SHORT REPORTS

Comparison of sulglycotide with cimetidine in short-term treatment of duodenal ulcer: double-blind controlled trial

Cimetidine is the only one among the various commercially available anti-ulcer drugs that has been studied satisfactorily.¹ Therefore trials comparing cimetidine with conventional ulcer treatments are of special interest. We report the findings of a multicentre double-blind trial to compare the ulcer-healing effects of cimetidine and sulglycotide, a drug derived from porcine duodenal mucosa² whose efficacy in the treatment of peptic ulcer in man has been reported in uncontrolled trials.³

Methods and results

Eighty-one male outpatients entered the trial after giving informed consent. All had endoscopically confirmed active duodenal ulceration. The diameter of the ulcers was from 0.5 cm to a maximum of 1.5 cm. Patients were randomly assigned to one of two treatment regimens for one month: (1) sulglycotide 100 mg in granule form together with one placebo tablet identical with cimetidine three times daily with meals, and a further 200 mg of sulglycotide with two placebo tablets at bedtime; or (2) cimetidine 200 mg with placebo granules identical with sulglycotide three times daily with meals, and cimetidine 400 mg with granulated placebo at bedtime. The patients took aluminium hydroxide tablets as required to relieve pain. The amount of antacid taken and the incidence and duration of pain were recorded. The patients were reviewed after 15 and 30 days. Endoscopy was repeated at the end of the course of treatment. Blood was taken for routine haematological and biochemical analyses and urine specimens examined before, during, and after the treatment.

Seventy-seven of the 81 patients completed the trial. Four (two sulglycotide, two cimetidine) dropped out for non-medical reasons. The two treatment groups were comparable in their ages, duration of ulcer, and ulcer size. The ulcers completely healed in 24 out of the 38 patients on sulglycotide and in 32 out of the 39 on cimetidine (table). The difference

Endoscopy findings in 77 patients with duodenal ulcer after one month's treatment with either sulglycotide or cimetidine

No of patients	Treatment	Ulcers completely healed		
		Total	Ulcer diameter	
			0.5-1.0 cm	1.0-1.5 cm
38	Sulglycotide	24 (63%)	23 (96%)	1 (4%)
39	Cimetidine	32 (82%)	27 (84%)	5 (16%)

between the two groups was not significant ($P > 0.05$). Ulcers < 1 cm in diameter healed equally well with both drugs: in 23 out of 31 (74%) patients on sulglycotide and in 27 out of 29 (93%) on cimetidine. The incidence and severity of pain and the number of antacid tablets taken decreased sharply from the first week, the effect of cimetidine being significantly greater and more rapid ($P < 0.05$). None of the healed cimetidine patients had pain compared with 8% of the sulglycotide patients ($P < 0.05$), while the percentage of unhealed patients who had symptoms was smaller with cimetidine (14%) than with sulglycotide (64%) ($P < 0.01$). There were no significant abnormalities in the haematological and biochemical findings. No untoward signs or symptoms were reported during or after the study.

Comment

This study shows that the healing effect of sulglycotide and cimetidine did not differ significantly except in very large ulcers. These did not heal well in either group, but did better with cimetidine. The proportion of patients healing with sulglycotide was similar to that reported in a recent multicentre double-blind trial against placebo (74%).⁴ Sulglycotide completely inhibits peptic activity *in vivo*.⁵ Therefore perhaps inhibition of pepsin alone allows duodenal ulcers to heal irrespective of acid secretion. The efficacy of cimetidine in treating duodenal ulcer is generally accepted. This trial shows that sulglycotide is also a drug with therapeutic potential in duodenal ulcer.

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- ¹ *Proceedings of an International Symposium on H₂-receptor antagonists*, Amsterdam 1978, ed W Creutzfeldt.
- ² Bertellini, G, Butti, A, and Piantanida, B, *Arzneimittelforschung*, 1971, 21, 244.
- ³ Butti, A, Imbimbo, B, and Becatti, R, *Clinica Europea*, 1972, 11, 401.
- ⁴ Wormsley, K G, personal communication, 1978.
- ⁵ Prino, G, Liotti, A, and Allegra, G, *American Journal of Digestive Disease*, 1972, 17, 863.

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Synovial rupture of rheumatoid elbow causing radial nerve compression

In a study of 82 patients with elbows affected by rheumatoid arthritis (RA), Hanna *et al*¹ found none with clinical evidence of radial nerve compression, while only 6 (4%) of the 152 elbows examined had some delay in nerve conduction times of the posterior interosseous branch. We describe a case of RA with synovial rupture of the elbow resulting in an acute compressive neuropathy of the radial nerve.

Case report

In 1971 a 56-year-old man had pain in the feet, which in 1976 was diagnosed as seropositive erosive RA. In 1977, when his hands and left elbow became affected, penicillamine 250 mg daily was added, the dose being increased over nine weeks to 250 mg thrice daily. In May 1978 the drug was discontinued because of thrombocytopenia (platelet count $69 \times$

Elbow arthrogram (anteroposterior and lateral views) showing extravasation of contrast from synovial cavity into surrounding soft tissue.

$10^9/l$; $69\,000/mm^3$). Two weeks later he developed painful swelling of the left forearm. Within two days he complained of pins and needles in the ipsilateral thumb and index and middle fingers.

Examination showed a tender, fluctuant swelling below the left elbow anterolaterally, the forearm being 1 cm bigger in circumference than the other. There was also diminished sensation to light touch and pinprick over the dorsal aspects of the lateral three fingers and adjacent part of the hand but no muscle weakness. On investigation the haemoglobin concentration was 14.0 g/dl , platelet count $213 \times 10^9/l$ ($213\,000/mm^3$), and erythrocyte sedimentation rate (ESR) 33 mm in first hour. The Rose-Waaler test was positive (titre 1/128). Plain radiographs of chest, hands, and elbows were normal, but those of the feet showed erosions. During arthrography of the left elbow there was prompt extravasation of injected contrast (Conray 280) into the soft-tissue swelling (see figure). Electrodiagnosis showed a normal right radial nerve sensory action potential of $20\,\mu V$ over the first dorsal interosseous muscle on stimulating 10 cm proximally; on the left, however, there was no potential visible. No denervation was found on electromyography of extensor digitorum communis.

The patient was advised to rest the arm in a sling, and 50 mg hydrocortisone acetate was injected into the elbow. A week later the swelling subsided and the forearm was only 0.5 cm bigger than the other. Penicillamine was reintroduced but not increased above 500 mg daily. Four months later, when the arthritis was well controlled and the ESR 12 mm in first hour, there was no recordable change in nerve conduction. In March 1979, 10 months after the onset of paraesthesiae, sensation was returning in the left thumb.

Comment

Rupture of synovial effusions is a recognised complication of RA and commonly occurs from the knee.² Less often it affects the elbow.³ In our patient synovial rupture was confirmed by arthrography. He experienced paraesthesiae in the hand within two days after noticing the forearm swelling. In keeping with the sensory deficit in the hand, we could not record the radial nerve sensory action potential. Absence of motor nerve disorder, both clinically and electrodiagnostically, suggested an isolated pressure neuropathy affecting the purely sensory superficial terminal branch of the radial nerve as it descends anteriorly over the lateral epicondyle of the elbow. Another explanation is vasculitis with occlusion of the vasa nervorum, which may complicate RA.⁴ Against this in our patient was the absence of digital ischaemia or other cutaneous lesions of vasculitis, usually present in such cases.⁵ Moreover, there was no evidence of neuropathy elsewhere.

¹ Hanna, B D, *et al*, *Rheumatology and Rehabilitation*, 1975, 14, 212.

² Jayson, M I V, *et al*, *Annals of Physical Medicine*, 1969, 10, 175.

³ Goode, J D, *Annals of the Rheumatic Diseases*, 1968, 27, 604.

⁴ Schmid, F R, *et al*, *American Journal of Medicine*, 1961, 30, 56.

⁵ Pallis, C A, and Scott, J T, *British Medical Journal*, 1965, 1, 1141.

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Extracranial to intracranial micro-revascularisation for the treatment of completed ischaemic stroke

Extracranial surgery for internal carotid artery occlusion has had poor results, and attention has turned recently towards the possible improvement of cerebral blood flow by intracranial procedures. Jacobson and Suarez¹ first used the operating microscope for anastomosing small vessels, and Donaghy² reported seven human middle cerebral artery embolectomies using a T tube. Yasargil and Donaghy³ then reported the first successful extracranial to intracranial bypass in man. The end of the superficial temporal artery was anastomosed to the side of a cortical branch of the middle cerebral artery. We report here the results of extracranial to intracranial revascularisation in nine patients.

Patients, method, and results

Completed strokes had occurred between one and a half and 12 months (mean six months) before surgery. The main deficits were weakness of the arm, dysphasia, intellectual deficit, and to a lesser extent, reduction in power in the leg (see table). All patients were alert and had some evidence of retained function in the affected hemisphere. All patients had low density areas comparable with cerebral infarction on computerised axial tomography. The deficits were stable at the time of surgery and were thought unlikely to show further spontaneous improvement. A power record of the affected muscles, an assessment of the speech deficit, and regional cerebral blood flow estimations were performed and repeated after the operation. Psychological assessment was carried out before and after operation; the tests used were of intelligence, verbal fluency (in the left-sided cases), visual perception (in the right-sided cases), and memory for both verbal and visual material.

The operation⁴ was performed by a vascular surgeon with experience in microvascular surgery, together with a neurosurgeon. Using the operating microscope, the end of the superficial temporal artery was anastomosed to the side of an appropriate cortical branch of the middle cerebral artery using interrupted 10/0 sutures.

All nine of the anastomoses remained patent as assessed by arteriography or directional doppler; with a patent anastomosis it is possible to feel the pulsation of the superficial temporal artery. All patients are alive and well at the time of writing, and the procedure had an insignificant morbidity; there was no skin flap sloughing and the scar proved to be neat and lie with the hair line. Cerebral blood flow and oxygen utilisation increased. The first patient of the series had striking improvement in power. Before the operation his right arm had been totally paralysed for six weeks, and within 10 days after the procedure movement appeared, first at the shoulder, then at the elbow, and finally at the wrist. At the end of six weeks his arm movements were completely normal. Three months later his inaccessibly stenosed internal carotid artery occluded and he suffered a recurrent completed stroke. His deficit on this occasion was severe but the arm was relatively spared. Most patients had a reduction in tone with improvement in power, enabling an improvement in gait and stability (see table). The results of psychological testing were summarily expressed as differences between preoperative and

Summary of results in nine patients

Case No	Age and sex	Presentation	Duration of history before surgery (months)	Clinical neurological outcome	Difference on psychological tests	
					Verbal (intelligence/fluency/memory)	Non-verbal (intelligence/perception/memory)
1	55 M	Completed stroke, R arm totally paralysed	1½	R arm returned to full power	+5/+2/+1	+2/-/+1
2	46 M	Completed stroke, weakness L arm and leg, mild dysphasia, no sensory loss, left handed	4	Improvement in power and walking ability	+1/-/+1	0/+5/0
3	59 M	Completed stroke after TIA, R arm and leg weakness, dysphasia	1½	Improvement in power and walking ability		
4	52 F	Completed stroke, weakness and in coordination L arm	9	Improvement in left arm power	-1/-1/-1	0/+8/+1
5	69 M	Completed stroke, weakness in L side of face, arm and leg	4	Improvement in power, speech, and walking ability	0/-/0	0/-/+2
6	57 M	Completed stroke after weakness in R arm	7	Improvement in power, speech and walking ability	+1/-/+2	+1/-/-2
7	67 M	Completed stroke, weakness R arm and leg, poor memory	6	No change	+3/+6/+2	-1/-/-6
8	62 M	Completed stroke, weakness R arm and leg, severe dysphasia	12	No change		
9	57 M	Intellectual deterioration, R carotid occlusion	3	Further deterioration	-4/-/-2	-4/-/0