Kenney (1943), Edwards (1947), and Gross *et al.* (1951) all implicated an altered clotting mechanism, due to various factors, as responsible for thrombophlebitis migrans in this syndrome.

It has been suggested that estimation of the coagulation power of the blood may be an aid in the diagnosis of cancer (Bock and Ransche, 1926). The results, however, are contradictory (Van Allen, 1927; Perlmann and Rodin, 1927). The reports of these authors were prior to the use of anticoagulant therapy. The failure of anticoagulant therapy to control these thrombotic episodes even with dangerously low prothrombin concentration indicates that altered blood coagulation is not an adequate explanation.

Raynaud's phenomenon associated with malignant disease is rare. Bennett and Poulton (1928) suggested that in their case it was due to a malignant gland invading the right inferior cervical ganglion. Raynaud's phenomenon, however, was bilateral. In Case 5 no secondary deposits were found above the diaphragm.

Coman (1953) discussing blood-borne tumour metastases, points out that direct dissemination of cancer cells depends mainly on their entrance into the vascular channels, and that the size of the emboli vary from single cancer cells to fairly large clumps. It is also known that cancer cells have amoeboid properties and can invade tissues, including the walls of the veins.

It is suggested, therefore, that both thrombophlebitis migrans and Raynaud's phenomenon, when associated with malignant disease, are due to emboli of malignant cells. The size of the emboli determines the site in which they settle: in the lungs, causing pulmonary infarcts; in the digital arteries, resulting in Raynaud's phenomenon; or in the peripheral veins, causing thrombophlebitis. In most cases the emboli are small, and the formation of a thrombus around the malignant cells results in the death of these cells. This is the commoner event. Occasionally, this does not occur, and a metastatic deposit can then be observed to develop.

Summary

Four cases of thrombophlebitis migrans associated with carcinoma are reported. In three cases the primary growth was in the lung; in the fourth it was in the head of the pancreas.

One case of Raynaud's phenomenon with carcinoma of the stomach is reported.

Attention is drawn to the fact that, when thrombophlebitis migrans is not adequately controlled by anticoagulant therapy the possibility of an underlying carcinoma must be considered.

It is suggested that the mechanism is due to emboli of malignant cells and that there is no underlying alteration in blood coagulation.

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FACIAL DIPLEGIA IN THE GUILLAIN-BARRÉ SYNDROME

BY

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The Guillain-Barré syndrome, or acute infective polyneuritis, has aroused much interest in recent years. It is still a matter of discussion whether or not it is a disease *sui generis*, and recent opinion seems to favour a diverse aetiology for the syndrome. Laurans (1908) published the first adequate review of the condition, emphasizing the widespread flaccid paralysis and the facial diplegia. Guillain *et al.* (1916) did not recognize the facial diplegia as part of the syndrome, and it was not until 1936 that Guillain admitted the frequency of this form of paralysis. Holmes (1917) described cases in soldiers which were obviously of this syndrome, and Bradford *et al.* (1919) published a series which included 17 cases with involvement of the seventh nerve. Petch (1949) gives an excellent review of the subject, stating that the aetiology is unknown and the infective nature unproved. He described eight cases, in two of which bilateral facial paralysis (facial diplegia) had occurred. Bannwarth (1949) regards the condition as a parallergic process which may occur in a great variety of diseases, and Shaby (1949) also takes the same view of its multiple aetiology. Other authors hold the opinion that it is primarily an infective process of unknown, probably virus, aetiology—hence the name “infective neuroitis” used in American literature. McIntyre (1937) and de Jong (1940) indicate three groups—acute cases ending in recovery; cases running a protracted course, often with development of permanent paralysis; and cases terminating fatally, usually with bulbar paralysis.

The present report includes six cases, all females, who developed bilateral facial paralysis. Four of these were seen before paralysis of the face or body had developed, and the principal reason for reporting them is the observation, not hitherto recognized, that in the early stages the deep tendon reflexes are exaggerated. It is maintained from observation of these cases that it is possible to diagnose the condition in the pre-paralytic phase, and in three of the cases the development of facial diplegia was forecast before the onset of paralysis. Diagnostic points in the early stage of the disease are intense paraesthesiae, beginning in the feet, hands, and limbs, and spreading in severe cases all over the body. One patient said she felt as if she had “passed through fire.” At the same time, pain is experienced in the muscles of the limbs, mainly in the proximal groups; pain in the eyes is common; and on clinical examination exaggeration of the deep reflexes is present, with no objective sensory change, and there is muscle tenderness on pressure; later the deep reflexes disappear and varying degrees of paralysis ensue. Four of these six cases had a preceding febrile illness six to thirteen days previously. A differentiating point from poliomyelitis is the widespread tenderness, which is bilateral and symmetrical, and as a rule principally affects the proximal groups of limb muscles.

Case 1

A married woman aged 36 was seen at home on July 20, 1948. Six days previously she had developed a right facial paralysis with inability to taste. Her eyes were aching, and

she had had a headache and pain in the back of the neck and shoulders. Her legs felt sore to the touch and her bowels had not been opened for five days. She had found difficulty in talking. There had been no sore throat. On the morning of the 20th she developed a left facial paralysis, which was probably the reason for her difficulty in speaking, although she herself had not noticed that the left side of the face was paralysed.

On clinical examination there was bilateral facial paralysis with inability to close both eyes. She could not raise her legs from the bed, and dorsiflexion of the feet was poor. The arms could not be raised and there was a poor grip in both hands. Muscle tenderness was pronounced in the thighs, in the anterior tibial group, in both calves, and in the trapezius and deltoid on both sides. The deep reflexes were absent in all four limbs: there was no apparent sensory loss or any neck rigidity. Examination of the eyes showed full eye movements, but the pupils did not react to light or on accommodation.

She was admitted to hospital, where a lumbar puncture on July 22 showed: pressure, 140 mm.; protein, 120 mg. per 100 ml.; no cells. A blood count was normal. She had a persistent fever until August 10; it then gradually settled. The reflexes returned to the arms in a few days, being present on July 23, and by the end of September the knee-jerks had also returned. Within a month the right side of the face had recovered, although the left side was still weak. A further lumbar puncture on August 12 showed protein, 40 mg. per 100 ml., and 80 lymphocytes per c.mm. She eventually made a complete recovery, and has had no further neurological symptoms since.

Case 2

A married woman aged 27 was admitted on November 23, 1948. Thirteen days previously she had had a febrile attack with a little temperature, and felt ill. Six days before admission she had felt pain between the scapulae, which became worse over the next two days; she then developed severe headache, and this had persisted up to the time of examination. She had had tingling in the legs three days before admission, and on the morning of the 23rd she woke with the left side of the face paralysed. There was numbness down the left side of the body, more marked in the left leg, and she said the bedclothes felt cold.

On clinical examination she was nervous, with a heart overacting at 100, and had a complete left facial palsy and a partial right facial palsy, affecting the upper half of the face. There was no obvious motor or sensory loss at this stage, but the thigh muscles and shoulder-girdle muscles were tender on pressure. The left knee-jerk was brisk and the right normal. Both ankle-jerks were absent. The plantar reflexes were flexor.

She ran a temperature up to 100° F. (37.8° C.) for three days. A lumbar puncture on November 29 showed protein 50 mg. per 100 ml., and no cells. A blood count was normal. No further paralysis developed, but the facial paralysis persisted, and, although there was a gradual recovery, she still had weakness of the left side of the face in July, 1949. In February, 1950, she was still conscious of a little weakness of the left angle of the mouth.

Case 3

A married woman aged 35 was admitted on February 21, 1949. She had had an occipital headache for a fortnight, and four days before admission had had pains in the legs, spreading up into the back and into both arms. She had had tingling in the feet and legs, and for two days had repeated vomiting.

When first seen she was tender in the muscles of the calves and thighs and in the deltoids; the right knee-jerk was absent, but the other deep reflexes in the arms and legs were brisk. A lumbar puncture on February 22 showed normal pressure, 140 mg. protein per 100 ml., and 15 cells, all lymphocytes.

On February 28 she complained of numbness in the arms, her hands felt weak, the arm reflexes were now absent, and the left knee-jerk was also sluggish. All arm muscles were tender on pressure. Three days later she developed a bilateral facial palsy, more pronounced initially on the left side, but by March 7 it was complete. All her deep reflexes were now absent. She had severe pain in the arms and legs, with loss of power in the shoulder-girdle, pelvis, and thigh muscles. At the end of March her facial movements were returning and the limb movements were stronger. In April her deep reflexes gradually returned and she made a complete recovery. A year later, however, she still complained of some numbness in the feet.

Case 4

A married woman aged 21 was admitted on October 4, 1950. Six days previously the tips of her fingers and toes felt numb, and this gradually spread over the body. Movement of any muscle caused pain. Three days later she had bitemporal headache with a feeling of pressure on the eyes. She could not open the mouth properly, and had some photophobia. She vomited on the day of admission. There had been no preceding sore throat or febrile illness.

On examination she had marked tenderness in the muscles of the arms, forearms, thighs, and calves, and in the masseters and temporal muscles. She was unable to open her mouth or to articulate properly. Limb movements were normal, with no apparent loss of power, although movements were painful. The triceps-jerks and biceps-jerks were absent, and at this stage the knee-jerks and ankle-jerks were both brisk; the plantar reflexes were flexor, and there was no sensory loss to pinprick or to cotton-wool or vibration sense. The optic fundi were normal, but she had pain on trying to look up. Her facial movements, apart from inability to open her mouth, were normal. A blood count showed nothing abnormal.

The following morning there was weakness of the left side of the face; this became complete by the evening, and the next morning there was bilateral facial palsy. She rapidly developed weakness of the arms and legs, the pain in the limbs increased, and by the end of the third day after admission, coincident with the onset of paralysis, the deep reflexes disappeared. With this widespread paralysis there was fear that diaphragmatic paralysis might ensue, so she was transferred to the isolation hospital.

Within 24 hours diaphragm movements became weak and she was placed in an iron lung, but the movements returned within 48 hours and she was taken out of the lung. The paralysis of the limbs, however, persisted.

She was readmitted to Harefield Hospital on October 16, 1950. The muscle tenderness was now very much better and she was feeling well in herself, but said that her hands felt like "two wet fish." She was still unable to chew, but could swallow with no nasal regurgitation. She now had some loss of light touch sensation and to pin-prick over her finger-tips.

By October 23 movements began to return in the arms and the lower lip. On November 1 there was a slight movement of the right side of the face. By the 14th she could use her limbs and could feed herself. From this time recovery was rapid, and by December 18 muscle recovery was complete, except for slight weakness in the left upper facial muscles. Within a month this had also recovered completely.

She was subsequently seen at intervals over the next two years, when there was no sign of recurrence.

The C.S.F. findings were of interest: October 5: cells less than 1; protein, 190 mg. per 100 ml.; chlorides, 740 mg. per 100 ml. November 6: red blood cells, 3; white blood cells, 1; protein, 240 mg. per 100 ml.; sugar, 72 mg. per 100 ml. November 21: pressure, 200; 2 lymphocytes, 3 red blood cells; protein, 210 mg. per 100 ml.; Lange 2111000000. December 4: clear colourless fluid; pressure, 160; 3 white blood cells; protein, 300 mg. per 100 ml.; Lange 1221111100.

Case 5

A married woman aged 47 was seen at home on July 16, 1952. Thirteen days previously she had a febrile illness diagnosed as influenza. Ten days later diffuse irritation occurred all over the body. The following day her eyes hurt on movement, and that night she had pain in the arms and legs—it felt as if it was “in the bones.” The following morning she still had the pain, and found she could not read small print. During the course of the day the irritation of the skin increased. She felt “as if she had passed through fire.” Trembling fits occurred which she could control voluntarily.

On the morning of July 16 she had diplopia, which had persisted. She had had retention of urine for seven hours, but later passed urine with difficulty. Her appetite was poor and she vomited once.

Clinically she complained of diplopia on looking to the left and blurring of vision on looking to the right, but there was no obvious ocular palsy or nystagmus; the optic fundi were normal, and the visual fields were not restricted. Her speech was normal, but her doctor and her husband commented that cerebration was slower. There was no loss of power in any limb, but the muscles were tender on pressure in the thighs, calves, and arms. All her deep reflexes were exceedingly brisk. The plantar reflexes were flexor. Apart from a doubtful loss to pin-prick sensation on the legs, there was no sensory change. The heart rate was 130 and regular, the heart sounds were normal, and the blood pressure was 135/95. A blood count was normal, and also the urine.

She was admitted to hospital, where she was found to have a raised C.S.F. pressure, 90 mg. of proteins per 100 ml., and fewer than 5 cells per c.mm.

Two days after admission she developed generalized weakness of all limbs, together with poor diaphragm movements, and was transferred to the isolation hospital. Her deep reflexes became sluggish, but did not disappear, and at the same time she developed bilateral facial palsy. The respiratory paralysis did not develop, and she was transferred to Harefield Hospital. A further lumbar puncture on July 27 showed: protein, 80 mg. per 100 ml.; 1 lymphocyte per c.mm.; chlorides, 750 mg. per 100 ml.; Lange 0000000000. She improved rapidly, and was discharged on August 5, having completely recovered all facial and limb movements.

Case 6

A married woman aged 42 was admitted to the isolation hospital on July 30, 1953, with a history of pain behind the eyes and left facial palsy for two days. Six days previously she had had some pain in the back with general malaise. On the day before admission she developed a right facial palsy also. She had tingling in the hands and feet, but no other symptoms. On admission there was bilateral facial palsy, eye movements were full and normal, and muscle power was normal in all limbs. The ankle-jerks were absent, but knee-jerks were brisk. Within two days all her deep reflexes except the triceps-jerks disappeared. Lumbar puncture showed 110 mg. protein per 100 ml., and 1 polymorph cell only. Her blood count was normal.

She was transferred to Harefield Hospital on August 4. By this time, however, her deep reflexes had returned, though they were sluggish, but the ankle-jerks were still absent. Repeat lumbar puncture on August 17 showed 65 mg. of protein per 100 ml., and no cells. Recovery of facial movements was very slow, and she was discharged home on September 12 with a little but still incomplete movement of the face.

Treatment

No specific treatment was given other than physiotherapy when the acute stage had subsided.

Summary

Six cases of the Guillain-Barré syndrome are described. All had facial diplegia. In four of them, exaggeration of deep reflexes was observed in the pre-paralytic stage. Paraesthesiae and bilateral muscle tenderness followed by widespread paralysis of limbs were a feature in five of the cases.

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Medical Memoranda

Sudden Simultaneous Arterial Embolism Involving All Four Limbs

The following case is unusual enough to merit recording.

CASE REPORT

A man aged 58 was admitted to hospital complaining of difficulty in passing water. There was a history of a urethral discharge some 30 years previously, followed by a course of injections in the buttock, but no history of venereal sore. He had remained well until some 12 months before admission, when he noticed progressive difficulty in passing water. The stream got smaller and smaller and eventually stopped. At this point he attended a V.D. out-patients department, where a diagnosis of urethral stricture was made and his urinary retention was relieved after the passage of a curved sound. A blood W.R. taken at this time was found to be positive (strong). He did not attend for further treatment until retention occurred a second time some six months later. Instruments were again passed, after which he voided about 18 oz. (510 ml.) of urine loaded with pus. He was admitted to hospital the following day.

Examination revealed a rather pale ill-looking man. The presence of a stricture in the bulbous urethra was confirmed instrumentally; slight bleeding followed the passage of a medium-sized curved sound. Shortly afterwards, prior to a more detailed physical examination, he had a syncopal attack when paying a visit to the toilet. Loss of consciousness was brief: he was seen by a medical officer within a few minutes and ordered strict rest in bed, warmth, and copious fluids. During the afternoon his temperature rose to 102° F. (38.9° C.), but his general condition was described as satisfactory.

The next day he seemed to have recovered from his syncopal attack. There was hardly any urethral bleeding, and urine was passed quite satisfactorily. He was ordered to remain on strict rest in bed and to take copious fluids. His condition was satisfactory for the next five days, but the following night he complained to the nursing staff that his limbs had suddenly become painful, and on examination they were found to be discoloured.

Examination early the following morning showed a very ill-looking man with a dry brown tongue and labial herpes. Both hands and both forearms were cold and cyanosed and completely pulseless. Arterial pulsation could be felt in both antecubital fossae, but none at all below this level. Both legs were similarly cold and pulseless: the skin of the right leg was mottled blue and white; mottling was not present