Perforation of Bladder by a Lippes Loop, Resulting in Calculus Formation

by

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An Intra Uterine Contraceptive Device (IUCD) which perforate the uterus anteriorly in the region of the upper cervical canal or just above the internal os can continue forwards to perforate the bladder as well, rarely. Probably due to its rarity, the condition is usually not diagnosed prior to intervention.

In reviewing the literature over the l ast decade, Zakin (I984) found 8 cases reported out of which in only two cases had the diagnosis been made prior to the therapeutic procedure.

Case Report

Mrs. HMD a 31 year old gravida 4, para 3 was referred to us on 28th October 1984 for removal of an IUCD. She had presented elsewhere with a history of dysuria, lower abdominal pain and haematuria of 3 months duration, and been treated as a case of urinary tract infection, while a plain X'Ray of abdomen antero posterior (AP) view had shown a Lippes Loop.

A Lippes Loop had been inserted 9 years earlier in a peripheral clinic. She had not experienced any undue pain or bleeding per vaginum nor had any urinary symptoms then or later. She could not recall having felt the threads of the device at all. About one year later, she had an abortion after a period of amenorrhoea of about 12 weeks. An evacuation of uterus had not been done. Two years later she had had a normal delivery at term and the child was six years old now.

Another Lippers Loop was inserted by us and a plain X'Ray of abdomen and peivis AP view obtained. This revealed two lippes loops. One with normal contuours lying longitudinally in the centre of the pelvis and another with its convolutions splayed out, lying transversely above the first (Fig. 1). The first was obviously the device introduced by us and hence intra uterine. The second was the 'missing device' which was thought to have perforated the uterus and transmigrated into the peritoneal cavity.

A laparotomy was done under general anaesthesia to remove the device and carry out a sterilization, as requested by the patient. At surgery, a careful search of the peritoneal cavity failed to reveal the device.

On examiuation of the bladder, the device was palpable through its wall and a hard mass was palpable within its lumen. A cystotomy was carried out and a Lippes Loop with calculus formation around it was removed (Fig. 2). The device was partially attached to the bladder wall.

Sterilization was carried out using Pomeroy's technique. Post operatively an indwelling catheter drained the bladder continuously for 10 days Ampicillin was given prophylactically. On discharge her urine was free of infection and there were no complications.

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FIGURE I

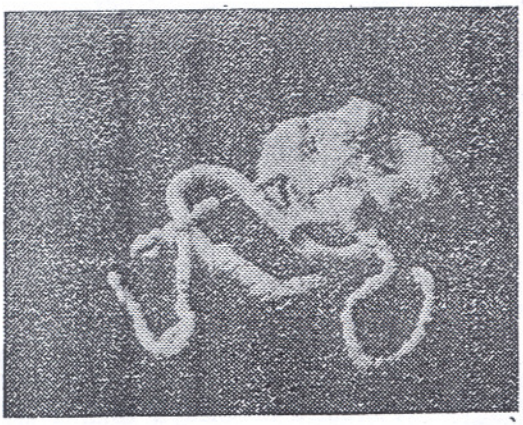


FIGURE II

Discussion

Uterine perforations by an IUCD, are most often asymptomatic except for a history of 'missing threads.' Bladder perforations however are characterised by urinary symptoms of recurrent cystitis. Episodes of dysuria, frequency, haematuria and suprapubic pain are common. Hence, although attacks of cystitis are relatively common in females, repeated attacks starting after the insertion of an IUCD should arouse suspicion of bladder perforation (Zakin 1984) Perforation of the uterus usually occurs during the initial insertion. However the perforation of the bladder may be only partial, at that time.

An IUCD after partial perforation could gradually continue on its forward path aided by the uterus contrcting and trying to expel it, and come to lie completely within the lumen of the bladder subsequently.

Our patient was asymptomatic for 9 years. It is possible that initially she had partial perforation which could account for the lack of urinary symptoms. Her 3rd pregnancy may have terminated in an abortion due to a significant portion of the device being present within the uterine cavity at the time. Howevr it is well-known that even with an intact IUCD pregnancies can continue upto term. Hence, exactly how long the device had been intravesical is not known. For a calculus to form, the device should have been intravesical sufficiently long for it to act as a nidus for the deposition of radiopaque urinary salts around it.

Sanowala et al ('974) reported a similar case where urinary symptoms and suprapubic pain appeared more than 6 years after insertion of a Lippes Loop.

The technique of inserting another IUCD and obtaining an X'Ray is quite useful in the diagnosis of a transmigrated device. Visualisation of a calculus to which the extrauterine device is attached points to a bladder perforation, (Neutz et al 1978, Sanowala et al 1974). In our patient, although the faint opacity of the calculus s visible on the X'Ray it was overlooked priorr to surgery.

A lateral film is essential in all cases. Because of undue reliance on the AP view only, many mistakes occur. The Bladder being a central organ in the mid pelvis, intravesical devices have been erroneously diagnosed as being intrauterine (Zakin 1984). In a lateral film the IUCD may be sufficenntly anterior to suspect a bladder perforation. Rarely however, it could also be in the anterior cul de sac, the utero vesicle fold of peritoneum, or in the retropubic space of Retzius (Zakin et al 1981 a) .

Intravenous urography or retrograde cystography are useful especially in partial perforations of the bladder. Proper timing of the excretory film in the former and proper dilution of the radiopaque solution in the latter are necessary for optional radiopacity of the medium and the device. (Zakin et al 1981 b, Zakin 1984).

Uterine perforation by IUCD are classified according to the degree of perforation (Zakin et al 1981 a). Ultrasonagraphy can be useful in the diagnosis. However in contrast to both diagnostic and therapeutic advantages in type 2 and 3 uterine perforations, Laparoscopy is of far less value in type 4 involving the bladder. After ultrasonagraphy and or radiological evaluation cystoscopy becomes mandatory (Zakin 1984).

Removal of the device would depend on its exact location and the size of an associated calculus if present.

If the device is lying free in the bladder, without a large calculus it could be removed at the same time that it is demonstrated, using the cystoscope. (Zakin et al 1981 b, Zakin 1984).

Although vaginal cystotomy has been reported suprapubic cystotomy is the procedure of choice for removing an IUCD perforating into and adherent to the bladder wall. (Neutsz et al 1978, Saronwala et al 1974, Zakin et al 1981 b, Zakin 1984).

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MPSIA - A STUDY OF ITS CURENCE AND MATERNAL SEQUELAE

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More than two hundred years ago, Alexander Hamiiton described eclampsia as 'a disease which is always attended with the utmost hazard, and frequently kills the woman like a fit of apoplexy'.

Although a rare disease in the developed countries where good antenatal care is available, it still remains an important cause of foetal and maternal morbidity and mortality, in the developing world. Eclampsia does not come as a bolt of lighting as the term implies but is often insidious in onset in the form of pre eclampsia. Good antenatal care with early detection and treatment of pre eclampsia can prevent this serious condition which not only carries a high perinatal mortaltiy, but is also a major threat to the mothers life.

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Materials and Methods

The patients in this study are from admissions to the University Obstetrics and Gynaecology unit of the General Hospital Galle, Sri Lanka during the two year period October 1st 1980 to September 30th 1982. The criteria for diagnosis of eclampsia were generalised convulsions, hypretension gross oedema and albuminuria in pregnancy. All other convulsive states such as epilepsy, cerebro vascular accidents and neurological lesions were excluded.

Results

(*a*) *Incidence*

Out of a total of 3441 deliveries there were 13 eclamptics giving an incidence of 0.378% of the total number who delivered only 32% were premigravidae. Seven out of the 13 eclamptics were primigravidae, giving an incipence of 0.636% compared with the 0.256% incidence in the multigravidae.

TABLE : Distiibution of cases according to age and parity

Age / Gravidity	< 20 Yrs.	20-24 Yrs.	25-29 Yrs.	30-35 Yrs.	Total	%	Average Age	Incidence
Primi	2	3	1	1	7	54	21.7	0.636%
Mnlti	—	4	1	1	6	46	23.7	0.256%

(b) *Antenatal care*

Four cases had no antenatal care.

Four had limited care (less than 3 attendances at a midwife only clinic). Five had good antenatal care (seen by a medical officer on 3 or more occasoins).

Of the last category preeclampsia had been detected in 4 of them.

Two had refused admisson while the other two developed past partum eclampsia following delivery by induction. The other case presented with severe pre eclampsia and developed intrapartum eclampsia, following induction of labour. None of the patients had been seen prior to 02 weeks of gestation.

(c) *Relation to gestational age*

Table II: Distribution of casses according to gentational age

accurence	Period of gestation (in weeks)					Total	%
	< 31	31-35	36-40	> 40	*Not known		
Ante partum	—	1	2	—	4	7	53.8
Intra partum	—	1	1	1	—	3	23.1
Post partum	—	1	1	—	1	3	23.1

Mean gestational age = 32 weeks

*Clinical estimation of the gestational age (according to the fundal height) in the cases where the dates were not known:

> 31 weeks - 1

31 - 35 weeks - 3

36 - 40 weeks - Nii (Mean = 32 weeks)

(d) *Blood pressure, oedema and albuminuria*

Significant generalised oedema was present in all the cases. The highest recorded blood pressure was 210/140 mm Hg, and the lowest 130/90 mm Hg. The mean diastolic pressure in the series was 114 mm Hg, wilh 9 out of the 13 cases having a diastolic pressure of 110 mm Hg, or more.

Except the two cases of moderate to severe pre eclampsia admitted for induction, the rest had significant albuminuria on admission. Of the former two, one developed albuminuria during the labour and the other only with the onest of post partum eclampsia.

(e) *Convulsions*

10 patients (76.9%) had only the presenting fit which was quickly controlled. One patient had a history of convulsions at home but none since admission. One patient had two post partum fits occuring within $\frac{1}{2}$ an hour of delivery. One patient had a total of seven convulsions over a period of $5\frac{1}{2}$ hours. She was delivered within 5 hours of the first.

(f) *Drugs used*

The drugs used for the initial control of convulsions and for subsequent sedation varied according to the case and our previous experience, The drugs commonly used were Morphine. Sodium Amytal Diazepam, Pethidine and Largactil (often intravenously and sometimes intramuscularly).

e of delivery

III: Mode of delivery in relation to labour

Labour	Mode of delivery			
	Normal vaginal	Forceps	Caesarian section	Total
Spontaneous	4+1 twin delivery	3	1	10
Induced	2	1	1	4
Total	8	4	2	14
Percentage	57.1	28.6	14.3	

(h) *Foetal outcome*

TABLE IV: Foetal outcome in relation to birth weight

Birth Weight	*Still Births		Live Births	Neonatal deaths	Total foetal loss	Percentage foetal loss
	A	B				
< 1.0 Kg	—	—	—	—	—	—
1.0 — 1.5 Kg	1	2	1	—	3	75
1.6 — 2.0 Kg	2	0	3	1	3	60
2.1 — 2.5 Kg	1	0	2	0	1	33
2.6 — 3.0 Kg	0	0	2	0	0	00
> 3.0 Kg	—	—	—	—	—	—
Total	4	2	8	1	7	50

*Still Births A = Death in utero prior to admission to the unit

B = Death in utero after admission to the unit

Average birth weight in the series = 1.9 Kg.

(i) *Maternal complications*

TABLE: V.

Complications	Number of C...
Pulmonary oedema	01
Prolonged coma	01
Respiratory depression	01
Abruptio placentae	01
Acute renal failure	01
Hyper pyrexia	Nil
Uncontrollable convulsions	Nil
Disseminated intravascular coagulation	Nil
Cerebro vascular accident	Nil
Trauma	Nil
Urinary tract infectiou	06
Chest infection	05

Three cases (23%) presented with major complications and one of them had pulmonary oedema, prolonged coma and respiratory depression.

(j) *Maternal mortality*

Nil

Discussion

Thirteen cases of Eclampsia were diagnosed during the two year period of study. The incidence of 0.378% is considerably higher than that reported by workers in the more developed counrries.

Donald (1979) – In Queen Mothers Hospital, England
1964 – 1965; 0.25%
1970 – 1971; 0.11%

Wightman et al (1978) in Cardiff, Wales, 1965-1974: 0.072%

Although in the study the relative proportions of primigravidae to multigravidae were almost equal the actual incidence (which depends on their relative proportions in the total number of deliveries) was found to be more than two and a half times higher in the primigravida. Dawn and Sinha (1979) in India, found only 19 multigravidae out of a total of 141 cases of eclampties. Wightman et al (1978) in Wales found that the incidence in primigravidae was (0.136% and 0.031% respectively).

Since eclampsia is essentially a preventable condition, poor antenatal prophylaxis is the primary factor responsible for its occurence. This was well seen in this study where 61... of patients had no proper antenatal care, ...f of whom had no care at all. Even with pr...er antenatal care, some mothers refuse admission to hospital. As Dewhurst (1981) suggests this is due to either the mother being unable to understand the gravity as she has no symptoms, or she

is unable to temporarily adandon the husband and children (due to various reasons). In our study, antepartum eclampsia remained the commonest presentation (53.8%) and all of them had their initial convulsion at home. The rest were equally distributed between the intrapartum and the p(partum periods.

In countries where antenatal care has vastly improved, antepartum eclampsia has become retativeiy less common and post partum eclampsia more so.

The stress of labour is thought to aggravate a moderate situation which then progresses to

TABLE VI: **Occurence of Eclampsia**

Source	Ante partum	Intra partum	Post partum
Queen Charlotte's Hospital, London 1967–1969 (Dewhurst 1976)	1	8	17
Confidential Enquiry in the United Kingdom 1973–1975 (R. H. S. S. 1979)	10	1	7
Bankura Sanmilani Medical College Hospital India, 1975–1977 (Dawn and Sinha 1979)	104	13	22
Cardiff, Wales 1965–1974 (Wightman et al 1978)	14	17	12
Queen Mother's Hospital, England 1964–1971 (Donald 1979)	8	12	11
Present study 1980–1982	7	3	3

eclampsia, In our study it was noted that in the patients developing intra and post partum eclampsia the sedation was inadequate inspite of early warning signs. With early detection and treatment of pre eclampsia, and early delivery of cases with impending eclampsia, the ante partum onset of eclampsia could be prevented; but if the same vigilance is not continued during and soon after labour, intra and post partum eclampsia cannot be prevented.

Complications

(1) *Pulmonary oedema*

This results from a combination of hypertension, exhaution, and fluid and electrolyte imbalance. All three factors were thought to be responsible in our patient who responded to large doses of frusemide.

Gedekoh et al (1980) recorded 3 our of 52 eclamptics developing pulmonary oedeme due to excessive intravenous fluid administration, while Dawn and Sinha (1979) had an incidence of 12.8% in their series of 141 cases. A specific myocardial lesion due to hypoxia, has also been incriminated (Lawson, 1982).

(2) *Prolonged coma and respiratory depression*

This is very sinister. and may follow a cerebro vascular accident or raised intracranical pressure oedema following convulsions. The patient in this study who developed this complication had been given intravenous Morphine, for the control of convulsions, and the condition was reversed with Lethidrone. Morphine is known to cause a raised intracranial pressure and acidosis by

respiratory depression. On account of this both Lawson (1982) and Donald (1979) advise against its use.

(3) *Abruptio placentae*

In this condition the maternal prognosis is serious as it was in our patient who went into a state of shock soon after delivery and had to be resuscitated with rapid blood transfusions. Because of their preexisting hypovolaemia and haemo-concentration, eclamptics tolerate blood loss very badly. The hypovolaemia is related to the reduction in the capacity of the intravascular compartment bo generalised vaso spasm (Lawson 1982). Hence replacement of fluid should be carefully monitored to prevent circulaiory over-load (Hibberd et Rosen 1977). Our patient developed convisions only after resuscitation, by which time the blood pressure was noted to have risen to 160/110 mm Hg. from 130/90 mm. Hg. prior to delivery. In this case, injudicious intravenous infusions may have precipitated eclampsia.

(4) *Acute renal failure*

Oliguria is a common accompaniment of eclampsia, and reflects its renal effects. It needs no specific therapy immediateiy, the termination of pregnancy being the first priority as powerful diuretics such as frusemide reduce the blood volume further and aggravates the impaired renal perfusion (Lawson 1982). Sometimes this can progress to actue tubular necrosis or massive cortical necrosis. One partient in this study presented in this thus, with persistant oliguria and rising blood urea levels 48 hours after delivery. Since there was no response to Mannitol and Frusemide, she needed dialysis.

(5) *Hyperpyrexia*

Not seen in this study. In the absence of any infection it is thought to be due to anoxic changes in the temperature regulrting centres in the midbrain (Lawson 1982). Since it can be detri-mental to both heart and brain it requires urgent therapy. Dawn and sinha (1979) reported an incidence of 9% of cases in their study develo-ping a temperature of more than 105 ° F.

(6) *Uncontrollable fits*

Our patients who had 5 convulsions within 5 hours cannot be strictly included under thiscategory because the fits were soon controlled by the delivery of the baby. Various measures have been adopted in this condition including intra-venous Pentothal Sodium. Chan aud Dillikon in 1970 (as buotet by Lawson 1982) have reported a case where 17 fits had occured within eight hours and paralysis with D-Tubocurrarine was necessary before they could be brought under control.

(7) *Disseminated intravascular coagulation*

The hypercoagulable state seen in a normal pregnancy seems to be exaggerated in pre eclam-psia and eclampsia (Boner et al 1971). This is probably due to the release of thromboplastic substance which may arise from blood platelets or from the release of trophoblastic tissue and amniotic fluid into the uterine circulation. However the deposition of fibrin could be secondary to tissue damage following immunological reactions (studd 1977). It is now believed that the coagu-lopathy which is seen in eclampsia and pre eclampsia is more a cause rather than an effects of the condition (Symonds 1979). In the present study there was no clinical evidence of this condi-tion, which is said to be rare in Sri Lanka. Wightman et al (1978) had two cases out of 43 who developed clinical msnifestations of this condition. Gedekoh et al (1980) recorded one due to this condition found 24 out of 46 eclam-ptics to have thrombocytopenia.

(8) *Cerebro vascular accidents and other neuro-logical sequelae*

In our group of eclamptics, there were no cases with these complications which occur primarily due to the hypertension and secondarily due to convulsions. Gedekoh et al (1980) found 9 of their 52 cases developing significant neurological sequelae, of which two died of cerebro vascular accidents.

(9) *Trauma due to the convulsions*

There were no serious injuries in our patients. Gedekoh et al (1980) record an impressive list of complications including fractures, dislocations, amputation of tongue and even two car collisions!

(*10*) *Infections*

Inspite of prophylactic antibiotics being used routinely, there was a high incidence of chest infections (38%) and urinary tract infections (46%) in this study. Collection of secretions and hypostatic pneumania, and aspiration of vomitus was responsible for the former while catheterization was the probably cause of the latter. All patients in this study had indwelling catheters for accurate estimation of urine output. One patient developed a lung abscess probably due to aspiration of vomitus.

Mode of delivery

Vaginal delivery was preferred in the unit and 86% of the cases were delivered vaginally, and about one third of them required assistance with 'lift out forceps.' The principle was to first control the convulsions and then induce labour after 12–24 hours sedation, if spontaneous labour was not established by then. This conservative regime was first suggested by Strogonoff about 50 years ago. He used Morphine by injection together with Chloral and Bromide per rectum for heavy sedation. The fairly high maternal mortality was drastically reduced by this method. The principles of his management are still followed save that more effective and safer drugs are used and there is a tendency for earlier delivery. In our series only two patients were delivered by caesarian section. The first for an obstructed labour due to a dead foetus with shoulder impaction, and the second for foetal distress, where the baby was salvaged. In the past, caesarian section in eclampsia was associated with a high maternal mortality, perhaps because it was used as a last resort. However Menon (1961) in Madras, Lopez Llera (1967) in Mexico, and Crichton et al (1968) in Durban have shown that it is a relatively safe procedure. But all of them have shown that the maternal remains the same whether the patient is abdominally or vaginally. Only the foe come is significantly improved.

Menon (1961) and Lopez Llera (1967 shown that the outlook for both mother and is worse, the longer the delivery is de Delivery should therefore be effected pro by whatever means, and this ensures the prognosis for both mother and baby. I series of 10 mothers who developed eclar before delivery the mean time duration presenting convulsions to delivery was 15 h the shortest time being ½ an hour and the lo 47 hours. (The patient who took 47 hou deliver presented with one convulsion at a p of gestation of approximately 30 weeks, clinic Death in utero occured 18 hours later, wi further convulsions, and the eclamptic dramatically subsided. Spontaneous labour established after about 36 hours. The fo weighed 1 Kg at birth).

Maternal mortality

Although there were no maternal deaths in series, it still remains a major cause of mate mortality especially in the developing w (Lawson 1982). During the period 1973–19 the most frequent cause of maternal death England and Wales was hypertensive disease pregnancy of which 54% were due to eclamp Even out of these 21 cases of maternal dea associated with eclampsia were due to cereb haemorrhage. (Confidental Enquiries i Maternal Deaths 1979).

Maternal mortality is highest with ante a intra partum eclampsia and also tends to higher the shorter the period of gestation (Dewhurst 1981).

Maternal mortality rates reported by va workers are as follows.

Menon (1961) out of 402 patients in Madras:	2.2%
Lopez–Llera (1967) out of 107 patients in Mexico:	10.3%

Lean et al (1868) out of 90 paiients in Singapore:	3.3%
Prichard et Pritchard (1975) out of 72 patients in Texas:	Nil
Wightman et al (1978) out of 72 patientns in Wales:	Nil
Dawn et Sinha (1979) out of 55 patients in India:	9.0%
and out of patients:	17.5%
Gedekoh et al (1980) out of 52 patients in Pennsylvania:	5.8%

But as these studies were carried out under very different conditions and circumstances using different modes of management, these results are not strictly comparable.

Summary

Thirteen cases of Eclampsia presenting over a two year period were studied with reference to their occurence and maternal sequelae. The relatively high incidence found and the fact that antepartum eclcmpsia was the commenest mode of presentation, reflects poor antenatal prophylaxis. Three paticnts presented with serious complications and these were discussed together with other possible complications. Inspite of prophylactic antibiotics, chest infections and urinary tract infections were common. Vaginal delivery after the convulsions were controlled, was preferred to abdominal delivery. There were no maternal deaths.

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