women the portal can often be concealed in the edge of the breast. The portal must be flushed both monthly with heparinised saline and after each use.

Patients

Port-A-Cath systems were inserted in 40 patients (16 men, 24 women) at three centres. Thirty seven patients had malignant disease, two asplastic anaemia, and one Christmas disease. The cephalic vein was used for access in 26 patients, the internal jugular vein in 10, the subclavian vein in two, and the external jugular and external iliac veins in one each. Eleven patients received cover with prophylactic antibiotics when their portals were inserted.

At insertion nine patients had abnormal white cell counts, two patients abnormal clotting variables, and five patients platelet counts below 20×10^9/l. The portal was used for blood sampling, as well as administration of virtually all the commonly used cytotoxic agents and blood and blood products.

Thirty six patients found the Port-A-Cath entirely satisfactory, but four found it unacceptable, due largely to discomfort when inserting the needles. The six patients who had previously had an arteriovenous fistula or Hickman catheter for prolonged venous access much preferred the Port-A-Cath. The portals had been in position for an average of 11·6 months (range 1-24 months), and most were still in use, only six having been removed.

Five important complications have occurred, all attributable to errors in the use of the portal. Two portals became infected. One portal was extruded spontaneously when the wound broke down after the start of high dose chemotherapy within 24 hours of inserting the portal. Two portals thrombosed, but one was cleared with urokinase. Four patients died with their Port-A-Cath in situ. None of these deaths could be attributed to the Port-A-Cath.

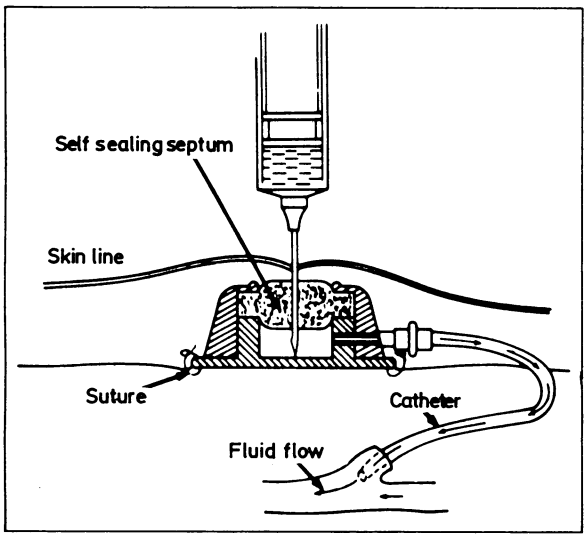

Diagram of implanted portal.

Comment

The Port-A-Cath system is a totally implantable unit that is simple to use. It allows blood samples to be taken or drugs to be administered intermittently over a long period. The only potential source of infection is an inadequate sterile technique at injection. This accounted for the two septic complications in our series. Meticulous technique is essential, and we suggest that only experienced staff should use the portal. Thrombosis is an avoidable complication if the portal is flushed monthly with heparinised saline, although several patients lasted over two months between flushes. There is an undoubted learning curve with this system, and we have encountered fewer complications as experience has accrued. Our results, however, compare favourably with those of other series.[4,5] Patients can inject the portal themselves. One patient has successfully injected factor IX 150 times.

The Port-A-Cath offers considerable advantages over alternative forms of prolonged central venous access. It provides a reliable and easily managed long term route to the central venous system for the clinician, and is acceptable and inconspicuous for the patient, allowing normal life between courses of chemotherapy.

We acknowledge the generosity of Pharmacia (UK), who provided the portals for this study.

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Bleeding from peptic ulcers and use of non-steroidal anti-inflammatory drugs in the Romford area

We are conducting a trial of electrocoagulation in the treatment of bleeding peptic ulcers. A recent report of peptic ulcers related to piroxicam[1] prompted us to examine our data to see how many patients presenting with bleeding peptic ulcers were taking this or other non-steroidal anti-inflammatory drugs. We did not distinguish between different formulations or proprietary preparations of the same drug.

Present series and results

Between November 1982 and March 1985, 460 patients with a presumptive diagnosis of upper gastrointestinal bleeding were examined by gastroscopy within 24 hours of admission. A total of 204 patients were noted to have peptic ulcers, which were bleeding or had stigmata of recent haemorrhage (adherent clot or visible vessel). The data collected about these patients included medication before admission. Unfortunately, other information such as length of treatment was not collected routinely as this was not central to our study. For this reason also data were not collected on patients bleeding from other causes.

Of the 204 patients, 53 (26%) had been taking non-steroidal anti-inflammatory drugs before admission. The table shows the number and percentage of patients taking the commonly found drugs and their mean age. It also shows the frequency of prescription of those drugs nationally and within the North East Thames region in 1983.

There was a difference between the observed distribution and that expected from the North East Thames prescribing data ($\chi^2=13{\cdot}6$; df=4; $p<0{\cdot}01$). This difference lay mainly with piroxicam and ketoprofen, which were observed more frequently, and with ibuprofen, which was observed less frequently than expected from the prescribing frequency. This assumes that the prescribing frequencies in the Romford area were representative of the regional figures.

Of the 14 patients receiving piroxicam, 12 were taking this drug alone. It is claimed that both piroxicam and ketoprofen are rarely associated with upper gastrointestinal haemorrhage[2-4] and so might be given to those patients with previous peptic ulceration. The table shows that few of the patients presenting with bleeding peptic ulcers while taking these drugs had a history of peptic ulceration.

Comment

It is suspected that non-steroidal anti-inflammatory drugs may be associated with peptic ulceration and its complications. If piroxicam and ketoprofen are rarely associated with gastrointestinal haemorrhage, then we might expect fewer patients to present with peptic ulcer bleeding while receiving these drugs. Both drugs are commonly prescribed, however, so we tried to obtain a comparison with the prescribing frequency in our region. From this it appears that these drugs are more frequently associated with gastrointestinal bleeding than might be expected. We cannot exclude the possibility that other drugs prescribed before those indicated might have contributed to the peptic ulceration. The possibility of the district having a different prescribing pattern from the region also cannot be excluded. Unfortunately, more local data are not available, so there is no way of confirming or refuting this.

Bleeding from peptic ulcers and use of non-steroidal anti-inflammatory drugs (NSAIDs) in Romford area compared with local and national prescribing habits

NSAID	No (%) of patients with bleeding peptic ulcers taking NSAID	Expected values from regional prescribing data	Mean age (years)	Patients with ulcer history	No (%) of prescriptions for NSAIDs in 1983 (thousands)†	
					Great Britain	NE Thames region
Indomethacin	9 (17)	10·8	72·1	2	3268 (17)	259 (20)
Ibuprofen	2 (4)	8·8	71·0	1	3544 (18)	211 (17)
Naproxen	6 (11)	5·2	74·5	2	2408 (12)	124 (10)
Piroxicam	14 (26)	6·7	68·6	1	2216 (11)	161 (13)
Ketoprofen	8 (15)	2·2	70·6	0	405 (2)	51 (4)
Others*	14 (26)	19·3	71·6	3	7683 (39)	460 (36)
Total	53 (100)	53	71·1	9	19524 (100)	1266 (100)

*Including sulindac, benoxaprofen, phenylbutazone, flurbiprofen, azapropazone, fenoprofen, mefenamic acid.
†Source: one in 200 sample, SR1 Branch, DHSS.

The Committee on the Safety of Medicines has received 457 reports of upper gastrointestinal bleeding associated with piroxicam since 1970 and 195 associated with ketoprofen since 1971. The corresponding figure for indomethacin since 1965 is 419 (Committee on the Safety of Medicines, personal communication).

We had difficulty locating the data on prescribing frequency given in the table; if such figures (which are Crown copyright) were published problems could be identified early and investigation made. Certainly more detailed studies seem justified.

We have notified the Committee on the Safety of Medicines and Pfizer Limited.

We thank the Medicines Division of the Department of Health and Social Security and Mr D Hewitt and Mr I Spooner, of the DHSS, for their help and advice; Mr Simon Day for statistical help; and our medical and surgical colleagues for allowing us to study their patients.

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Progressive renal failure in a remnant kidney

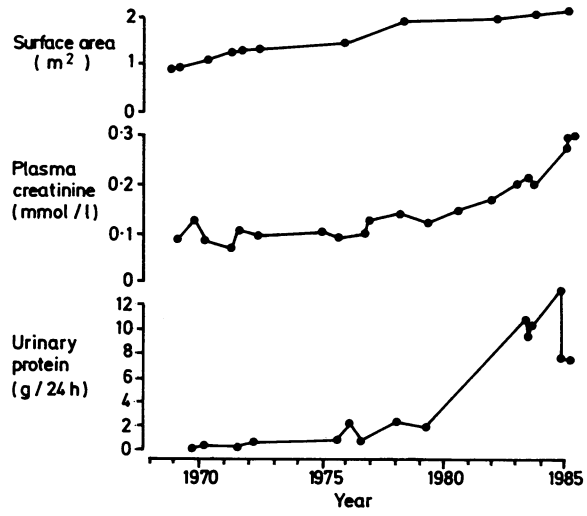
Three quarters nephrectomy in the rat causes glomerulosclerosis in the remnant kidney and progressive azotaemia.[1] Moderate renal failure may start processes that cause further decline of renal function even when the initial mechanism of injury no longer operates and the remaining kidney substance is normal. Similar events may ensue in the human kidney if a substantial proportion of renal tissue is ablated and contribute to the progression of various renal diseases.[2]

We describe a boy aged 3 who lost three quarters of his renal tissue as a result of an accident and ultimately developed heavy proteinuria and declining creatinine clearance. He provided a unique opportunity to observe the effect of the loss of three quarters of the renal substance in man.

Case report

On 13 May 1964, a boy aged 3 was hit by a van and sustained laceration of the left groin, fractured ribs on the right side, diffuse abdominal tenderness, and shock. He passed a small amount of urine stained with blood, remained oliguric for one week, and required three sessions of haemodialysis. His blood urea concentration fell to 7 mmol/l (42 mg/100 ml) in October 1964. On 9 June 1964 abdominal radiographs showed extensive calcification in the left renal area and upper part of the right kidney, which persisted to a lesser degree in 1969. Intravenous pyelograms in 1969, 1974, and 1978 showed hypertrophy of the lower pole of the right kidney, which had a normal pelvicalyceal pattern. The left kidney did not function.

Renal function remained stable, and there was no appreciable proteinuria until March 1972, when the plasma creatinine concentration was 100 μmol/l (1·1 mg/100 ml), creatinine clearance 44 ml/min (56·4 ml/min/1·73 m²), and 24 hour urine protein 0·82 g (figure). Modest proteinuria persisted until June 1983, when 24 hour urinary protein was 11 g, plasma creatinine concentration 212 μmol/l (2·4 mg/100 ml), and creatinine clearance 44 ml/min (36·2 ml/min/1·73 m²).



Twenty four hour urinary protein, plasma creatinine concentration, and calculated surface area from 1969 until 1985.
Conversion: SI to traditional units—Plasma creatinine: 1 μmol/l≈ 11·3 μg/100 ml.

Hypertension, hyperphosphataemia, hyperuricaemia, and hypoalbuminaemia were not observed during regular follow up, but in December 1984 the plasma creatinine concentration had risen to 330 μmol/l (3·7 mg/100 ml) and creatinine clearance fallen to 32 ml/min (25·5 ml/min/1·73 m²).

An open renal biopsy specimen showed 25 glomeruli, of which two were totally sclerosed, seven showed segmental sclerosis with giant hyaline thrombi and occasional intraglomerular foam cells, and the remainder were large with prominent mesangial regions but otherwise normal. A few tubules were atrophic, mildly dilated, or contained protein casts. The interstitium contained a few small aggregrates of chronic inflammatory cells. Small arteries and arterioles showed mild focal hyalinisation. Immunofluorescence showed moderate quantities of IgA and Clq and less quantities of IgM and C3.

Comment

The lower pole of the right kidney became hypertrophic, and our patient grew normally throughout childhood, during which there was no proteinuria. Mild proteinuria eight years after the accident and heavy proteinuria associated with a decrease of creatinine clearance after 19 years indicated the development of progressive renal disease. Although histology was compatible with idiopathic progressive focal glomerulosclerosis, the rarity of progressive focal glomerulosclerosis and the similarity of the appearances in the three quarters nephrectomised rat suggest that the renal lesion and proteinuria were the result of extensive loss of renal substance.

Hyperphosphataemia, hyperuricaemia, and hypertension may contribute