An irregular cystic area of liver cell loss and haemorrhage (top) is seen near a terminal hepatic venule (below) (haematoxylin and eosin).

Neutrophils and eosinophils were present in moderate numbers in the portal infiltrate in addition to lymphoid cells. Serum enzyme activities remained raised, and a biopsy specimen on day 11 showed continuing features of rejection.

At day 16, a biopsy specimen showed less rejection but some canalicular cholestasis and sinusoidal dilatation. There was a single small haemorrhagic focus in the parenchyma. By day 28, more prominent, cyst-like haemorrhagic foci were present, containing remnants of the reticulin framework (figure). Sinusoidal endothelium seemed to have been partly lost in these areas, but was present elsewhere in the hepatic acini. Features of rejection in the portal tracts were now very mild, and no endothelial lesions were identified in portal venules or terminal hepatic venules (centrilobular veins). Cholestasis persisted. The patient subsequently improved, and no further liver biopsies were performed.

The patient had received no medication in the months before overdose, nor was she taking oral contraceptives. Immediately after admission she received dopamine. After transplantation she was given methyl prednisolone (0.8 mg/kg daily from day 1, changed to prednisolone on day 4), azathioprine (about 1.5 mg/kg daily from day 11), and cyclosporine (10 mg/kg daily from day 13). Mezlocillin and metronidazole were given during the first week, and amphotericin, acyclovir, and ranitidine from day 3. Doses of dextropropoxyphene/paracetamol and dihydrocodeine were given during the first week.

Vascular lesions reported after liver transplantation include endotheliitis as part of cellular (acute) rejection,⁴ and narrowing of arteries and arterioles as part of chronic rejection.⁵ We have observed venous thrombosis leading to the Budd-Chiari syndrome in one of our patients (unpublished observation). To this list must now be added peliosis hepatis. Azathioprine has been implicated after renal transplantation, but in the patients reported² the peliosis has developed months

rather than days after beginning of treatment, although it appeared earlier after treatment with anabolic steroids.¹ We cannot entirely rule out a possible role of drugs, but all the drugs given to our patient are commonly used after transplantation. The pathogenesis of peliosis remains controversial, and venous outflow obstruction, endothelial damage, and liver cell necrosis have been implicated; the presence of reticulin strands in the lesions supports these last two factors.

We suggest that in our patient the blood cysts may have been formed during a process of liver cell loss following endothelial damage. This is usually seen in portal or hepatic veins rather than sinusoids, but it is possible that the unusual location of the damage reflects the mismatching of blood groups between graft donor and recipient.

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Postoperative necrotising granulomata in the cervix and ovary

Postoperative necrobiotic granulomata in the prostate and bladder have been well documented. In the female genital tract they have, more recently, been described in the fallopian tube,¹ cervix,² and ovary.⁴ We have recently seen two such cases. In the first, necrobiotic granulomata were seen in the lower uterine segment scar of a hysterectomy specimen from a 29 year old woman who had had an uncomplicated lower uterine segment Caesarean section six months previously. In the second case, similar granulomata were present in the right ovary of a 39 year old woman who had undergone a right ovarian wedge biopsy 18 months earlier at the time of bilateral salpingectomy and left oophorectomy for lower abdominal pain. Both patients had no clinical or laboratory evidence of tuberculosis, sarcoidosis, rheumatoid arthritis, parasitic or venereal disease.

The granulomata in both cases were of similar appearance (figure). They were small in size with brightly eosinophilic staining central areas of fibrinoid necrosis around which were well defined zones of palisaded

histiocytes admixed with inflammatory cells, including plasma cells, lymphocytes, and occasional eosinophils. A few Langhans' type giant cells were also present. No birefringent material was identified in either case and staining with silver methenamine, Ziehl Neelsen, periodic acid Schiff and Gram stains was negative.

The pathogenesis of such lesions has not been well defined. No cases have so far been described in which there has not been preceding diathermy or surgery, or both, at the same site. Diathermy is particularly associated with the subsequent development of necrobiotic granulomata. The primary event may be that of traumatic tissue damage. Using x ray energy dispersive analysis, Spagnolo *et al* showed the presence of sulphur peaks in the granulomata of three out of four cases examined, but not in bladder tissue remote from the granulomata.⁵ They concluded that sulphur may have been released from damaged collagen and elastin which are rich in disulphide bands. Such damage would create tissue "foreign" to the host, thus stimulating an immune reaction of which the hypersensitivity response may at least play a part. That such granulomata are not com-

Granuloma with central area of necrosis (N) surrounded by histiocytes, lymphocytes, and occasional giant cells (arrow) (haematoxylin and eosin).

monly seen may suggest that host factors are also important.

Postoperative granulomata are generally considered to be asymptomatic. They are, however, important in other ways. Firstly, they must raise the possibility in the pathologist's mind of previous surgery, and secondly, they should not be confused with other granulomatous diseases including tuberculosis, which would precipitate further unnecessary investigation and treatment. Their occurrence in the urinary and female genital tracts may reflect both the use of diathermy and the incidence of second operations in these areas, but it seems likely that necrobiotic granulomata will be found in other sites where there has also been previous surgery.

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Traumatic aneurysm of the left common carotid artery

Traumatic aneurysms of the major branches of the aortic arch are relatively uncommon, but those of the left common carotid artery are rare indeed, there being only four cases reported to date. We present a case of traumatic aneurysm at this site and suggest a mechanism of production.

A 40 year old man collided with a car while riding a motor cycle and was admitted to hospital in a haemodynamically stable condition, but in deep coma with a large bruise on the left shoulder. A computed tomogram of the head showed generalised cerebral oedema and haemorrhagic bifrontal contusions. A chest x ray picture showed capping of the left lung apex with blood, and emergency arch aortography showed a false aneurysm of about 2 x 3 cm at the origin of the left common carotid artery with slight flow delay up that vessel. As repair of this lesion would have required aortic arch clamping a conservative approach was adopted due to the patient's poor neurological state which did not improve. The patient died seven months later.

Pathology

At necropsy there were numerous established wedge shaped areas of infarction affecting the cortex as well as white matter in the left internal carotid arterial territory associated with old organised emboli in the branches of

Figure 1 The aneurysm of the left common carotid artery was identified as the lesion (A) arising from a disruption in the vessel wall (X) of the normal left common carotid artery.

the middle cerebral artery. There were also two fresh infarcts in the middle cerebral artery distribution associated with organised thrombus which was not adherent to the vessel wall and hence was recent. The heart was macroscopically normal.

The aneurysm of the left common carotid artery was identified as the lesion (A) in fig 1 which arose from a disruption in the vessel wall (X) of the normal left common carotid artery (B). Microscopical examination of the lesion (fig 2) showed a histologically normal intima and normal medial elastic laminations.

The artery, however, had an axial intimo-medial tear where its lumen communicates with that of the aneurysm which was itself lined by organised thrombus; the latter was presumably the source of embolism. As the aneurysm is false its wall is composed of adventitia. Histological examination of the brain showed areas of haemosiderotic gliosis in the superior cerebellar peduncles, the brain stem, and the splenium of the corpus callosum (typical of shearing injury).¹

To date, 150 cases of traumatic carotid occlusion have been reported, but of these only four affected the left common carotid artery.²⁻⁴ The sites for carotid occlusion are at or just above the bifurcation (58%), between the bifurcation and the base of the skull (29%), in the carotid canal (9%), and the common carotid (4%).⁵ The mechanisms by which such occlusion may be produced are varied and four types are described.³ (1) A

direct blow to the anterior triangle of the neck; (2) a glancing blow to the head, hyperextending, rotating and laterally flexing the neck away from the side of the lesion causing the carotid to be carried upward and backward, stretching it over the transverse process of the third cervical vertebra; (3) blunt intraoral trauma; (4) basal skull fractures causing occlusion in the petrous portion.

Why the aneurysm occurred at the origin and not the bifurcation of the vessel is not clear. Congenital medial weakness may have played a part but this is not a tenable hypothesis in the light of the microscopic evidence which clearly showed all layers of the arterial wall to be normal. The hyperextension, rotation, and lateral flexion of the neck, thereby distracting the carotid artery, combined with counter traction on the aortic arch via the left subclavian artery, may have produced the localised forces necessary to disrupt the arterial wall at this rare site. Evidence that such forces occurred at the time of injury is provided for carotid distraction by the histological findings consistent with an old rotatory injury, and for subclavian artery traction by the presence of the large bruise noted on the patient's left shoulder.

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Figure 2 Microscopic section of fig 1.