used instead. Suitable regimes are described together with a number of pitfalls and special situations in DKA management in the third and final part of this series.[5] The first paper in this series is on Pathogenesis and Diagnosis.[6]

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Diabetic Ketoacidosis in Adults: Part 3. Special situations

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ABSTRACT

Prompt diagnosis and treatment of Diabetic Ketoacidosis (DKA) with the correct administration of intravenous (i.v.) fluids, fixed rate insulin infusion (FRII) and guideline-based K^+ replacement are essential for optimum outcomes. However, treatment guidelines may need to be adapted in special situations such as pregnancy, end stage renal disease or where resources, such as infusion pump equipment, may not be available. Children require treatment according to specific paediatric guidelines particularly to minimise the risk of cerebral oedema. Although DKA is a serious and complex medical emergency, skilled medical care can reduce mortality rates to below 1%.

Key words: Diabetic ketoacidosis, pitfalls, pregnancy, renal disease, infection, acute abdomen.

INTRODUCTION

In this, the third of a three-part review of Diabetic Ketoacidosis (DKA) in adults, we focus on pitfalls in the management of DKA and on special situations where the usual management outlined in Part 2[1] may need to be adapted. Pitfalls refer to situations where clinicians managing DKA may be misled into taking incorrect decisions unless they are familiar with the issues. They also include some areas of controversy such as in the indications for the use of sodium bicarbonate infusions in patients with very severe acidosis.

PITFALLS IN THE MANAGEMENT OF DKA

Hyponatraemia

It is common for DKA patients to be initially hyponatraemic. This is due to hypertonicity driving water from the intracellular to extracellular fluid compartments which dilutes the extracellular concentration of sodium ($[Na^+]$). With improvement of hyperglycaemia, $[Na^+]$ will rise. The following formula may be used to 'correct' the $[Na^+]$ concentration for hyperglycaemia.[2]

Corrected Na^+ = Measured Na^+ + 2.4 {(glucose (mmol/L) -5.5)/5.5}

In effect, this predicts the level to which the measured $[Na^+]$ will rise after correction of hyperglycaemia. If this prediction suggests that the $[Na^+]$ may rise to a very high level (say >155-160 mmol/l), then cautious use of half-normal (0.45%) saline rather than 0.9% Saline may be considered during the period of volume restoration. It is worth pointing out that although there is considerable controversy over the use of half-normal (0.45%) saline solutions in the management of DKA, a patient whose blood glucose level has fallen to below 14 mmol/l who is treated according to the protocol recommended in the second part of this series may be receiving simultaneous infusions of both normal saline and 10% glucose which, once the glucose has been disposed of by metabolism, storage or excretion, may in effect amount to an infusion of hypotonic saline.

High Total White Cell Count

A raised total white blood cell count may be observed in DKA even in the absence of infection. This is thought to be a consequence of catecholamine secretion in response to hypovolaemia.

Infection

Infection is a common cause for DKA. It needs to be considered and treated if present but not all patients presenting with DKA have an infection requiring treatment. DKA can sometimes be misdiagnosed as infection (particularly malaria or an 'acute abdomen'). Occasionally a severe infection such as meningitis may precipitate, but then be masked by, DKA as both conditions may cause drowsiness, low blood pressure, acidosis and renal failure. Until recently, there has been no absolute failsafe method for detecting the presence of a serious infection requiring treatment during DKA other than by taking a careful history and appropriate samples e.g. blood or urine cultures, being aware of the risk of masked infection and having a low threshold for reassessment particularly where progress in resolution of DKA and/or clinical improvement is not as expected. However, there is some evidence that procalcitonin measurement, where available, particularly in association with documentation of fever >38^{0}C may be helpful in both ruling in, and in excluding, serious bacterial infection in patients with DKA.[3]

Raised amylase

Raised serum amylase is common in DKA, even in the absence of pancreatitis. Abdominal pain is also common due to an acute ketone-induced gastroparesis. However, DKA may also be precipitated by and mask an acute abdomen so careful clinical observation is required particularly where recovery is not progressing as rapidly as expected.

Ketones

Measurement of 3-β-OHB through capillary blood samples provides the best indicator of ketone body levels. Urinary ketone tests rely on nitroprusside reactions which have a high affinity towards acetoacetate and acetone but not 3-β-OHB. This can provide false reassurance in the initial stages of DKA. During DKA, low insulin levels and high counter-regulatory hormones increase 3-β-OHB: acetoacetate ratio to 10:1. With successful treatment, 3-β-OHB levels decrease whilst acetoacetate levels increase. Measuring urinary acetoacetate levels as the patient improves may therefore over-estimate the degree of ketosis which sometimes unnecessarily prolongs treatment intensity.[4]

Bicarbonate use

The acidaemia in patients with DKA is caused mainly by ketoacids but there may be additional contributions from renal impairment and lactic acidosis. The use of bicarbonate is generally unnecessary and potentially harmful as it could reduce serum [K^+], interfere with tissue oxygen delivery through effects on the oxy-haemoglobin dissociation curve, prevent hyperventilation and it may cause a paradoxical worsening of central nervous system acidosis because CO_2 diffuses through the blood brain barrier faster than the infused bicarbonate. At a pH of 6.9-7.1, administering bicarbonate does not improve recovery outcomes and could cause harm. At a pH of <6.9, these are no good data but many would fear the imminent development of cardiovascular collapse and would give a sodium bicarbonate infusion to correct the pH to >7.0. [5,6]

Hyperchloraemic metabolic acidosis

During treatment of DKA, large volumes of administered Normal Saline frequently result in a hyperchloremic metabolic acidosis as the kidneys preferentially excrete ketones over chloride anions. Up to 90% of patients remain hyperchloremic between 8 to 20 hours.[7] In general, this form of acidosis is rarely a major clinical issue and will resolve spontaneously within hours to a day or two. It does not require specific treatment (beyond stopping any unnecessary Normal Saline infusions after resolution of ketoacidosis and restoration of oral fluid intake) and does not indicate delayed resolution of DKA. The mistake often made is to assume that the DKA is not resolving even though ketones (and lactate) are low because of the persisting low bicarbonate concentration and this is a particular risk where ketone measurement is not available and treatment is being monitored with HCO_3^- measurement.

Cerebral Oedema

The development of cerebral oedema is very rare in adults treated according to these guidelines. If there is deterioration of consciousness despite improved metabolic state, especially accompanied by signs of raised intracranial pressure such as bradycardia and hypertension, consider brain imaging (ideally by MRI if available, otherwise CT) to look for cerebral oedema or another CNS insult that could depress conscious level (e.g., stroke, venous sinus thrombosis, cerebral haemorrhage). Management of cerebral oedema will require specialist support and generally includes mannitol, hypertonic saline and dexamethasone. Although rare in adults, cerebral oedema is a real risk in children who should be treated according to specific paediatric guidelines (see the section on 'Children and young people' below which includes an approach to treatment of suspected or proven cerebral oedema).

Glucometer reading 'Hi'

Bedside glucometers using test strips will usually register a result of 'Hi' when sampled capillary blood glucose levels exceed a maximum limit for the test, typically around 28-33 mmol/l depending on brand. It is important that a laboratory (or blood gas analyser) blood glucose level is then obtained so that the actual fall in blood glucose levels may be appreciated, and insulin infusion rate adjusted if appropriate.

Distinguishing Hyperosmolar Hyperglycaemic State (HHS) from DKA

Distinguishing these two diabetes emergencies is usually straightforward on the basis of the presence or absence of ketoacidosis (favouring DKA) and hyperosmolality (usually >320 mOsm/Kg) without significant acidosis favouring HHS. HHS is typically encountered in older individuals with type 2 diabetes whose blood glucose levels are typically ≥30 mmol/L and who have profound hypovolaemia. However, the distinction may be less clear cut particularly as there is no universally accepted biochemical criteria for the diagnosis of HHS which remains a clinical diagnosis supported by characteristic laboratory findings. Acidosis (pH<7.3, HCO_3^-<15 mmol/l) may accompany HHS but when it does so, is typically due to the accumulation of lactate ions (lactic acidosis) and/or due to associated Acute Kidney Injury rather than primarily due to the accumulation of ketones. In this situation, blood hydroxybutyrate concentrations will be well below the level used as a diagnostic criterion for DKA (i.e., below 3.0 mmol/l) and the treatment approach should be directed towards management of HHS rather than DKA. In some cases of HHS with marked hyperosmolality (>320 mOsm/Kg) and profound dehydration, there will be significant acidosis (pH<7.3, HCO_3^- <15 mmol/l) associated with ketone accumulation to levels more typically associated with DKA (i.e., blood hydroxybutyrate > 3.0 mmol/l or ketonuria ≥2+). In this situation, the diagnosis is best considered to be a mixed form of HHS/DKA and treatment would be as for DKA with possible adaptation for the age, co-morbidity, precipitant, degree of hypovolaemia and degree of hyperosmolality present. Expert involvement may be helpful in such mixed cases although the DKA pathway, particularly as regards insulin infusion, should be followed initially.

DKA IN SPECIAL SITUATIONS

Lack of availability of an insulin infusion pump

In circumstances of limited resources, it may not be possible to use fixed rate insulin infusions with shortage of infusion pumps or trained nursing staff. In this situation, a loading dose of soluble insulin intramuscularly (10-20 units) followed by 5 units hourly can be given.[2]

Alternatively, if available, rapid acting insulin analogues may be administered subcutaneously on an hourly basis in the management of mild and moderate DKA. This has shown similar outcomes to FRII when combined with fluid rehydration.[8] A suitable regime is to give an initial s.c. insulin injection of 0.3 units/kg body weight, followed by 0.1 units/ kg/ hour s.c. until blood glucose reaches 13.8 mmol/l at which time the insulin is reduced to 0.05 units/ kg/ hour, and the IV fluids changed to Dextrose (D) 5% in 0.45% saline to maintain blood glucose at about 11.1 mmol/l until resolution of DKA. Alternatively, patients can be managed with 2-hourly s.c. injections so that they receive an initial dose of 0.3 units/ kg followed by 0.2 units/kg 1 h later and then again every 2 hours until blood glucose reaches 13.8 mmol/l. At that time, the insulin dose is reduced to 0.1 units/kg every 2 hours, and the IV fluids changed to D5% in 0.45 saline to keep blood glucose at about 11.1 mmol/l until resolution of DKA.[8]

DKA in end stage renal disease or dialysis

This is an uncommon situation since insulin clearance by the kidneys is reduced in end stage renal disease (ESRD) and failure to generate an osmotic diuresis in response to hyperglycaemia further reduces the risk of DKA. In fact, hyperglycaemia may paradoxically lead to fluid volume expansion through increased thirst and hence fluid intake with absence of the osmotic diuresis that leads to dehydration in non-renal patients.[9] Fluids should be administered with great caution in apparently hypovolemic patients with 250ml boluses (0.9% NaCl or Dextrose 10% as appropriate) in this situation.

The insulin infusion rate in ESRD is generally similar to that in patients with normal renal function but more caution is needed as the risk of hypoglycaemia is greater due to reduced insulin clearance. Once blood glucose is < 14 mmol/l, strongly consider reducing the rate of insulin infusion to 0.05 units/kg/hour. More concentrated dextrose solutions may be needed to avoid overload.[9] Potassium excretion is impaired in renal failure and together with the lack of osmotic diuresis; there is little or no potassium loss through the kidneys. On the contrary, end stage renal failure patients with severe acidosis may develop hyperkalaemia and may require dialysis. Potassium should be added to infusion fluids with great caution, if at all, in patients with ESRD.

Euglycaemic DKA with SGLT2 inhibitors

Euglycaemic DKA is a rare complication of Sodium-glucose Cotransporter-2 (SGLT2) inhibitors (e.g., empagliflozin, canagliflozin, dapagliflozin). Patients may develop DKA despite having a blood glucose level

of <11mmol/L. SGLT-2 inhibitors are linked to a small increased risk of diabetic ketoacidosis in individuals with both type 1 and type 2 diabetes. It is important to note that urine testing may be unreliable in detecting the development of ketoacidosis in this situation as SGLT2 inhibitors may impair urinary ketone excretion so blood ketone testing should be used in patients taking these drugs in whom DKA is suspected. As always, prevention is key and patients at risk of ketoacidosis (e.g., those with type 1 diabetes, ketosis prone type 2 diabetes or very poorly controlled type 2 diabetes who would best initially be treated with insulin) should not generally be prescribed SGLT2 inhibitors. Even in those not considered to be at high risk, ketoacidosis secondary to SGLT2 inhibition can develop in dehydration, stress, starvation, excessive alcohol consumption, acute medical illness or other catabolic states which shift metabolism to fat dependence and these agents should be stopped during such intercurrent illness.[10]

If DKA is confirmed in this situation, the SGLT-2 inhibitor should be stopped immediately and ketoacidosis treated conventionally (noting that glucose infusion may be required from the outset). Use of a variable rate insulin infusion (VRII) rather than a fixed rate insulin infusion may be needed to avoid hypoglycaemia and hypokalaemia.[10]

Pregnancy

DKA in pregnancy may occur in women with type 1 diabetes, type 2 diabetes or gestational diabetes and presents particular risks to the woman and her unborn child and specific challenges in its management.

Prevention is vital and most importantly includes the provision of specialist services to diagnose and treat dysglycaemia during pregnancy in women with both pre-existing and gestational diabetes mellitus. Care should be taken to titrate insulin in pregnant women receiving corticosteroids to promote foetal lung maturation in situations of anticipated pre-term labour (doses typically need to rise by 25-40% or more) and to avoid the use of sympathomimetic tocolytic agents (e.g., ritodrine, terbutaline) where possible (or for carefully titrated insulin infusion where this cannot be avoided).

In normal pregnancy, there is a state of respiratory alkalosis with a compensatory reduction in bicarbonate concentration which reduces the buffering capacity of the blood thereby reducing the threshold for the development of metabolic acidosis. There is an accompanying insulin resistance in pregnancy that may further predispose to DKA development. Aside from the precipitants of DKA for adults in general, specific factors during pregnancy include protracted vomiting (hyperemesis gravidarum) and the administration of specific drugs with counter-regulatory hormone type effects such as steroids for fetal lung maturation or tocolytic sympathomimetic agents. In women at risk of DKA, these should be avoided where suitable alternatives can be used instead. Euglycaemic DKA (DKA with presenting blood glucose levels <11 mmol/l) is more common in pregnancy, due to the glucose disposal of the feto-placental unit, increased renal glucose losses during pregnancy, increased maternal glucose utilization and increased volume of distribution of glucose. These effects will be augmented where DKA is precipitated by excessive vomiting or starvation which will dehydrate and deplete hepatic glycogen stores respectively. Euglycaemic DKA requires infusion of glucose from the outset in order to permit insulin infusion (either as fixed or variable rate) in sufficient dose to suppress ketogenesis. Consideration should be given to administering thiamine iv prior to glucose particularly where starvation/hyperemesis has been longstanding to prevent the development of Wernicke's encephalopathy.

DKA in pregnancy may cause maternal complications such as acute kidney injury, adult respiratory distress syndrome, cerebral oedema or death. Fetal mortality has been estimated as being up to 36% but permanent fetal morbidity may also occur due to hypoxia, reduction in cerebral glucose uptake or utilization, exposure to a period of reduced uteroplacental function, electrolyte disturbances and maternal or fetal cardiac dysrhythmia.

A decision to deliver should be individualized and multidisciplinary but in general, the focus will be on restoration of maternal cardiovascular and metabolic stability rather than delivery unless consideration of maternal wellbeing, gestational age and fetal condition (e.g., fetal heart monitoring) suggest a potential benefit from delivery.[11]

Children and young people under 18 years of age

The management of children with DKA differs in a number of important ways from that of adults and is not covered here in detail. Children are more vulnerable to the development of cerebral oedema during DKA treatment in particular and management is designed to minimize the risk of this whilst ensuring prompt resolution of DKA. The reader is referred to comprehensive guidelines on the management of DKA in children for further information.[12]

SUMMARY AND CONCLUSIONS

Prompt diagnosis and treatment of DKA with the correct rates and volumes of i.v. fluids together with fixed rate insulin infusion and guideline-based K+ replacement form the mainstay of treatment.

Treatment protocols may need to be adapted in special situations such as pregnancy, end stage renal disease or where resources such as infusion pump equipment may not be readily available and children require treatment

according to dedicated paediatric guidelines particularly to minimise the risk of cerebral oedema.

Although DKA is a serious and complex medical emergency, skilled medical care can reduce mortality rates to below 1%.

The other papers in this series are on Pathogenesis and Diagnosis[13] and Management.[1]

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Gunshot injury to the face: a case report

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ABSTRACT

Treatment of gunshot wounds in the maxillofacial region is complex. Current literature supports immediate treatment. Wounds vary widely. The nature of the injuries must be carefully assessed using the Kanzanjian and Converse's principles of plastic surgery as guidance, but always adapting to specific needs. Management is dependent upon the type of weapon, the bullet's characteristics, kinetic energy, place of impact, as well as the patient's general health status. A case is presented of a gunshot injury to the face with a review of the literature.

Key words: Gunshot injuries, extent of damage, management, antibiotics

INTRODUCTION

Gunshot injuries (GSIs) cause significant morbidity and mortality which may be instantaneous. Those involving the head and neck can be devastating especially when they affect vital structures. They present challenging surgical problems. Similar injuries occur in both military and civilian settings. Some geopolitical conflict areas in Africa have GSI as the second most common cause of death. There is an increasing incidence of GSIs worldwide, particularly those involving the face.[1-3]

The extent of damage is dependent on a number of factors including the magnitude of energy transferred, distance travelled by the missile, type of bullet, and the anatomical structures encountered. High-energy transfer gunshots fired at close range inflict the most damage. The extent of tissue damage depends on internal lacerations, compression of tissues and the temporary cavitations along the projectile path. Secondary injuries are also possible following impact with bone, which sets other missiles (bone fragments) into motion on their own paths, causing additional injury.[4-6]

The face and neck region is packed with vital structures in a relatively small volume of space. Even the smallest of movements by a penetrating missile may injure a major vein, artery and main nerve trunk simultaneously. The leading cause of death in penetrating neck trauma is major vascular injury causing uncontrollable haemorrhage. Treatment is challenging especially when the bullet or its fragments are lodged within the vicinity of vital structures. Most bullets or their fragments are highly contaminated with serious consequences especially when there is associated tissue loss. Tissue damage is both direct and as a result of energy dissipated from the inherent kinetic energy transferred to the tissues.[7, 8]

Missile injuries are described as penetrating (25%), perforating (38%) and avulsing (37%). Some gunshot wounds are through-and-through injuries, but in many patients the bullet enters with no visible exit wound. In such situations, the bullet's trajectory and final destination may be unpredictable. There may be an unknown extent of bony damage and consequences and a threat to the patient's airway from haematoma or oedema.[9, 10]

The ideal time and method of treatment remains debatable. Several surgeons maintain that because of the mechanism of injury, early aggressive primary reconstruction might not be ideal. They opt for initial conservative management

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followed by a staged secondary reconstruction to obtain satisfactory functional and aesthetic results. Alternatively, others advocate early management of facial deformity. The steps taken include: securing the airway, controlling haemorrhage, identifying other injuries and preventing additional injury, and repair and/or reconstruction of the traumatic facial deformities.[9-11]

CASE PRESENTATION

A 47-year-old African-Namibian man of black ethnicity was referred from a regional hospital to the Emergency Department of Oshakati intermediate hospital, following a GSI to his face. The injury was the result of a 'stray' bullet, shot from a gun at a distance less than one metre, while he was manipulating the gun. He experienced intense pain over the right side of his face, and bleeding from the wound. The amount of blood loss was difficult to ascertain. He was rushed to the local regional hospital after bystanders had applied improvised bleeding-control measures. Surgical wound toilet was done without extension of the wound. Augmentin and metronidazole intravenously and anti-tetanus vaccines were administered, and the wound was dressed but not sutured, leaving a large open wound.

He was referred to our intermediate hospital three days later. He had right mandibular pain, with difficulty with opening his mouth and chewing. He was fully conscious and haemodynamically stable. There was a laceration on the maxillomandibular area (8 cm in length) with features of infected granulation tissue (Figure 1).

The right side of his cheek region was swollen and tender, showing multiple pieces of bone and necrotic tissue. The mandible was fractured. There was no active bleeding, subcutaneous (surgical) emphysema, or dyspnoea and stridor. He had total loss of cutaneous sensation over the distribution of the right mandibular branches of the trigeminal nerve. The ophthalmic division of the trigeminal nerve was intact.

Figure 1. Appearance of the wound three days after injury.

A CT scan without contrast of the facial bones was done which revealed the presence of a comminuted mandibular fracture. The bullet was not seen in the CT scan images. Apparently, part of it was fragmented and the rest and was removed at the regional local hospital. (Figure 2).

We proceeded with exploration of the bullet entry wound. A comminuted fracture of the body of the mandible was confirmed. Debridement was done and the wound was dressed. The patient was started on antibiotics with clindamycin 600mg IV tds and metronidazole 500mg IV tds, plus tramadol 100 mg IV bd for the pain.

Consent was signed for surgery under general anaesthesia. The fracture was accessed through a Risdon approach

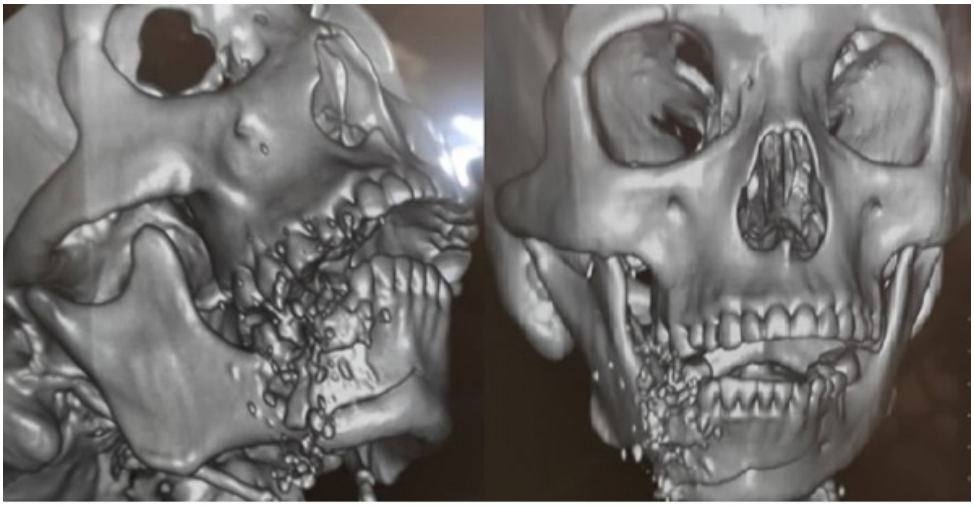

Figure 2. CT scan without contrast with 3D reconstruction of facial bones.

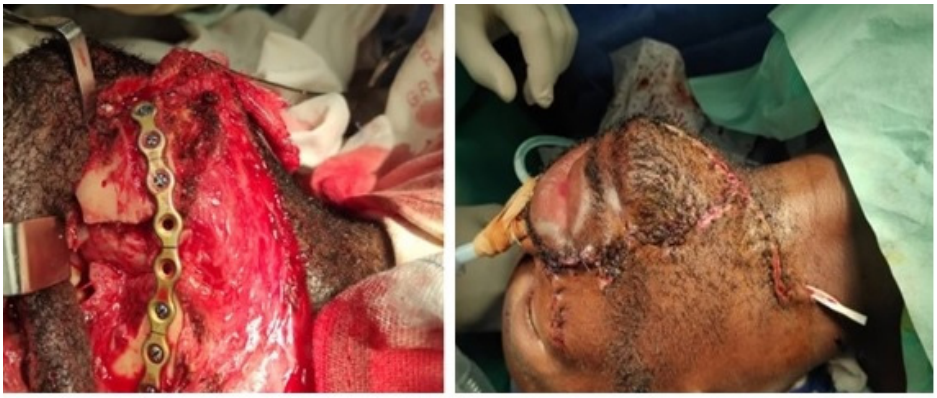

Figure 3. Mandible reconstruction and advance facial flap

on the neck. Loose pieces of bone without periosteum were removed from the wound and foreign bodies, which consisted of small pieces of bullet, which were not clear on the CT scan.

Intermaxillary fixation was performed to keep the bone in position and provide adequate occlusion. To treat the fractures a 2.4 mm reconstruction plate was placed on the fractures to reconstruct the mandibular body with a 2 mm titanium plate on the parasymphysis, fixed in place with screws 2mm and 1.5 mm respectively. Closure by layers of the periosteum, mucosa and the cheek muscles was done using vicryl 3-0. To reconstruct the facial skin defect, a flap was advanced from the parotid and neck region. Skin suture was done with nylon 4=0. (Figure 3).

The patient was kept in the hospital ward for two weeks after surgery to ensure proper dressing and to monitor the wound healing. Feeding was carried out using a nasogastric catheter until the wound was mostly healed and closed to avoid communication between the mouth and neck structures. After two weeks the patient was discharged and the nasogastric catheter was removed.

A review appointment was arranged every week. Unfortunately, a fistula developed on the skin close to the mandible inferior border after a month, due to suture reaction and it was removed with local anaesthesia. A piece of suture was found in relation with it. The patient progressed well, having an acceptable aesthetic and functional outcome. A CT scan was repeated to check the position of the plate and the bone healing process. (Figure 4).

DISCUSSION

Gunshot injuries of the face can be devastating often resulting in comminuted fractures of the affected bones and facial disfigurement. Many forms of treatment for this condition have been described. The closed reduction with intermaxillary fixation of the fractures has the advantage of preserving the periosteum with the maintenance of the blood supply and lower infection rate. However, the treatment time is longer, nutrition is restricted and hygiene is difficult.[8, 9]

Regarding the treatment by means of open reduction and internal fixation: it is extremely important to understand that the stage of wound debridement and removal of any and all tissues that appear to be unviable, be it part of soft or hard tissues, can dictate the success of treatment and risk of infection. In our case the debridement procedure was performed, making prognosis more favourable.[9, 10]

We used 2.0mm plates and screws to plate viable small fragments, and 2.4mm to treat large fractured fragments so to support all muscle-occlusal loads during mastication until bone consolidation. This type of fixation presents good functional results.[10, 11]

It is of fundamental importance to understand that because this trauma causes avulsion of soft and bone tissues leading to a high infection risk. Pre-operative antibiotics for at least seven days is advisable to combat infection and subsequent osteomyelitis. The antibiotics of first choice should include penicillins with beta-lactams, such as ampicillin + sulbactam or amoxicillin + potassium clavulanate or clindamycin

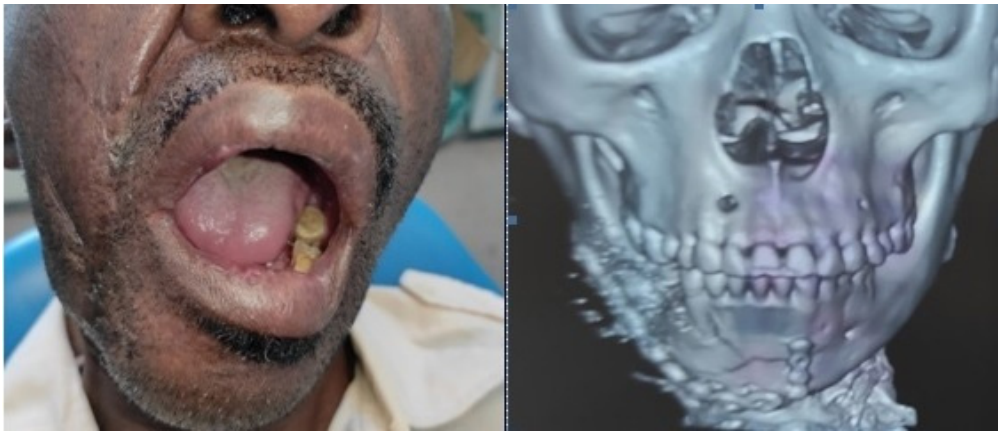

Figure 4. Patient appearance two months after surgery and follow up CT scan

plus metronidazole. In the case presented there was loss of bone with exposure of the intra-oral wound and because the wound was caused by a firearm, the antibiotic therapy protocol was carefully followed.[12]

Post-operative management is crucial bearing in mind the complexity of the surgical procedure and extensive tissue manipulation. There is a risk of generating severe oedema and later wound dehiscence and consequent infections even with antibiotic cover. Thus, supervision over the first two weeks postoperatively is essential, after which muscle physiotherapy is initiated through spatula therapy. The latter consists of exercises opening the mouth using a wooden tongue depressor and bi-digital mouth opening. This is essential for gaining amplitude in the mouth opening and avoiding excessive muscle fibrosis.

CONCLUSION

Facial fractures caused by firearms are of great complexity and an enormous challenge to maxillofacial surgeons in terms of initial management and reconstruction. Usually there is extensive damage to, and loss of, hard and soft tissue, the wounds are contaminated.

Within the world literature, there remains controversy concerning the approach to management. For the patient described in this report we adopted early surgical management with careful in-patient and out-patient monitoring. This resulted in a good functional outcome and aesthetic appearance.

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What do teachers do? A framework for improving teaching in healthcare education

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Much of sub-Saharan Africa needs increased healthcare capacity. South Sudan is no exception, and for some years the government has prioritised this as a policy goal. The challenges to the provision of healthcare in South Sudan have been noted previously.[1] More than 80% of medical services are provided by international organisations.[2] In the medium and long term, South Sudan needs to be able to produce and retain healthcare practitioners: doctors, nurses, midwives, and those associated practitioners whose skills are needed to increase the country's capacity to deliver healthcare.

At the operational level, progress depends upon the further development of institutional resources, and organisational effectiveness. This requires international support, and the continuing efforts of institutions such as Juba University Medical School, the College of Physicians and Surgeons, and the College of Nursing and Midwifery. What can we do as individuals to support this goal?

We can strive to improve our healthcare teaching.

Healthcare needs safe, competent practitioners who learn through teaching, mentoring, and supervision. So, in classroom and clinic, the healthcare teacher plays a vital role in helping the next generation of clinical practitioners to develop their knowledge and skills. In this context, learning is a process of expanding ability across eight trajectories,[3] during which it is important to practice, safely, in those areas of work where abilities are challenged.[4] Figure 1 represents this diagrammatically. It is important that the teacher knows where the boundaries lie for each learner, and structures their work and supervision to enable both learning, and safe practice.

'Healthcare practitioners' include all the various healthcare roles that the people of South Sudan require. By 'teacher', I mean teachers in university and college settings, but also supervisors and colleagues in the workplace, for it is in the workplace that people learn their healthcare practice - from experience, and from each other. 'Learners', include students, doctors, dentists, nurses, midwives, and associated healthcare practitioners, in undergraduate and postgraduate settings, and throughout their working lives.

Healthcare teachers often work in difficult circumstances. To be effective, they have to realise they are indeed teachers, as well as clinicians, and they have to commit themselves to the improvement of their teaching. This is perhaps more challenging in the workplace, than it is in the university or college. Given a sense of identity as a teacher, and motivation to improve, teachers can decide what to do by thinking about their own practice, seeking ideas for doing things differently, and by seeking a clear understanding of how people learn.

To support this process, we intend to publish, over several issues of the South Sudan Medical Journal, a series of educational 'How to…' papers. These are offered as guides to self-directed improvement by healthcare teachers. They will include practical tips, and educational ideas. They will also address scholarship in healthcare education, by giving guidance on research methods and evaluation in education.

The aim is to target the essentials for the practising healthcare teacher, and so we will focus on supporting the educational activities listed in Table 1. Not all healthcare teachers will do all of these, but most will do some of them.

Teaching large and small groups, involves using a variety of interactive techniques,

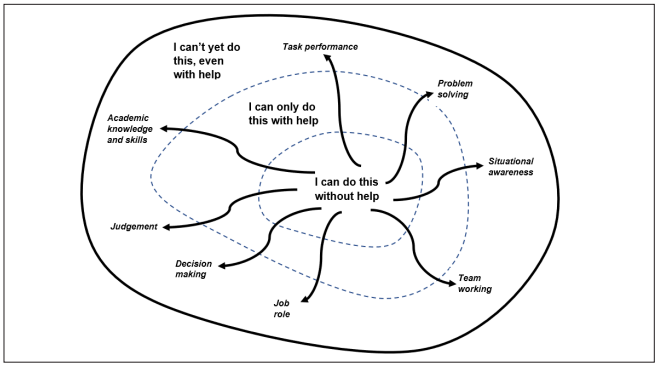

Figure 1. The expansion of skills and knowledge of healthcare practitioners

The aim is to expand the size of the inner core, and reduce the size of the outer sphere. The role of the teacher involves locating the learner on this diagram, and providing appropriate help.

audio-visual aids and learning resources suitable to the lecture theatre, classroom, and the size of the group. Teaching in a clinical setting has to fit into the demands of the healthcare service – the needs of both patients and learners have to be accommodated. Knowing the learner's needs, informing, demonstrating, questioning and providing feedback, are important. An associated responsibility is to support in-service learning. Here, considering a junior colleague's learning needs in the allocation, structuring and supervision of work are important, as is the provision of normative feedback, and having appropriate expectations of performance.[5]

Assessment might be formative, directed towards helping the learner understand their progress, strengths and weaknesses, and what they can do to improve. This is part of everyday teaching, and is often provided through feedback and discussion. Summative assessment is used to confirm that a learner has achieved the required standard on completing a course or programme.

Teachers often have to respond to concerns about performance. In the university setting this will usually focus on academic performance. In the workplace, difficulties might touch upon a range of issues (see figure 1). There may be a need to provide for patient safety, and for adequate supervision, coaching, or mentoring. Reviewing learners' progress is an appraisal, designed to help learners take stock of, and manage, their overall progress against goals. If progress is not meeting requirements, then the teacher will need to know their options, and how to proceed in the best interest of the learner, and the healthcare profession.

Finally, teachers have a responsibility for the design, evaluation, and improvement of educational practice. For some, this might involve carrying out research in education, to improve local practice and to contribute to scholarship. Others may be in a position to evaluate and improve their own teaching, a taught course, or a clinical workplace, to better support learning.

Table 1. What do healthcare teachers do?

1.	Teach in a range of settings
1.1	Teach large groups.
1.2	Teach small groups.
1.3	Teach in a clinical setting.
1.4	Support in-service learning.
2.	Assess performance
2.1	Assess performance using formative methods.
2.2	Assess performance using summative methods.
3.	Support progress
3.1	Respond to concerns about performance.
3.2	Review learners' progress against educational goals.
4.	Improve healthcare education
4.1	Evaluate and improve teaching practice.
4.2	Evaluate and improve a taught course.
4.3	Evaluate and improve the clinical workplace to support learning.
4.4	Carry out research in healthcare education.
5.	Develop programmes of healthcare education
5.1	Design a new course.

That, then, is the framework for improving teaching in healthcare education. We will publish practical guidance in support of these objectives, over the coming months. One way of using these articles will be to identify your own improvement priorities as a teacher, and to use the information in the articles to help develop your own learning plan. Meeting with colleagues, to form a learning group, would allow discussion of teaching experiences, educational ideas, and improvement opportunities. We hope that a wide range of teachers will find the articles useful in supporting their self-directed learning, in South Sudan, but also those in similar roles elsewhere.

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