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A CASE OF RIGHT ABDUCENS PARALYSIS FOLLOWING ACUTE MASTOIDITIS—GRADENIGO'S SYNDROME.

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This case is reported, not because of its being unique, but rather because of its being a typical example of that group of symptoms and functional disturbances which Gradenigo has described, and which are known to us as Gradenigo's syndrome. The principal manifestations are purulent discharge from the ear, abducens paralysis, neuralgia, pain over the frontal and parietal regions. As this was the first case of the kind in my experience, I searched the limited amount of literature at my disposal, in the hope of finding some explanation for its occurrence, and was rewarded by finding a most excellent article by Dr. Harold Hays, which he read before the New York Academy of Medicine, Section on Otol-ogy, in February, 1913, giving an excellent digest of the sub-ject up to that time.

Dr. Hays reports Dr. Chas. E. Perkins as having had six cases during the winter of 1913, and that he had collected ninety-five from the literature. He also reports a most inter-esting case from the pen of Dr. John R. Page, seen in the service of Dr. Robert Lewis at the New York Eye and Ear Infirmary.

This case was particularly interesting, as the signs and symptoms were indicative of quite a grave condition—pain over right side of head, nausea and vomiting for six days, free purulent discharge from canal, temperature varying from 100° to 105°, slight traces of albumin in urine, slight papil-litis in right eye and diplopia, developing six days previous. Despite these symptoms there was no mastoid tenderness at any time, but mastoidectomy was performed on account of

the sixth nerve paralysis, the high temperature, peculiar symptoms, etc. "The mastoid was found to be quite sclerotic, with only a few fairly large cells which were filled with granulations. No free pus was found; the sinus was exposed throughout a great part. No dura was exposed. The antrum was opened and curetted out, the tip was completely removed." Lumbar puncture was performed on the table, and fifteen cubic centimeters of clear fluid was withdrawn—this was free from albumin, and the culture from it was negative after twenty-four hours. The following day temperature was 101°, discharge lessened, patient reports diplopia less marked. There is a well marked nystagmus on rotation of eye to left. Patient went on to speedy recovery.

Quoting further from Dr. Hays' article, Perkins in his paper remarks that intense headache, "especially in frontal region, severe pain in eye or deep in orbit, neuralgia of the fifth nerve, or paralysis of some of the muscles supplied by it, are symptoms, one or more of which have been noticed in fifty-five out of the ninety-five cases reported." This means that there is some interference with the Gasserian ganglion by the process going on at the apex of the petrous bone.

Dr. John Dunn has kindly allowed me to append brief notes of three cases he has seen, as follows:

Case 1.—Male, aged seventeen years. Mastoiditis, right side, with sinus involvement. Paralysis complete of the right external rectus, appearing after operation and entirely disappearing after a few weeks.

Case 2.—Girl, aged twenty years. Radical mastoid. There appeared the day following operation, or first noticed at that time, a right internal squint. There was no loss of motion of the right external rectus, but the right eye turned in toward the nose whenever both eyes were used for usual purposes. This condition was still present three or four years after the operation. Dr. Dunn has doubts about this being a typical case, and thinks it might have been an old squint of childhood which had reappeared as the result of the chloroform and bandaging of the right eye. He had seen a similar occurrence in a young man operated on for pterygium, the operator having bandaged the eye after the operation. In this case the refractive error was high, while in his own case it was only 1.0 D.

Case 3.—Female, aged twelve years. Simple mastoid operation; complete paralysis of external rectus of corresponding side. First noticed paralysis three days after operation. When last seen, about one month after operation, the paralysis had practically disappeared. This disappearance was gradual.

In none of Dr. Dunn's cases were there present any symptoms of involvement of the Gasserian ganglion. He mentions the well known fact that herpes zoster ophthalmicus often occurs as a complication of paralysis of one or more eye muscles; more often involving the third than the abducens, however.

Many authors offer testimony of the involvement of the eye muscles and even the facial as a complication of zoster. There was in the case I report, however, no symptoms of this trouble except the intense neuralgia.

My case is as follows: On October 7th, N. D., female, white, aged eight years, was brought to me suffering with acute catarrhal otitis media of the right ear. She had had some pain the previous night, but was comfortable when I saw her. The membrane was pinkish but not bulging, and I advised a cathartic and rest in bed. The child was pale and badly nourished, and gave a history of having had one or two previous attacks of earache at twelve month intervals. The membrane, however, had never ruptured, and the attacks only lasted for three or four days. The tonsils were hypertrophied, ragged and diseased, and there was quite a good sized adenoid hypertrophy; despite this the mother said the child had never had difficulty in breathing. I advised her, however, to bring her back in two weeks, in case she had no trouble with the ear before that time, and I would then remove the tonsils and adenoids. The earache never reappeared, and in about two weeks I removed the tonsils and adenoids under ether, as the ear had apparently become entirely normal, both in appearance and function. The following day patient left the hospital and the same night began to suffer again with pain in right ear. Two days after this I was called, as the pain had not ceased and the temperature was 102°. I found the membrane bulging, and a free paracentesis was done. This relieved pain and a free discharge began.

There was slight tenderness over right mastoid. The after-

noon of the next day the temperature was again 102° , and tenderness was increased. I then sent patient to hospital and opened the right mastoid. No free pus was found, but there was a large cell above and behind the antrum filled with granulation tissue, and the whole cellular structure of the mastoid was beginning to show granulation and was soft and very vascular. The mastoid cells were cleared out and the entire tip removed; the sinus outlined and dura exposed over a small portion of sinus where the inner plate was softened. The sinus looked healthy, and no further bone was removed. The wound was sutured above and packed with iodoform gauze. The operation was performed at noon on October 28th. From this time on for five days patient made rapid improvement, slept well, temperature varying between 99° and 101° , the latter being the highest recorded at any time during her post-operative period. The pulse ranged between 90 and 115, generally about 90.

On the night of November 4th the patient showed great restlessness and was fretful; the temperature, however, showed no important rise, being only 100° , pulse 106, respiration 24. The first dressing had been given on the previous day, and I felt that the restlessness was possibly due to some discomfort from that, so disregarded it.

November 5th. Patient very restless, fretful and nervous, and complaining of great pain over supraorbital region and in right eye. Wound dressed, sutures removed—found everything in good condition. Had blood examination made for malarial parasite on account of the neuralgia. This was negative. Aspirin, grains two, controlled headache. Paroxysmal neuralgic attack in brow and extending to right ear recurred about 1 a. m., lasted a short while, and patient slept latter part of night; and from this time to afternoon of next day, patient was bright and comfortable. At 6:30 p. m., however, she had a slight return of pain, which quickly subsided under two grains of aspirin.

November 7th. No discomfort in forenoon, but return of neuralgia, and quite severe, at 1:30 p. m., lasting at intervals until 9 p. m., despite aspirin.

November 8th. Patient comfortable, wound dressed; temperature, normal; pulse, 88; respiration, 20. Allowed to sit up.

November 9th. Patient had a good day and night. Temperature, normal. Patient allowed to go home.

From this time the patient was brought to my office every other day for dressing, and her mother reported that while she had had no rise of temperature or other signs of trouble, her neuralgia came on more or less severely every afternoon. The child's appearance was not good, and she showed the effect of suffering, which was so great at times that it became necessary to use one-eighth grain of codein with the aspirin to obtain relief.

On November 26th, nearly one month after the operation, and more than three weeks after the neuralgic symptoms appeared, she complained of diplopia, and I discovered that there was complete paralysis of the external rectus muscle of the right eye. On account of this unusual and, to me, alarming occurrence, her persistent headaches and her ill appearance, I feared a cerebral complication, and brought her again to the hospital. There an exhaustive neurologic examination was made by Dr. William B. Newcomb, without throwing any light on the trouble. The sensory and motor conditions were perfect, reflexes normal, eye grounds and pupillary reflexes normal, the only abnormality being the right abducens paralysis and the affection of the fifth nerve. Lumbar puncture was done, draining off several cubic centimeters of perfectly clear fluid, under no pressure, which proved sterile. Blood test showed no great leucocytosis. Temperature, 98°; pulse, 88; respiration, 22. Patient was given aspirin, grains two, every four hours; urotropin, grains five, twice daily. Codein sulphat, grain one-eighth, p. r. n., to control neuralgia.

She was very bright between the paroxysms of pain. Temperature remained normal, and under the treatment, supplemented by syr. ferri. iodid, her attacks grew less severe and frequent until November 23rd, when they disappeared altogether and she was looking very much better. She was allowed to return home on November 27th. There has been no recurrence of the attacks, the paralysis has gradually improved, and at this time is almost unnoticeable. The child is rosy and well.

The etiology of this peculiar and alarming train of symptoms, especially alarming when occurring in conjunction with

mastoiditis or following operative procedure for the same, is unfortunately obscure. We know the general etiology of acute acquired paralysis of the ocular muscles to be either intoxication, infection or traumatism. I am inclined to the belief that intoxication or toxemia is the underlying cause of these cases, together with traumatism when the mastoid is sclerotic, and considerable force must be applied by chisel and mallet to open the bone. In my case, however, no force was necessary, as the bone was soft and the principal work was done with curette.

We are all familiar with the fact that ophthalmoplegia externa occurs in lead poisoning, alcoholism, ptomaine poisoning, etc. It has also occurred in migraine—here the symptoms of neuralgia almost always precede for a long period the paralysis of the ocular muscles. The intoxication theory at least seems to fit my case as a general etiologic cause, but just why the fifth and sixth nerves should be the only ones affected in this class of cases is to me most obscure. If we are willing to admit traumatism as the cause, the explanation would be simpler, as the Gasserian ganglion and the sixth nerve are very close together at the apex of the petrous bone, and an effusion in this region from traumatism could explain the involvement of the two nerves. The purulent otitis media, the third symptom of the syndrome, could only affect these nerves by direct extension to the dura or brain at the apex of the petrous bone. However, the mildness of the nervous symptoms in my case would certainly seem to exclude this, or it must affect them indirectly by the toxemia produced. Had there been evidences of herpes zoster ophthalmicus, I should have better understood the paralysis of the abducens. I admit that this may have been a typical case of zoster, but as I believe that toxemia is a great factor in this, it brings me back to the same conclusion as to etiology.

Gradenigo's conclusion is that from some cause a circumscribed leptomeningitis is produced at the tip of the petrous bone. Lubet Berbon objects to the theory of intracranial lesion, and believes its origin to be reflex. Bonnier concurs in this opinion. Many celebrated authorities believe that sinus involvement or intracranial lesion of some kind probably exists, among these Habermann and Jurgensmeyer. Urbantschitsch rather holds to this theory, but admits possibility of

reflex cause. Spira has observed it following la grippe. Schimanowski reports a case in the course of a diffused external otitis. Geronzi believes it due to toxemia. Finally, Baratoux, in the *Archives of Laryngology*, after a detailed historic study of the question, concludes that it is reflex in origin. The advent of these symptoms into a case one has operated upon for what seems to be an ordinary one of simple mastoiditis, must necessarily be somewhat alarming, and a wider study and discussion of this subject will, I am sure, not be without profit to every otologist.