The Bulletin of the
Kandang Kerbau Hospital,
Singapore.
Vol. IV—No. 2—October 1965

Acute renal failure in obstetrics and gynaecology

by

T. A. Sinnathuray, MBBS (M), FRCS (EDIN. & GLAS.), MRCOG.

KANDANG KERBAU HOSPITAL, SINGAPORE.

Introduction

It is well over 60 years, since anuria due to massive cortical necrosis was first described by Bradford and Lawrence in 1898; but little progress was made in this subject or in anuria, in general, until World War II when the problem of post-traumatic renal failure following crush injuries to skeletal muscles aroused considerable interest. It then became clear that the pathological changes found in the kidneys in these fatal cases of crush injury with resultant acute renal failure and eventual death, were also manifested in many other clinical conditions.

It is stated that about 50 per cent of female patients with acute renal failure are associated with pregnancy (Ober et al, 1956); and that this condition accounts for about 6 per cent of maternal deaths (Parker et al, 1959). In general, it is stated that acute renal failure has a survival rate of approximately 50 per cent (Ramsay, 1964). However, a review of the literature reveals that the survival rates following acute renal failure are influenced by many factors, such as age-group, sepsis, coexistence of medical and surgical conditions, and the regime of therapy instituted. It is especially encouraging to note that acute renal failure in obstetrics and gynaecology is a potentially salvageable condition, since the obstetric patient is usually in the younger age group and often without other complicating stigmata. It is therefore, obvious that vigorous therapeutic measures should be utilised in the management of these cases.

Definition and Concept

Acute Renal Failure is a clinical state in which there is a sudden and severe reduction of renal function in an individual, who previously had normal parameters. It is invariably associated with a marked reduction in the daily urinary output to below 400 ml. This state of acute Renal Insufficiency brings in its trail a sudden derangement in the haemodynamics of the cardiovascular system, and also in the biochemical and metabolic patterns of the circulating blood, which if severe and remain unreversed for long, will invariably predispose to the patient's death.

Aetiology

The classification of the aetiology of acute renal failure is to a great extent dependent on the biasness of the medical speciality under consideration. Thus physician, surgeon and obstetrician will each vary to some extent on their pattern of classifying the aetiology of acute renal failure. Viewed from a physio-pathological basis, acute renal failure is the end-result of any one or more of the undermentioned pathology in the renal tract (Table I).

TABLE I

Physio-Pathological Aetiology

(After H.E. De Wardener (1961)).

1. Severe functional changes without structural damage.
2. Severe functional changes with acute structural damage.
3. Functional changes of perhaps moderate severity but occurring in a patient with chronic structural damage.
4. Acute urinary tract obstruction.

However, from the clinician's viewpoint, acute renal failure in obstetrics and gynaecology may be the sequelae of any one of the aetiological factors enumerated in Table II.

TABLE II

Clinical aetiology of acute renal failure in obstetrics and gynaecology

1. Shock
   - (i) Neurogenic.
   - (ii) Oligaemic.
   - (iii) Bacteraemic.
   - (iv) Anaphylactic.
2. Septicaemia.
3. Fulminating PET/Eclampsia.
4. Concealed Accidental Haemorrhage.
5. Fulminating Renal Infection.
6. Intra-Vascular Haemolysis
   - (i) Blood Groups
   - (ii) Bacteria
   - (iii) Chemicals
7. Acute Urinary Tract Obstruction.
8. Nephrotoxic Agents.

1. Shock

Shock is a clinical state of acute peripheral circulatory failure and can occur with a fair degree of frequency in obstetrics and gynaecology. Shock of neurogic origin is classically associated with severe traumatic obstetrical procedures or accidents, such as ruptured uterus and intra-uterine manipulations without adequate anaesthesia. Shock in concealed accidental haemorrhage is partly of neurogenic origin. Oligaemic shock can be the sequelae of severe antepartum or postpartum haemorrhage, severe abortions, ruptured ectopic pregnancies, or even extensive uterine ruptures. Bacteraemic shock in obstetrics and gynaecology is relatively uncommon, and when it does occur, it does so with criminal septic abortions. Anaphylactic shock is also rare in obstetrics and gynaecology, the commonly cited examples are penicillin induced-anaphylaxis and the rare condition of amniotic fluid embolism.

Whatever be the mechanism of shock in obstetrics and gynaecology, acute renal failure is a potential hazard if the shock is severe and of prolonged duration. The renal failure in these cases is the sequelae of acute renal vascular insufficiency leading to severe functional changes in the kidney with or without acute structural damage.

2. Septicaemia

Septicaemia in obstetrics and gynaecology is usually the sequelae of uncontrolled sepsis associated with septic abortions and puerperal uterine infection. Less frequently it may follow puerperal septic thrombophlebitis. Common causative organisms are streptococci, Proteus Vulgaris, Bacterium Coli and Chlostridum Welchii. Fulminating septicaemia predisposes to renal infection, which, in its turn, causes renal ischaemia, and this may proceed to acute tubular necrosis, with resultant acute renal failure. Severe chlostridial infection also precipitates intravascular haemolysis which, if severe enough can predispose to acute renal failure, as will be shown later.

3. Fulminating PET/Eclampsia

Fulminating pre-eclamptic toxaemia and eclampsia is invariably associated with a state of toxaemic nephropathy. This is characterised by acute renal vascular insufficiency which predisposes to acute tubular necrosis and resultant state of acute renal failure. Ober et al (1956) state that maternal death in the patient with acute eclamptic nephropathy usually occurs from fulminating cerebral or pulmonary oedema.

4. Concealed Accidental Haemorrhage

Concealed accidental haemorrhage is usually a sequelae of fulminating toxaemia of pregnancy, although not always so. Non-toxaemic severe concealed accidental haemorrhage may sometimes follow sudden decompression of a gross hydramnios, or severe direct trauma to the uterus, such as a kick on the lower abdomen, or a motor accident.

The mechanism of acute renal failure in cases of concealed accidental haemorrhage is still a matter of some speculation. The severe shock that accompanies this condition may be the main factor precipitating acute renal circulatory insufficiency with resultant acute renal failure (Bourne and Williams, 1962). Others have speculated on the possible existence of additional factors which precipitate the classical bilateral cortical necrosis seen in these cases of concealed accidental haemorrhage, Whatever the mechanism of acute renal failure in this

condition, pathologically tubular necrosis can follow less serious types of concealed accidental haemorrhage, and cortical necrosis is the sequelae of very severe types of this condition. Bull et al (1955) also hold the view that the occurrence of cortical necrosis may well depend upon the severity of the initial retro-placental bleeding, and that the extent of cortical necrosis is probably also closely proportional to the severity and extent of the accidental haemorrhage.

5. Fulminating Renal Infection

Infection of the renal parenchyma can be acute or chronic in nature, or it may be an acute inflammation superimposed on a chronic renal infection. Ascending acute pyelonephritis, and flare-up of chronic pyelonephritis are said to be common complications of pregnancy, and if untreated, or inadequately treated can predispose to considerable destruction of the renal parenchyma, with resultant state of acute functional renal insufficiency . Such forms of acute renal failure were encountered with a fair degree of frequency in the pre-antibiotic era; however the ready availability of a broad spectrum of antibiotics now has ensured the possibility of effectively controlling and curing these infections, long before crippling renal parenchymatous destruction could take place.

6. Intra-Vascular Haemolysis

Severe degrees of intra-vascular haemolysis sufficient to precipitate acute renal failure, are rarely encountered in obstetrics and gynaecology. Mismatched blood transfusions are probably the commonest of the haemolytic causes of acute renal failure. Less commonly, chlostridium welchii or haemolytic streptococcal septicaemia, following upon a criminal septic abortion or puerperal uterine sepsis, may precipitate a severe state of intra-vascular haemolysis with resultant acute renal failure. Chemical(drugs) induced haemolysis are less frequently encountered in obstetrics and gynaecology. Whatever the underlying aetiology, severe intra-vascular haemolysis predisposes to acute renal failure by precipitating a reduction in the circulating blood volume, which in its turn predisposes to acute renal ischaemia and acute tubular necrosis.

7. Acute Urinary Tract Obstruction

Acute renal failure from acute urinary tract obstruction can obviously follow bilateral pelvic or ureteric obstruction, or unilateral obstruction to a single functioning kidney. This indisputable truth is often forgotten when considering the differential diagnosis of acute renal failure. In the context of obstetrics and gynaecology, acute urinary tract obstruction can arise from the severance or ligation of the ureters during pelvic surgery, be it hysterectomy, caesarean-hysterectomy or the more radical Wertheim-hysterectomy. Damage to the ureters during surgery is more liable to occur in the hands of the inexperienced gynaecologist. However, in the presence of advanced chronic pelvic infection, pelvic endometriosis or pelvic cancer, the avoidance of damage to the ureters can be a tricky problem even for the experienced gynaecologist. Acute-on-chronic renal failure can also follow bilateral compression and obstruction of the pelvic ureters in those cases of uterine cancer, where there is extensive peri-ureteric infiltration with cancerous growth, or with fibrosis following radiotherapy.

The striking feature of acute renal failure due to acute urinary tract obstruction is that the flow of urine is completely suppressed, in contrast to the low flows which are usually found with acute structural damage to the renal parenchyma. The only exception to this rule is in the acute renal failure of acute glomerular nephritis, and in the total bilateral cortical necrosis, that can occur with a severe concealed accidental haemorrhage. The therapeutic implication of this fact is that if acute renal failure is associated with complete or almost complete suppression of the urine, a cystoscopy should be performed and the ureters catheterised to try to relieve the obstruction; if this is unsuccessful it is necessary to release the urine above the obstruction by a nephrostomy (De Wardener, 1961).

8. Nephrotoxic Agents

Nephrotoxic agents as a cause of acute renal failure in obstetrics and gynaecology is quite rare. It is well known that some abortifacient agents used by criminal abortionists may contain mercury, arsenic or lead, which are also

considerably nephrotoxic. Sulphonamides in high concentrations can also be sufficiently nephrotoxic to precipitate acute renal failure. All these nephrotoxic agents act either directly by causing the death of those tubular cells which transport the chemical agent from the blood into the lumen of the tubule, or indirectly by causing intense renal vasoconstriction with resultant renal ischaemia, and under both these circumstances there is acute tubular necrosis with resultant acute renal failure.

Pathology

The pathology of acute renal failure, irrespective of the clinical aetiology, may be schematically presented for discussion, as shown in Figure 1.

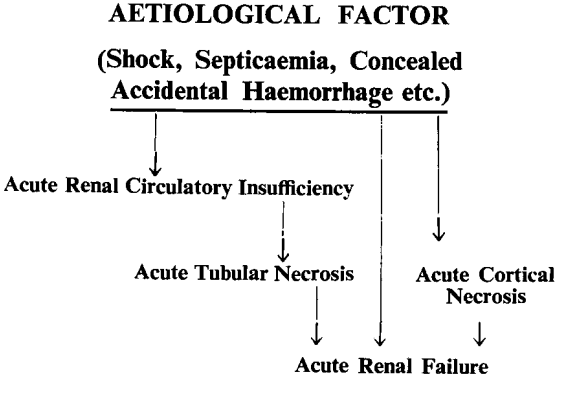

Fig. 1 - Pathology of acute renal failure

Acute renal circulatory insufficiency is one of the commonest and the most important of mechanisms of acute renal failure in practically all branches of medical practice. In obstetrics and gynaecology, acute renal circulatory insufficiency may be the sequelae of severe shock, septicaemia, fulminating toxaemia of pregnancy/eclampsia, and intra-vascular haemolysis. Presumably also, renal vascular spasm resulting in renal anoxia can arise from the effects of nephrotoxic agents, or from the evoking of the utero-renal reflex caused by undue intra-uterine tension, as would be the case in concealed accidental haemorrhage. However produced, acute renal circulatory failure will be followed by acute tubular necrosis, especially if the period of renal anoxia is prolonged for two or three hours or more. Hence the importance of treatment of haemorrhagic shock by adequate blood transfusion with the minimum of delay (Bourne and Williams, 1962).

In obstetrics and gynaecology, although *acute tubular necrosis* is usually secondary to a state of acute renal circulatory insufficiency, it may also result directly from the deleterious effects of fulminating renal infection, nephrotoxic agents, acute urinary tract obstruction, and last but not least, secondary to concealed accidental haemorrhage. In fact, all the obstetrical and gynaecological aetiological factors, that have been discussed, predispose to acute renal failure by precipitating acute tubular necrosis.

Acute bilateral cortical necrosis is a rare pathological cause of acute renal failure occurring usually in the last trimester, and after a severe "*concealed accidental haemorrhage*". Occasionally it may follow severe eclampsia, and exceptionally it can be the type of renal necrosis to follow extensive trauma, and thus occur in males as well as females (Bourne and Williams, 1962).

The actual factor determining the occurrence of acute cortical necrosis is, as yet, not at all clear. There is marked shock, and this may be the main factor in the causation of massive cortical necrosis. However, it does seem that the occurrence of cortical necrosis may well depend upon the severity of the initial retroplacental bleeding; and it is highly probable that the extent of necrosis is also closely proportional to the severity and extent of the accidental haemorrhage (Bull et al, 1955). Of course, acute tubular necrosis can also follow concealed accidental haemorrhage, usually of the less serious type. As a differential clinical diagnostic feature of acute cortical necrosis, MacGillivray (1950) noted that these cases displayed a rise of temperature on the second or third day, rather on the lines of pyrexia about this time in cases of coronary thrombosis.

In severe cortical necrosis, the outer ¼ inch of the kidney is white, and contrasts sharply with the medullary portion of the kidney. In gross cortical necrosis, there is no regeneration of the glomerular tissue—the whole nephron is dead—and the prognosis is fatal. Histologically, there is, in these bad cases, widespread degeneration amounting to necrosis throughout the

ccrtex and involving all the glomerular tufts, as well as the tubular portion of the nephron in the total cases. The afferent glomerular vessels are dilated and the interlobular arteries are markedly engorged (Bourne and Williams, 1962).

However, the minor and focal cases of cortical necrosis may survive providing that a sufficiency of the glomerular area is undamaged and sustains no further damage. To ensure this latter point it would seem probable that in treating cases of concealed accidental haemorrhage early blood transfusion in adequate quantities is perhaps valuable. Artificial rupture of the membranes is also theoretically desirable, since it will lower the intra-uterine pressure, and thereby minimise or abolish the so-called utero-renal reflex (Bourne and Williams, 1962).

Conclusions

Acute renal failure in obstetrics and gynaecology is the principle cause of acute renal failure in females, and an important cause of maternal mortality. It is a potentially salvageable condition. Early diagnosis and the prompt institution of treatment should considerably enhance the maternal salvage in acute renal failure associated with pregnancy.

Summary

1. Acute renal failure is an important facet of obstetrical and gynaecological practice.
2. A concept of acute renal failure has been put forth.
3. The aetiology of acute renal failure has been tabulated and discussed from the viewpoint of the obstetrician and gynaecologist.
4. The physio-pathology of acute renal failure in obstetrics and gynaecology has been briefly discussed.

References

Bourne, A.W. & Williams, L.H. (1958): "Recent Advances in Obstetrics and Gynaecology", 9th Ed., Churchill, London, p.131.

Bourne, A.W. & Williams, L.H. (1962): "Recent Advances in Obstetrics and Gynaecology", 10th Ed., Churchill, London, p.149.

Bradford, J.R. & Lawrence, T.P.W. (1898): J. Path. Bact., **5** 189—as cited by Bourne and Williams (1958, 1962).

Bull, G.M. et al (1955): Lancet, **2,** 1152.

De Wardener, H.E. (1961): "The Kidney", 2nd Ed., Churchill, London, p.127.

MacGillivray, I (1950): J. Obstet. Gynaec., Brit. Emp., **58,** 92.

Ober, W.B. et al (1956): Am. J. Med., **21,** 781.

Parker, R.T. et al (1959): South. M. J., **52,** 251.

Ramsay, A.G. (1964): Clin. Obet. & Gynec., **7,** No. 1, 219.