CURRENT SURVEYS

Injuries to the upper urinary tract

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The commonest causes of rupture of the kidney are either road traffic accidents or rugby football. A kidney can be damaged by a direct blow in the loin, such as when a player falls on the ball and the pack forms a loose scrum, kicking everything in sight including the victim's loins. Another form of sport in which renal damage is not infrequent is horse-riding. If the rider is thrown and lands across the top pole of a jump, he is often falling laterally at the time of impact, and it is the rider's loin that takes the blow. A kidney can also be injured indirectly by compression between the twelfth rib and the vertebral column. The mechanics of this injury can be best demonstrated at the time of operative exposure of the kidney, when the patient is in the lateral position with either the bridge raised or the table split. If a hand is passed across the peritoneal cavity the opposite kidney can be felt compressed between the lower border of the twelfth rib and the vertebral column. In fact the compression is such that it is impossible to pass a finger between the kidney and the lower border of the twelfth rib. If such compression occurred suddenly and forcefully, it could quite easily split the kidney, and this situation could arise either from a direct blow in the loin or, also, from sudden lateral flexion of the trunk.

The extent of kidney damage

Damage to the kidney parenchyma can be classified very simply into:

1. An incomplete split. This may involve a calyx without tearing the capsule or, conversely, it may be a simple split of the capsule without damage to the calyx.

2. A complete tear, both of capsule and calyx, which is probably the commonest type of injury encountered.

3. A complete split of the kidney into two separate halves.

A simple tear of renal parenchyma entering a calyx without damage to the capsule of the kidney can produce haematuria without a surrounding haematoma. A tear through the capsule without damage to one of the calyces usually produces only a small haematoma around the kidney and haematuria will probably be delayed as blood will only reach the calyx via damaged tubules. Neither of these injuries is likely to produce any serious consequences and the kidney will recover without deformity. The complete tear through capsule and calyx produces haematuria, a large loin haematoma and also leak of urine into the perirenal tissues. This urinary leakage is rarely of any clinical significance, as the urine is sterile and the leakage stops fairly readily. A complete split of the kidney usually means that one half will die of avascular necrosis. Cases, however, have been reported where both halves of an almost completely split kidney have ultimately survived and continued to function.

A separate group of injuries is that in which the renal pedicle is damaged. This may be a tear in the renal artery, or one of its branches, or complete avulsion of the major vessels. This latter type of injury is occasionally found in association with other major abdominal injuries, from which the patient rarely survives. These avulsion injuries were seen during the war from gunshot wounds, which penetrated the loin and produced a disruptive effect in the region of the renal vessels.

The diagnosis of injuries to the kidney

The diagnosis of a ruptured kidney is often made by the patient himself when he passes the next specimen of urine and finds that it contains blood. In a high proportion of rugger injuries of kidneys the patient will continue the game for a few minutes, or even until the end of the match, and will only report sick when he notices the haematuria. On examination these patients always have a raised pulse, and it is often impossible to feel the haematoma in the loin at this early stage as there is usually a considerable amount of guarding and the patient is too tender. It may not be until
the following morning, when the tenderness is easing off and the immediate bleeding has stopped, that the patient will allow examination of the loin and the haematoma can then be easily distinguished. In fact, it is probably unwise, when suspecting such an injury, to risk palpating the loin sufficiently firmly to feel the kidney, for fear of stimulating further haematuria.

Haematuria may be delayed as much as 24–48 hr after the injury. The only explanations that can be offered for this long delay are, either that there may have been bleeding into a calyx which has clotted and the haematuria is only seen after the clot is dislodged, or perhaps the milder type of injury, where the kidney is contused without calyceal damage, and blood reaches the renal pelvis only after hold-up and subsequent drainage down the tubules.

**Incidence of injury to pathological kidneys**

A kidney which is already diseased and, in particular, the hydronephrotic kidney, is more liable to damage by trauma. Out of fifty-nine cases reported by Slade, Evans & Roylance (1961), nine kidneys (15%) were found to have been pathological before injury. There is also the possibility of haematuria arising from a pathological kidney opposite to the side of injury. A small boy with haematuria gave a history of having fallen off a low wall a few hours previously. He was very well and only indicated his left loin as the site of injury. On examination there was no loin bruising, but a little deep tenderness in the loin. However, on intravenous pyelography his left kidney was normal, but his right kidney showed gross hydronephrosis. This was the opposite side to the side of the injury and, at nephrectomy, stale blood was found in this hydronephrotic kidney, confirming that the bleeding had come from that side.

Suspicion of pre-existing renal pathology should be aroused if the injury seems very slight and the patient’s condition is too fit to be compatible with a ruptured kidney.

**Management of kidney injuries**

Thirty years ago at least one in five renal injuries were explored surgically. In the last 20 years the approach to renal trauma has been much more conservative and exploration for such an injury is now a rarity.

The usual indications for surgery to a ruptured kidney are, either as a life-saving procedure where blood loss is obviously severe and persistent, or, secondly, where infection of the large haematoma around the kidney has occurred. Exploration usually amounts to nephrectomy and, for this reason, it is essential, before embarking on surgery, to check the opposite kidney by intravenous pyelography if possible. Solitary functioning kidneys are frequent enough to present a real danger when contemplating nephrectomy for trauma.

Working on the assumption that by far the majority of ruptured kidneys can be treated conservatively, the first approach should be to avoid aggravating the bleeding and to watch carefully to ensure that the bleeding is stopping. The patient should be put to bed and sedated. These injuries often occur in young active adults, who do not take kindly to being put to bed and told they must lie still until the bleeding eventually stops. Often they can only be kept adequately rested by heavy sedation.

To check on the amount of bleeding, a ½-hourly pulse must be taken at first, the time interval being increased only when the pulse rate is falling. The best guide to the reduction in bleeding is the appearance of the urine, of which all specimens must be saved in separate containers and each marked clearly with the date and time that it was passed. Change in the depth of colour indicates the quantity of bleeding and the change from bright red to brown discoloration indicates that the haemorrhage within the kidney is stopping. The patient should be encouraged to drink copiously, as he may develop some pain resembling a renal colic from the passage of clots down the ureter, which usually succeeds in passing these clots successfully.

Antibiotic cover should always be given, as the kidney will be surrounded by a haematoma of considerable size, which can be a perfect nidus for the development of any infection. The number of kidneys that had to be explored 30 years ago was largely due to infection occurring in the haematoma, with secondary haemorrhage at 10–14 days. Empirical antibiotic cover should therefore be started soon after the injury and continued for a full fortnight.

Blood loss into a loin haematoma can amount to 2 or 3 pints and therefore treatment will, of necessity, include replacement of this blood loss.

**X-ray investigation**

Intravenous pyelography should be carried out as soon as the patient is fit to move to the X-ray department. In the majority of occasions this investigation is postponed until the haematuria has ceased. The true value of this investigation in ruptured kidneys is not so much to obtain a picture of the extent of damage to the injured kidney, but an assessment of function of both kidneys and to ensure that the opposite uninjured kidney is quite normal.

The features demonstrable on intravenous
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pyelography of a recently injured kidney are distortion of the calyceal pattern of the kidney or even a leak of radio-opaque dye out to the periphery and into the perinephric tissues. Next there may be delayed or absent function on the injured side. The renal outline cannot be distinguished and there will be loss of the psoas shadow, due to the collection of blood in the perirenal tissues. One other feature commonly seen in renal injuries, as with any loin injury, is a lumbar scoliosis with its concavity towards the injured side, due to spasm of the quadratus lumborum and erector spinae muscles.

Late results of renal injuries

A transient hydronephrosis may occur 2 or 3 weeks after the injury, presumably due to the perirenal and periureteric haematoma. This hydronephrosis can be shown to resolve completely in most cases as the haematoma absorbs.

Occasionally urine leakage may persist through the rupture of the kidney, creating a steadily increasing mass in the loin, even though the haematurnia has ceased. Such a leakage may require surgical drainage and is not necessarily an indication for nephrectomy. A kidney so damaged, which leaks urine into the perirenal tissues, can still recover part or all of its function.

Slade et al. (1961) followed up cases of renal injuries with intravenous pyelography performed 1–10 years after the original accident. Of the fifty-nine cases, the initial X-ray in twenty-four patients showed some abnormality due to the injury and, out of those, nine still showed residual deformity one or more years later. Many of these abnormalities were virtually of no consequence and the function of the kidney was normal. Three, however, showed some permanent severe damage in the form of a staghorn calculus in one, and a functionless kidney in two cases. One of these functionless kidneys was due to obstruction from scarring in the renal pelvis. The other was due to impoverished blood supply, which had resulted in an ischaemic shrunken remnant. One feature that was clear from their review was that none of these complications could have been avoided by any surgical interference at the time of the original injury. On the other hand, where there was a calyceal rupture shown radiologically in the initial X-ray, almost half of the cases showed some permanent structural abnormality in the final follow-up picture. Incidentally, both the patients with the non-functioning kidneys are in fact symptom-free and have no evidence of hypertension.

It is interesting to compare their end results in this series of fifty-nine, where there was a mortality of three, all of whom died of multiple visceral injuries shortly after admission to hospital, with a series reported by Wright (1965) where there was a higher incidence of laparotomy for the original injury and the overall mortality proved to be 10%.

Penetrating wounds of the kidney

A penetrating wound may occur either as the result of a stab or gunshot wound. Here the problem is somewhat different in that the blood will extravasate in all directions. In the closed renal injury the haematoma around the kidney is confined by the perirenal fascia (fascia of Zuckerkandl), but in the penetrating wound blood will not only extravasate up and down the retroperitoneal tissues but, if the peritoneal covering of the kidney is also damaged, blood will be found intra-peritoneally.

Surgical intervention in penetrating wounds

Surgical intervention is nearly always necessary in a penetrating gunshot wound, because there is a much higher risk of injury to other viscera. Occasionally, during the war, it was reported that a kidney could be preserved even with injury to the renal vascular pedicle. Parker (1947) found a branch vessel of the main renal artery damaged and successfully tied this with complete survival of the kidney.

Injuries to the renal pelvis and ureter

The renal pelvis and ureter usually succeed in avoiding injury unless there is gross disruption of other structures in the abdomen. The ureter is well-protected and even in gunshot wound injuries was very rarely damaged.

In urological practice, however, the commonest cause of ureteric injury is the gynaecological operation. Here the ureter might be caught in a stitch at the time of anterior colporrhaphy or vaginal hysterectomy. Traction being put on the pelvic tissues during either of these operations distorts the anatomy and moves the ureter out of its normal position, making it increasingly vulnerable. At open operation, such as in a Wertheim's hysterectomy, the ureter is also at risk. Here, however, the damage is likely to be at a higher level, namely just below the brim of the pelvis, and reconstruction may be extremely difficult owing to the limited length of ureter available to re-anastomose to the bladder.

Spontaneous rupture of the renal pelvis and ureter is a very rare injury. It may be due either to the recent passage of a calculus, which has damaged the wall of the ureter, or, in one personal case the ureter was found to have been injured by a diathermy burn at an appendectomy carried out a week previously.

Finally, mention should be made of the pseudo-hydronephrosis which is a rare, late complication
of injury to the renal pelvis. As a result of the continued leakage of sterile urine, this fluid becomes encapsulated in the loin and collects around the kidney. On intravenous pyelography the kidney itself appears normal, but the leakage of dye can be detected in later films, as it diffuses into the space of the pseudo-hydronephrosis.

**References**


**Virus meningitis**

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The term aseptic meningitis was first introduced by Wallgren (1925) to designate what was thought to be a specific disease and was defined as a syndrome characterized by meningeal irritation associated with a cellular response in the cerebrospinal fluid which was sterile bacteriologically. It is now recognized as being due to a variety of agents the majority of which are viruses. Aseptic meningitis should now only be acceptable as a preliminary term pending investigations and as undifferentiated aseptic meningitis when an etiological diagnosis cannot be established.

The diagnosis of virus meningitis presupposes no overt involvement of the nervous system although separation from encephalitis is not always easy because in some patients a non-specific drowsiness or confusion may be present when, in fact, there is no evidence of an inflammatory reaction in the substance of the brain. Conversely in some patients with encephalitis the cerebral involvement may be so mild as to escape notice and only the meningitic signs and cerebrospinal fluid abnormality may be manifest.

**Etiology**

The recent advances in virus isolation enable an etiological diagnosis of virus meningitis to be reached in 50–70% of cases in the best equipped laboratories.

**A. Common causes**

(i) Enteroviruses: Poliomyelitis 3 types
Coxsackie group A 23 types
Coxsackie group B 6 types
ECHO 30 types

(ii) Mumps.

**B. Uncommon causes**

(i) Lymphocytic choriomeningitis.
(ii) Glandular fever.
(iii) Herpes simplex.
(iv) Adenoviruses.
(v) Arbor viruses.

An increase of cells in the cerebrospinal fluid is found in most cases of herpes zoster but as it is a disease of characteristic presentation and meningitic signs are rarely present it has been purposely excluded from this classification.

**Epidemiology**

Virus meningitis is world wide. The enteroviruses and mumps account for 90% of the known causes (Ch'iu, Ts'ao, Jen & Chang, 1965; Grist, 1965). Not all Coxsackie and ECHO viruses are associated with meningitis but an increasing number have been reported in recent years and in addition further types have probably yet to be identified. The enteroviruses although occurring at any time of the year reach their peak in the late summer and autumn. Surveys have shown that in any one place the incidence of the various viruses differs from one year to the next (Combined Scottish Study, 1964).

Children and young adults account for most of the cases and virus meningitis is infrequently encountered in infancy. The enteroviruses are spread by the faecal–oral route and as in poliomyelitis symptomless infection with Coxsackie and ECHO viruses are common, clinical illness developing in only a small minority. Lymphocytic choriomeningitis is rare in the British Isles and of little importance in most countries. Human infec-
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