tense, but needling did not produce any fluid, bloody or otherwise. Three burr-holes were then made on the right side, and dark blood clot and old blood were evacuated from both the subdural and the subarachnoid spaces on that side. A pronounced but only transient improvement took place in the patient's condition, and death occurred at 5 p.m. on November 25.

Post-mortem Findings.—Necropsy was performed 17½ hours after death. On opening the skull an extensive subarachnoid haemorrhage became apparent, and it was found to be present chiefly over the base of the brain, more particularly on the right side. Its source was a ruptured aneurysm 1 cm. in diameter on a branch of the right middle cerebral artery, fairly high in the Sylvian fissure. The brain was generally swollen, the right temporal lobe more than the left. On sectioning the brain the subarachnoid haemorrhage was seen to extend for about 3 cm. into the substance of the right temporal lobe from the region of the aneurysm. No other intracerebral haemorrhages were revealed. Gross hernias of the uncus and cerebellum were observed, the latter very marked indeed, so that it was only with difficulty that the cerebellar tonsils and the brain-stem could be delivered from the foramen magnum. The lower 1.5 cm. of the tonsils was swollen, discoloured, and haemorrhagic. Histological sections from these affected areas showed recent haemorrhagic necrosis, in which there was an acute inflammatory cell reaction, together with fibrinoid necrosis of the walls of several small vessels.

Discussion

The clinical diagnosis of subarachnoid haemorrhage, including episodes of unconsciousness, could be satisfactorily, if superficially, confirmed at necropsy on the basis of the findings described above. It is in trying to reconstruct the precise chronological details of these changes that problems arise.

It is probable that a potentially reversible degree of uncomplicated cerebellar coning developed early in the clinical course. However, the age of the necrotic process in the tonsils was only about 24–48 hours. This particular lesion, if precipitated by lumbar puncture, was more likely to be related to the last occasion than to the first two. The intracerebral haemorrhage, too, was of recent occurrence, but its histological details do not permit of such an accurate estimate of its age. It may have been precipitated also by the third lumbar puncture, but equally well may have arisen earlier, either spontaneously or in relation to the previous lumbar punctures. Irrespective of when and how this latter haemorrhagic lesion took place, it seems indisputable that it materially aggravated the degree of cerebellar coning already present. Schaller (1933) and Masserman (1934) pointed out that foraminal herniation is due not so much to a direct "plugging" effect following the withdrawal of cerebrospinal fluid as to oedema of the brain which develops subsequently. An intracerebral haemorrhage could be visualized easily as contributing to this. Clearly, in the present case, at least three distinct factors—subarachnoid haemorrhage, cerebral haemorrhage, and lumbar puncture—interacted in a complex manner to produce the pathological end-result described.

These speculations, academic and inconclusive though they may be, do not invalidate the general warning of the dangers of injudicious lumbar puncture in cases of subarachnoid haemorrhage. It is impossible to escape the conclusion that lumbar puncture in this case contributed at least to the cerebellar coning and necrosis, and possibly further subarachnoid bleeding. It would seem that, once the diagnosis is established by the withdrawal of evenly blood-stained fluid which leaves a xanthochromic supernatant fluid after centrifuging, there are no further indications for lumbar puncture in cases of subarachnoid haemorrhage.

Summary

A case of subarachnoid haemorrhage due to rupture of a cerebral aneurysm is described. Death occurred

after the third lumbar puncture. At necropsy, cerebral haemorrhage, cerebellar coning, and tonsillar necrosis were found. No reports of the last-named lesion can be found.

I am grateful to Professor J. H. Biggart and Dr. J. E. Morison for assistance and criticism. Mr. C. A. Calvert, F.R.C.S., kindly gave me permission to publish this case.

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ACUTE DERMATOMYOSITIS

BY

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Recent articles (Oppel *et al.*, 1950; Thorn *et al.*, 1950; Suzzman and Rudolph, 1951) have shown the value of cortisone and A.C.T.H. in acute dermatomyositis. A further case of this rare disease responding dramatically to cortisone therapy is described.

Case History

A domestic servant aged 24 was admitted to the Central Middlesex Hospital on May 14, 1952. She had been in good health till three months previously, when her fingers became stiff and swollen at times. A week before admission her left hand felt weak, and the following night a sensation of tightness developed in her throat. She could not swallow properly. When she awoke the following morning (May 10), both hands felt weak. They were not painful. The weakness and stiffness of the finger- and wrist-joints soon involved the elbows and shoulders, and later both her knees and hip-joints and her back. Her legs felt useless and her muscles became increasingly painful and tender to pressure. On the same day a bright red rash started on the dorsal surface of her hands, soon spreading up both arms, and a similar rash involved both legs. On May 12 her eyelids felt swollen and stiff and they were red-rimmed. This condition lasted only a few hours. Throughout this time she felt ill and had lost her appetite completely. She vomited on three occasions. Each movement was an effort, and she ached all over when she attempted to shift herself in bed.

Physical examination showed her to be well nourished but very ill. Her temperature was 104.6° F. (40.3° C.). Her tongue was heavily furred, and her skin was hot and clammy. There was a diffuse confluent erythematous rash on the dorsal aspects of both feet and on the upper arms. In other areas irregular red macules were present. The rash was mainly confined to the limbs, but had a few extensions on the skin of the abdomen and back. She was profoundly weak, being unable to sit up, and only with difficulty could she straighten her arms or bend her knees. All her muscles were tender on palpation and felt firm and full. The superficial reflexes were normal.

On May 15 her temperature was 103° F. (39.4° C.). The rash appeared to be clearing from the forearms and from the dorsal surfaces of her feet. However, she seemed weaker and to have become worse. She was totally unable to move herself and could swallow fluids only, as she had marked dysphagia. She had a troublesome watery diarrhoea and retention of urine developed, which required catheterization. Her general condition was deteriorating so rapidly that it was decided to give cortisone immediately, and 100 mg. was injected intramuscularly at 5.30 p.m. An hour later she could move herself in bed more freely and could bend her

knees. Next morning there was definite improvement. The temperature had fallen to 101.8° F. (38.8° C.). She could raise her arms above her head, sit up without help, and swallow without difficulty. Her muscles felt flabby and hypotonic. The knee-jerks had returned. The rash was fainter. Cortisone, 100 mg. twice daily, was given.

Improvement continued, and within four days the temperature had fallen to 98.6° F. (37° C.), the rash had almost completely faded, and her general condition had improved so greatly that cortisone was discontinued. The following day she had a partial relapse. The muscles became more tender and tense and she had two rigors, the temperature reaching 102° F. (38.9° C.). Cortisone, 25 mg. six-hourly, was recommenced.

On May 21 the rash reappeared on the back of both hands and the front of both arms. It was bright red and confluent. Her limb movements, however, were now quite free and her muscles were not tender. The rash became more extensive for the next two days. On the elbows and knees it was confluent, the flexures were pinky red and the eyes were red-rimmed. It then began to fade once more, and was last seen on May 6, when only small erythematous areas on the left forearm were noted.

On June 3 cortisone was discontinued. A.C.T.H. gel, 40 I.U. twice daily intramuscularly, was begun. For two days after the change there was a recurrence of slight muscle stiffness, and on June 5 the temperature rose to 102.4° F. (39.1° C.). However, the patient became rapidly stabilized on the dosage, and on June 10 she was allowed up.

On June 17 the A.C.T.H. dosage was reduced to 40 mg. once daily, and ten days later it was discontinued altogether, the patient having then had 1,500 mg. of A.C.T.H. and 2,200 mg. of cortisone in 43 days. Her history was now one of gradual but continued improvement. Muscle power increased steadily. At first inclined to shuffle along and to tire easily on walking, she walked normally on discharge. She could write distinctly and effortlessly. On July 8 she was discharged. There were then no abnormalities on physical examination.

The patient returned to her home in Ireland two days after discharge from hospital and has written to me on several occasions since. She has remained well, but complained of some weakness and stiffness of her wrist and finger-joints about six months after her return. As this continued she was admitted in April, 1953, at my request, to St. Laurence's Hospital, Dublin, under care of Dr. H. J. Counihan, who wrote to me that the changes present were like those of rheumatoid arthritis and that, apart from these changes in her fingers, her general condition was satisfactory. She was discharged from hospital again in May, 1953, and has been receiving injections of cortisone. According to the patient, her fingers and wrists have become fully mobile again—so much so that she is returning to London to recommence work shortly.

Most of the significant investigations are summarized in Table I.

TABLE I.—Blood and Urine Investigations in Case of Dermatomyositis

Date	Hb %	Leucocyte Count	Seg. Polys. (%)	Staff Cells (%)	Juvenile Polys (%)	Myelocytes %	Eosinophils %	Lymphs %	Monos %	Creatine Excretion (mg./24 hrs.)	E.S.R. mm./hr.	Westergren
14/5/52		9,600	90	1	—	—	4	5	—	—	—	33
15/5/52		Cortisone begun										
16/5/52										710		
18/5/52										376		
21/5/52	69	59,800	55	34.5	2.5	3.5	—	4	0.5	40	23	11
27/5/52												
30/5/52		13,000	82	5	—	—	—	13	—	—	21	11
3/6/52												
10/6/52										7.3		
11/6/52												
24/6/52											7	
27/6/52		Hormones (A.C.T.H.) discontinued										
8/7/52	90	5,300	71	2	2	—	—	22	3	—	4	

A muscle biopsy was carried out, and Dr. Walter Pagel reported as follows: "Fragments examined are fat tissue with some haemorrhages and peripheral muscle, with some occasional hyalinization, but without any regenerative or inflammatory cellular changes. The nuclei are well stained throughout." It is possible, however, to obtain an indefinite or negative biopsy report on material obtained from one muscle while material from another shows typical changes (W. Pagel, 1952, personal communication). Suzzman and Rudolph's case also lacked a positive biopsy report, but was in every way a typical acute example of the disease.

Not much emphasis has been laid on the quite frequent occurrence of a polymorphonuclear leucocytosis in acute dermatomyositis, but cases besides the present one showing this feature have been described (Table II).

TABLE II.—High Leucocyte Counts Found During Course of Acute Dermatomyositis

	White-cell Count	Polymorpho-nuclears	Primitive Polymorphs
Keil (1940)	70,000	With polymorphonuclear leucocytosis	
Holmes (1948)	63,600	89%	
Oppel <i>et al.</i> (1950) ..	64,600	60%	34%
Suzzman and Rudolph (1951)	31,000	81%	5%
Clein (1953)	59,800	55%	40.5%

Summary

A case of acute dermatomyositis in a young woman is described, the diagnosis being made on the classical clinical picture presented, despite the absence of definite histological proof. Prompt remission following the use of cortisone was noted, with partial relapse when it was discontinued too soon.

Emphasis is laid on a high polymorphonuclear leucocyte count during the course of the disease.

Treatment with A.C.T.H. or cortisone is believed to be the method of choice in acute cases.

I should like to thank Dr. R. A. J. Asher for his encouragement and permission to report this case, which was treated under his care; Dr. W. Pagel for carrying out the pathological examination and for advice; Dr. D. A. Brewerton for many helpful suggestions; and Dr. H. J. Counihan for reinvestigating the patient and forwarding his helpful report to me.

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The Wellcome Film Unit has produced two new 16 mm. films entitled "The Dissection of a Mosquito for Malaria Parasites" and "Pinning Mosquitoes," both in colour with sound. The first is a demonstration by Mr. P. G. Shute of the Medical Research Council's Malaria Reference Laboratory. After showing the phases of the life cycle of the malaria parasite within the mosquito, the film demonstrates how the salivary glands of the insect are pressed out and prepared for examination so that the sporozoites can be identified. The removal of the stomach and the detection of oocysts are then demonstrated. (Running time, ten minutes.) The second film shows how mosquitoes should be pinned, how to stage and label the pinned insects for permanent display, and how to pack them for dispatch through the post. It was produced in collaboration with the Department of Entomology at the London School of Hygiene and Tropical Medicine. (Running time, five and a half minutes.)