Acute Parotitis Associated with Depression and
Psychoactive Drug Therapy

David H. Rosen

ACUTE SUPPURATIVE PAROTITIS was first distinguished from mumps in 1834 by Brodie. Before the discovery of antibiotics it commonly occurred postoperatively or in debilitated states and had a high mortality rate. Following the introduction of antibiotics there was a striking reduction in both incidence and mortality rate. Acute parotitis had become so rare by the 1950’s that Robinson called it a “vanishing disease” but during the late 1950’s it reemerged. The 1960’s have seen a continued rise in the incidence of acute parotitis, whereas the mortality rate, although still alarming at 23% to 35%, has not increased since the mid-1940’s.

The increased incidence of acute parotitis appears to be related to the increase in the proportion of the population that is elderly, especially those in nursing homes because of chronic illness and the emergence of antibiotic-resistant bacteria. The disease has been associated with malnutrition, oral infection, and poor oral and systemic hydration. Xerostomia, with diminished salivary secretion, and poor oral hygiene predispose to the infection. The offending organism is usually Staphylococcus aureus. Acute pyogenic parotitis is characterized by sudden onset, local pain and swelling, and is sometimes accompanied by fever and chills. Other causes of acute parotitis include mumps, blockage of Stensen’s duct by a calculus, diabetes mellitus, alcoholic cirrhosis, and drug hypersensitivity reactions. Bilateral parotid enlargement has been reported in a patient taking thioridazine. Ragheb reported acute pyogenic parotitis in three geriatric patients, all of whom had dryness of the mouth as a side effect of prolonged use of one or more of the following psychoactive medications: triflupromazine, chlorpromazine, amitriptyline, and benztropine mesylate.

This article was stimulated by (1) finding only one report in the literature associating psychoactive drugs and parotitis, (2) finding no previous reports of the etiologic significance of severe depression and its vegetative components, and (3) the significance of this complication in view of the fact that depression is common and the use of antipsychotics and antidepressants is widespread and increasing.

CASE REPORTS

Case I: A 37-year-old white female came to the emergency room of a general hospital complaining of pain behind the right ear. She returned two days later acutely distressed because her boyfriend of eight years had suddenly left her the night before. She was hospitalized on a psychiatric ward, appeared very depressed, and was tearful. She gave a history of one week of depression...
that had worsened two to three days prior to admission. She was oriented to time, place, and person. Her memory was intact and her attention span was normal. She described auditory hallucinations of family members speaking to her, and had the delusion that someone was going to kill her. The diagnostic impression was severe acute depression in a person with chronic schizophrenia of the schizo-affective type. She was placed on suicidal precautions because of statements like, “I want to kill myself—there is no future.”

She had been taking the following medications daily for approximately one year: amitriptyline (Elavil) 300 mg., trifluoperazine (Stelazine) 60 mg., benztropine mesylate (Cogentin) 4 mg., and methylphenidate (Ritalin) 15 mg. These were continued at the same dosage on admission. Previous psychiatric hospitalization included a two-week stay thirteen months before on the same ward. Her diagnosis and condition had been essentially the same at that time. Her past medical history included no allergies and no history of diabetes mellitus or cirrhosis, but there was a positive history of mumps as a child and of hepatitis eight years before the present admission. The surgical history included only tonsillectomy as a child and the removal of two clitoral cysts two years before the present admission. The review of systems revealed the following symptoms related to the patient’s marked depression: insomnia (very little sleep during past three nights), anorexia and adipsia (she ate or drank very little during the past two to three days), xerostomia and dysphagia (she was bothered by a dry mouth and had trouble swallowing for two to three days prior to admission). She also had the aforementioned pain behind her right ear and a history of chronic constipation.

The admission physical examination revealed a blood pressure of 112/79 mm Hg, a pulse rate of 104 beats/minute, and a temperature of 98.6°F. Her mouth was very dry and revealed poor oral hygiene. There was a 2 × 2 cm mass (firm, fixed, and tender) behind and below her right ear. The rest of the physical examination, including the neurological examination, was unremarkable. Admission laboratory studies were as follows: CBC: Hgb/Hct, 13.1 g/100 ml/38.5%; WBC, 10,000/mm³ (neutrophils segs/bands, 57%/7%, lymphs, 29%, monos, 5%). Fasting blood sugar, 79 mg/100 ml, BUN, 11 mg/100 ml. Urine analysis: glucose, albumin, acetone were all negative. The following day the mass was enlarged slightly and had become warm to the touch. Medical and surgical consultations (including ENT) were obtained; all concurred with the diagnosis of acute parotitis. After a culture was taken of the purulent discharge from Stenson’s duct, the patient was started on procaine penicillin, initially 1,200,000 U, then 600,000 U orally, twice daily. Despite warm compresses and penicillin treatment, which included an extra 2,000,000 U intramuscularly during the second day, the patient’s condition worsened. On the third day penicillin was discontinued and she was started on methicillin, 1 g intramuscularly q6h. The initial culture was reported as “heavy growth Staphylococcus aureus” which was resistant to penicillin and sensitive to methicillin.

During the third day the pain was severe and the swelling had increased to 4 × 6 cm, extending to the right mandible. She was unable to open her mouth fully or to chew food. On the morning of the fourth day the patient was transferred to the surgical service and started on intravenous methicillin (2 g., four times daily). Incision and drainage was planned if she did not respond favorably to antibiotic treatment. The patient was very anxious about the transfer and had to be followed daily because of psychotic symptoms (auditory hallucinations) and her suicidal state. Daily psychoactive medications were reduced to Stelazine, 15 mg; Elavil, 150 mg; and Cogentin, 2 mg. In addition to intravenous methicillin, hot compresses were applied every two hours and daily parotid massage was begun. On the day of transfer her WBC was 12,000 mm³ (neutrophils, segs/bands 51%/22%; lymphs, 19%; monos, 5%; eosinophils, 2%). The culture of the parotid duct exudate after one full day of methicillin therapy (intramuscularly and intravenously) was predominately Klebsiella group, with few Staphylococcus aureus. X-ray studies while the patient was on the surgical ward showed no definite evidence of calculus in Stenson’s duct. By the second day on the surgical ward, the patient’s condition improved, with a decrease in the pain and the swelling. After one week of the above therapy the patient’s parotitis resolved and she was returned to the psychiatric service.

Comment: This patient’s history, laboratory evaluations, and hospital course ruled out several causes of acute parotitis: mumps, diabetes mellitus, drug hypersensitivity reaction, severe malnutrition, alcoholic cirrhosis, and a calculus in Stenson’s duct. The most likely etiology, therefore, appears to have
been xerostomia secondary both to psychoactive medications, and to the autonomic and vegetative aspects of her depression (anorexia and adipsia) that led to dehydration.

A decrease in saliva production in severe depression has been well documented. Dryness of the mouth coupled with poor oral hygiene causes inspissation of secretions in the parotid duct. The patient was on extremely high doses of three medications that have atropine-like effects. This powerful additive anticholinergic quality causes xerostomia and may contribute to parotid swelling by passive congestion. The diminution of salivary secretions leads to difficulty in swallowing food, poor oral hygiene, and change in the density and nature of the bacterial flora of the mouth. This combination of circumstances provides an ideal culture medium for pyogenic (specifically *Staphylococcus*) organisms, and the bacterial parotitis is then caused by retrograde extension of infection from the mouth into the parotid gland via Stensen's duct.

Case 2: A 59-year-old white male was admitted to the Langley Porter Institute with the past diagnosis of schizophrenic reaction, schizo-affective type. The patient had a gradual change in his behavior over the five years prior to admission characterized by days of poor judgment and mood swings. During the “high days” he was overconfident and he displayed drive, pressure of ideas, and exceptional energy. On the “low days” there was a dearth of ideas, retardation of both motor and mental activity which he attempted to counteract with thyroid extract and stimulants. Five weeks prior to admission the patient had a violent argument with his wife, during which he beat her. Following this incident, which his wife realized was not in keeping with his nature, she sought psychiatric help for her husband which he refused to accept. The patient was hospitalized involuntarily after two to three weeks of manic behavior characterized by euphoria, sleepless nights, extreme grandiosity (the patient spoke of becoming “the ruler of the world”) and decompensation at his job. The patient was in the hospital three weeks involuntarily and received chlorpromazine (Thorazine) 400–600 mg orally per day which kept his behavior within acceptable limits. The patient was then transferred to the Langley Porter Institute for a ten-week voluntary hospitalization.

On admission to Langley Porter the patient was considered to have a manic-depressive reaction, manic type. He spoke rapidly in an intelligent manner. His mood was labile varying from neutral to euphoric. He described his life and work in grandiose terms, at times approaching delusional proportions; he denied hallucinations. The patient's thought processes were characterized by tangential thinking and circumscription. He showed mild pressure of speech and flight of ideas. His memory was excellent and his concentration and attention were good. The patient had no insight into his disorder.

The patient's medical history was negative except for a past appendectomy and mild hypothyroidism. The patient had been on thyroid extract, caffeine, and glutamic acid almost daily for the past five years.

The patient's admission physical examination (including the neurological exam) was within normal limits except for an enlarged prostate and 2+ pedal edema which disappeared on stopping Thorazine. The admission laboratory data were as follows: CBC: hgb/hct, 16.5 g/100 ml/48.5%, WBC, 7500/mm³ (neutrophils seg/bands, 60%/3%, lymphs, 29%, monos, 3%, eosins, 5%). Urine analysis: Glucose, albumin and acetone—all negative. Fasting blood sugar = 94 mg/100ml.

On the day following admission, the patient's medication was changed from chlorpromazine (Thorazine) to perphenazine (Trilafon) because of the patient's pedal edema. The patient was also on benztrapine mesylate (Cogentin) 2 mg orally per day and Thyroglobulin (Proloid) 3 gr orally per day. The patient's dose of Trilafon was gradually increased from a starting dose of 48 mg orally per day to 136 mg orally per day during the first two weeks. After three weeks in the hospital, the patient became overly sedated, confused, and exhibited decreased concentration with a memory impairment. Because of this, Trilafon was discontinued and thioridazine (Mellaril) 200 mg orally per day was substituted. After seven days of Mellaril chemotherapy, the
patient complained of a painful soft tissue swelling over the left angle of his jaw. The patient was afebrile. Physical examination, at that time, revealed a firm and tender swelling in the left parotid region. The following day the patient's temperature was elevated to 101°F and a CBC done at that time revealed a WBC, 9450/mm³ (neutrophils segs/bands, 76%/2%; lymphs, 16%; monos, 4%; and eosins, 2%). An ENT consultation was obtained and the consultant's examination was reported as follows.

Left parotid tenderness and induration with no purulent discharge from Stensens duct. On milking the gland no saliva or discharge was expressed. No submaxillary or right sided involvement noted. Oral hygiene in general is good.

The consultant's impression was acute left parotitis and he advised (1) warm compresses every four hours during waking hours, (2) Procaine penicillin 600,000 U intramuscularly, then oral penicillin G 400,000 U orally, four times daily (3) SSKI 40 drops three times daily in juice, and (4) soft tissue X-ray of both parotids. All these recommendations were carried out in treating this patient's acute parotitis. The patient's parotitis resolved after one week of the above treatment. During this week of treatment the patient's Mellaril was decreased to 75 mg orally per day and theCogentin was discontinued. Unfortunately no culture was obtained from the left parotid duct. The X-ray of the patient's parotid glands revealed, "no evidence of calcification along the course of the parotid duct."

Comment: The etiology of acute parotitis has been linked to specific non-psychoactive drug hypersensitivity reactions. This case appears to represent a clear account of acute parotitis associated with a specific psychoactive drug treatment. This patient developed acute parotitis only after his medication had been changed to thioridazine (Mellaril) and he had been on the drug for seven days. There are three cases reported of Mellaril causing parotid swelling and this present case may indicate that this drug has a greater propensity than others to lead to subsequent acute parotitis.

DISCUSSION

Case 1 emphasizes the close association between emotional and organic processes. Depressed patients under acute stress will often develop severe insomnia, anorexia, and adipsia and will sleep, eat, and drink very little for days. Many of their psychic and somatic processes slow down during depressive periods, and the production of saliva may be markedly diminished. Depressed patients, schizophrenic or not, are likely to be on psychoactive medications that themselves cause dryness of the mouth. To minimize the occurrence of the set of conditions described above, drugs with atropine-like effect should be used at the lowest doses consistent with effectiveness, and the additive anticholinergic activity of antidepressants, antipsychotics, and antiparkinsonian drugs should be kept in mind.

It would be wise also to take measures to prevent acute parotitis by encouraging patients to (1) maintain good oral hygiene, (2) stimulate saliva secretions by chewing gum, sucking candy or mints, or even eating kosher dill pickles, and (3) assure good hydration by utilizing ice chips and other acceptable types of fluids.

To avoid the recently described "iatrogenic depression" we must not in turn produce "iatrogenic acute parotitis." We should prescribe antidepressants and antipsychotics with caution for depressed and for elderly patients.

Case 2 illustrates a clear example of acute parotitis secondary to thioridazine (Mellaril). Since this represents a specific psychoactive drug reaction, this
should be taken into account by physicians when prescribing psychoactive drugs for elderly psychotic individuals.

If, however, acute parotitis does develop, as in the cases reported, it warrants prompt evaluation and treatment. As soon as the diagnosis is made, a culture of the parotid duct exudate should be obtained. The early use of a sensitive antibiotic is critical. Since acute suppurative parotitis is a painful and potentially dangerous condition, immediate medical and surgical (ENT) consultation is valuable and often essential.

In conclusion, acute suppurative parotitis secondary to depression and psychoactive drugs, although admittedly rare, has an alarmingly high mortality rate. Since it can be prevented fairly easily, attention should be focused on this uncommon disease entity without encouraging the undertreatment of the more common problems of psychosis and depression.

ACKNOWLEDGMENTS

The author is indebted to the following individuals for their assistance: Norman J. Sweet, M.D., Charles E. Mengel, M.D., Morton R. Weinstein, M.D., Ames Fischer, M.D., Leon J. Epstein, M.D., Hugh E. Stephenson, Jr., M.D., Keith Hadley, M.D., Mary Ann Esser, and Leona Magni.

REFERENCES


