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Surgical parotitis

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by

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Introduction

Surgical parotitis is an acute inflammation of the parotid gland that most commonly occurs in the post-operative period. Surgical parotitis is an uncommon disease but carries a high morbidity and a high mortality. Penicillin resistant staphlococci ascending through Stensen's duct is the most accepted mechanism of infection. Poor oral hygiene and dehydration play an important role in increasing the susceptibility of the post-operative patient. My interest in this condition resulted when an acquaintance developed surgical parotitis, following an abdominal hysterectomy. Her condition was characterized by high fever, severe pain, and marked limitation of the temporomandibular joint. Her experience led me to review the medical records for the cases of surgical parotitis at Omaha Veterans Administration Hospital (OVAH) and University of Nebraska Hospital (UNH) and to review the literature on this disease.

Materials and Methods

Examination of the medical records at UNH and OVAH in the period 1948-1968 reveals 10 cases of surgical parotitis. Also, I reviewed the literature on this subject. Bacterial culture and sensitivity testing of the exudate from Stensen's duct proved the etiologic microorganism. Inspection of the medical records of the 10 patients for age, sex, hemoglobin, hematocrit, urine specific gravity, pre- and post-operative condition, and presence of wound infection aids determination of the pre-disposing factors to surgical parotitis. The clinical manifestations, post-operative day of occurrence, and treatment response summarize the clinical course. Treatment includes non-

operative and operative technique. Non-operative treatment incorporates warm moist packs, mouth wash, isolation, antibiotics, and irradiation. Operative technique comprises incision and drainage, and tracheostomy.

Results

From Table I, the review of the cases of surgical parotitis at OVAH and UNH from 1948-68 showed the patients ranged in age from 34 to 87 years with an average age of 70. There are six male and four female patients; but if only UNH patients are considered, there are four men and four women. The hemoglobin ranged from 8.5 to 14.4 with an average of 11.6. The hemocrit, determined on only three patients, ranged from 29 to 44 with an average of 38. In light of the average hemoglobin, the mean hemocrit probably reflects a high value. Urine specific gravity ranged from 1.005 to 1.024 with an average of 1.017. The urine specific gravity can only suggest hydration since so many underlying conditions can affect urine concentrating ability (e.g. hypokalemia). On three of the 10 patients, dehydration was specifically mentioned and decreased skin turgor was noted on another. Six of the 10 cases involved the right gland, three the left, and one was bilateral. Only one of the 10 cases had infection - gangrene of the foot in patient #1.

Table II shows that Staphylococcus aureus was uniformly cultured. In three of those cases, the staphylococcic organism was hemolytic and coagulase positive; in one the organism was coagulase negative; and in four of the cases, the organism was hemolytic. One case was non-hemolytic. Other organisms cultured included Streptococcus, Escherichia coli, and Aerobacter aerogenes. Three of the patients did not undergo culture and sensitivity testing.

Table I - Etiology

Patient	Sex	Age	Parotid Gland	Hemoglobin	Hemocrit	Urine Specific Gravity	Wound Infection
1	M	67	R	12.2		1.005	Yes
2	M	83	R	12.6	44	1.010	No
3	F	62	R	8.8		1.011	No
4	M	60	R	12.3		1.020	No
5	F	86	L	12.0		1.022	No
6	F	80	R	14.4		1.016	No
7	F	87	R	13.1		1.022	No
8	M	72	R	8.8		1.017	No
9	M	69	L	13.2	41	1.024	No
10*	M	34	Both	8.5	29	1.022	No

* Patient #10, Hodgkins disease, on Velban medication

Note - Patients 1 through 8 (UNH), 9 and 10 (OVAH)

Table II

Patient	Cultured
1	Staphylococcus aureus, hemolytic, coagulase positive
2	Staphylococcus aureus, streptococcis, Aerobacter aerogenes
3	Staphylococcus aureus, hemolytic, coagulase positive, Escherichia coli
4	Staphylococcus aureus, non-hemolytic
5	Staphylococcus aureus, hemolytic, coagulase positive
6	Not cultured
7	Not cultured
8	Staphylococcus aureus, hemolytic
9	Not cultured
10	Staphylococcus aureus, coagulase negative

Table III

Patient	Underlying Condition	Operation Performed
I	Poor oral hygiene, gangrene of the left foot	A-K amputation of left leg
2	Cholecystitis, choledocholithiasis, poor oral hygiene	Cholecystectomy, T-tube drainage
3	Cervical carcinoma, sigmoid carcinoma and widespread metastasis, pyelonephritis, poor urinary output	Colectomy and colostomy
4	Bilateral inguinal hernia, squamous cell carcinoma of the lower lip, metastasis	Incision of lower lip and left hemimandibulectomy with bilateral radical neck dissection
5	Small sacral decubitus ulcer, arteriosclerosis heart disease, atrial fibrillation, status post-operative left hip fracture with prosthesis inserted	Placed in traction
6	Essential hypertension, fracture of right femur, pneumonia, aseptic necrosis of femoral head, on tube feedings	Osteosynthesis
7	Fracture left femur, arteriosclerosis	Double hip spica
8	Benign polyp of the ampulla of Vater with complete obstruction of the common duct	Pancreaticojejunostomy, choledochojejunostomy, jejunojejunostomy, pancreaticoduodenostomy
9	Carcinoma of the lung, emphysema, arteriosclerosis, heart disease	Bronchoscopy
10	Hodgkins disease, spontaneous pneumothorax patient taking Velban (vinblastine sulfate)	Closed thoracotomy

Table IV - Clinical Course

Patient	Days Post-operative	Duration	Temperature	WBC	Response	Clinical Manifestations
1	65	28 days	106	16,920	Improved	swollen, red, tender
2	6	11	101	17,400	Improved	swollen, red, tender
3	107	8	---	30,600	Died	----------
4	18	28	101	12,000	Improved	fluctuant, red
5	11	11	100	10,200	Improved	swollen, tender
6	7	7	103	21,700	Improved	swollen, tender
7	6	??	99	8,800	Improved	----------
8	33	21	---	13,000	Improved	swollen, red, tender
9	24	4	101	------	Improved	swollen, red, tender
10	8	10	102	1,300	Improved	swollen, red, tender

Table V - Culture and Sensitivity Report on Staphylococcus Aureus Cultured

Patient	Bacit-racin	Oxacillin	Tetracycline	Chloram-phenicol	Novo-biocin	Penicillin	Strepto-mycin	Erythro-mycin	Kana-mycin	Neomycin
1	S	-	S	S	S	R	R	-	-	-
2	-	-	R	S	-	R	P	S	-	-
3	-	-	S	S	S	R	-	R	S	-
4	-	-	S	S	-	R	-	-	-	-
5	-	S	-	S	S	R	-	-	-	-
6	-	-	-	-	-	-	-	-	-	-
7	-	-	-	-	-	-	-	-	-	-
8	-	-	-	S	S	R	S	R	-	-
9	-	-	-	-	-	-	-	-	-	-
10	-	-	-	-	-	-	-	-	-	-

R = resistant
S = sensitivity
P = partial sensitivity
- - not done

Note - All organisms cultured are sensitive to chloramphenicol and novobiocin and all are resistant to penicillin

Note - Patients 6 and 7 and 9 and 10 did not have sensitivity and culture done

Table VI

Patient	Non-Operative					Operative	
	Warm Moist Packs	Lemon or Orange Juice or Mouth Wash	Isolation	Antibiotics	Radiation	Incision Drainage	Tracheostomy
1	Yes	Yes	No	Chloramphenicol Tetracycline	No	Yes	No
2	Yes	No	Yes	Chloramphenicol Rhondomycin	75r x 3d	No	No
3	Yes	No	Yes	Rhondomycin	No	Yes	No
4	Yes	Yes	No	Chloramphenicol	No	Yes	Yes*
5	Yes	Yes	No	Prostaphylin	No	No	No
6	Yes	Yes	No	Terramycin	31.5r bid x 6d	No	No
7	No	No	No	None	No	No	No
8	Yes	No	No	Chloramphenicol	No	Yes	No
9	Yes	No	No	None	No	No	No
10	No	No	No	Streptomycin Prostaphylin	No	No	No

*Tracheostomy performed on patient #4 not done for complications of surgical parotitis.

Table III shows underlying diseases of the patients and their subsequent operations.

Table IV reveals the surgical parotitis occurred from six to 107 days post-operatively with an average of 28.4 days. The duration of the disease ranged from four to 28 days with an average of 14 days. The WBC ranged from 1,300 to 30,600 with an average of 14,400. However, patient #10 with a WBC of 1,300 had Hodgkins disease and was being treated with Velban (vinblastine sulfate). If his count is excluded, then the mean WBC was 16,100. The temperature ranged from 99 to 103 with an average of 101. The disease manifested itself uniformly as a red swollen tender parotid gland. Nine of the patients improved in response to treatment. The other was a terminal cancer patient with widespread metastases. Surgical parotitis was surely a contributing factor.

Table V shows that Staphylococcus aureus was uniformly sensitive to chloramphenicol and novobiocin. It was uniformly resistant to penicillin. On four of the patients, culture and sensitivity testing was not done.

Table VI shows that both non-operative and surgical avenues were used for treatment. Warm moist packs were used in eight of the 10 patients. Lemon or orange juice or mouth wash was used in four of 10 patients. Isolation technique was employed in two of 10 patients. Chloramphenicol was used in four of 10 patients. Tetracycline of one type or another was used in four of 10 cases. Oxacillin was used in two cases. Radiation was used in two of 10 cases: 75r x three days in one case and 31.5r bid x six days in the other. An incision and drainage was performed in four of 10 cases. A tracheostomy was done on one patient, but not as definitive treatment of surgical parotitis.

Discussion

Cruveilheir (1) in 1836 in France, first described accurately surgical parotitis as a disease. He proposed that the infection ascends through the mouth and that suppression of the saliva precedes it. He found that gangrene and suppuration often occur simultaneously. Under the title "acute surgical parotitis" may be grouped all acute inflammations of the parotid gland except that occurring in viral mumps. "Post-pneumonia" since the disease occurs after a great number of other conditions. In the series reviewed at UNH and OVAH, one case (patient #10) occurs during treatment for Hodgkins disease.

The incidence of post-operative parotitis varies from author to author, but Picque (2) listed only two cases occurring among 7,200 surgical procedures (1:3600). Beckman (3) recorded three cases in 6,825 surgical procedures (1:2270). Collins (4) reviewed eight cases in 6,100 cases of post-operative abdominal conditions (1:762). Combs (5) reported nine cases among 13,000 cases in which anesthesia was employed (1:1450). Coughlin and Gish (6) noted 44 cases in 95,355 hospital patients (1:2167). Coughlin and Gish reported a complete absence of parotitis among 21,514 obstetric and newborn patients. Branson et al (7) reviewed 93 cases of parotitis, an incidence of 0.02% of admissions and a little more than one out of every 1000 operations of all kinds. Robinson (8) states that the incidence of parotitis is one in 2,835 cases at Barnes Hospital from 1921-1943.

Hemolytic Staphylococcus aureus, usually antitiotic resistant, was found to be the etiologic agent in most of the cases reported by Robinson(8). Lary (9) described poor oral hygiene, dehydration, and Staphylococcus aureus as important etiologic factors. Ragheb (10) reports three cases where tranquilizers were used with dry mouth

as a side effect. The tranquilizer's autonomic effect accounted for the dry mouth. Gilchrist et al (11) states "it has long been known that patients developing parotitis were usually seriously ill, in poor condition, dehydrated, undernourished, and with poor oral hygiene almost always Staphylococcus aureus was cultured from the inflamed parotid gland." Brown et al (12) noted, even in the pre-antibiotic era, the most common bacterial pathogen in surgical parotitis was Staphylococcus aureus. Anesthesia is incriminated by Lorhan (13) as playing a significant role in the development of this disease. Coughlin (6) reports five cases who had diabetes, two were in coma on admission. Coughlin (6) also reports eleven cases with advanced arteriosclerosis, seven cases with pharyngitis, one with bronchopneumonia, one case with a stone in Stensen's duct, 10 cases with peritonitis and seven cases with carcinoma. The cases at UNH and OVAH include nine post-operative patients and one medical patient. Most were elderly and somewhat dehydrated, Staphylococcus aureus was grown out in all cases who had culture and sensitivity testing done. Most had undergone anesthesia, Hence, the patients at UNH and OVAH are quite similar to those described in the literature. The portal of bacterial entry into the parotid gland has received much attention. Four routes are possible: (1) through the lymphatics; (2) through the bloodstream (hematogenous); (3) via the parotid duct, and (4) by direct extension from contiguous tissues. Hempstead (14), Adams (15), and Wilson (16) reported cases of parotitis, in their opinion, from direct lymphatic extension to the parotid. Coughlin and Gish (6) found no experimental or clinical proof of the extension of the infection to the parotid by way of the lymph channels.

Hobbs and Sneierson (17) felt the blood borne parotitis was

reflected by a normal appearing Stensen's duct. Coughlin and Gish (6) cite a case of their own to show that such a portal of entry may occur. In Paget's (18) 101 cases, there were only seven in which other lesions pointed to pyemia.

Coughlin and Gish (6) did not note parotitis being experimentally produced by infecting the contiguous tissues. Some cases have been reported following trauma, but the correctness of the diagnosis was questioned. Binder (19) presents a case which indicates trauma may provoke the development of post-operative parotitis.

Ascending of the micro-organism through Stensen's duct is the most widely accepted theory of the mechanism of post-operative parotitis. Cruveilhier first contended that the infection ascends the duct. In 1858, Virchow (20) concluded that Cruveilhier was correct. Custer (21) and his co-workers are supporters of the ascending infection theory. Crile and Manning (22) concluded the majority of infections are ductogenous in origin after showing staphylococci normally exist at the terminal third of Stensen's duct. Oral hygiene seemed to govern the concentration of such organisms.

Several contributing factors have been implicated by many authors. Lorhan et al states the administration of atropine and/or scopolamine, especially in the aged patient, may cause inhibition of salivary secretion. As mentioned above, Binder implicates a casual relationship between trauma and parotitis. Dehydration of the patient is felt to be an important etiologic factor by most authors (13, 6, 22, 11, 12, 10, 9, 23, 8, 19). Obstruction to the duct is an important factor according to Binder (19), and Hobbs and Sneierson (17), and Clifford. Avitamanosis, especially vitamin A depletion, results in atrophy of epithelial cells, among them the cells of the parotid gland-duct system (19, 7).

Indiscriminate pre- and post-operative use of antibiotics and hospital carriers harboring resistant organisms were felt to be important predisposing factors in the development of this infection by Petersdorf (25).

The clinical course of the 10 patients reviewed from UNH and OVAH is summarized in Table IV. Parotitis usually appeared five to seven days post-operatively, 70% of the cases appearing within the first two weeks (7). However, in the 10 cases reviewed, the mean number of days post-operative was 28.4. Patient #3 developed parotitis 107 days post- operatively. She was severely debilitated with widespread metastasis of cervical carcinoma. Patient #1 had gangrene of the foot and developed parotitis 65 days post-operatively. These two cases markedly lengthened the mean number of days post-operatively the parotitis developed and could properly be considered surgical parotitis not intimately associated with operative procedures. Petersdorf noted only three of seven cases of parotitis immediately associated with major surgical procedures.

The clinical picture is dominated by the sudden onset of a firm, warm, erythematous indurated swelling of the angle of the jaw and over the cheek associated with fever, local pain and tenderness (25, 6, 23, 9). This description agrees almost uniformly with the 10 cases reviewed at UNH and OVAH. The temperature ranged from normal to over 105°F and leukocyte counts rose to as high as 42,000 in many series (7, 11, 26). These findings closely correlate with these ten cases at UNH and OVAH. The duration of the parotitis was from a few days to several weeks, but most lasted approximately two weeks (7). The mean duration of the ten cases at UNH and OVAH was two weeks.

Branson gives a mortality of 36.8% (25/68). In 13 (19.1%) of

those deaths, the patient was sufficiently debilitated or the disease was of such a nature that the underlying cause was considered to be the actual cause. In the remainder, the parotitis was a major contributing factor. Carlson reports 46% of his patients were discharged improved; 18% died from other causes, but parotitis improved; 36% died with active parotitis present. Kruppaehne (27) reports 23% mortality from 1956-1960 at the University of Oregon. Petersdorf reports seven cases, two of whom died. Crile states that death from parotitis alone is rare but the additional burden of this complication may be the factor precipitating an unfavorable end result. In the 10 cases at UNH and OVAH, nine improved and one patient (#3) died. She had widespread metastasis from cervical cancer.

Petersdorf et al summarizes seven cases of staphylococcal parotitis according to the clinical picture, antibiotic-sensitivity patterns and phage types. Three of the cases were post-operative. The organisms were, in general, quite resistant and none were inhibited by penicillin, streptomycin and the tetracyclines. All the organisms were sensitive to neomycin, bactitracin, and novobiocin. There was a high prevalence of resistance to erythromycin and chloramphenical. Phage type and sensitivity patterns did not correlate. In his seven patients, three of the six strains and three of the four typeable strains were lysed by phages 80 and 81. Evidence in several centers indicates phage types 80 and 81 constitute a particularly virulent group of organisms and are responsible for most cases of staphylococcal infections acquired in hospitals (28, 29). In contrast to Petersdorf, the staphylococcus cultured from the patients at UNH and OVAH was uniformly sensitive to chloramphenicol. However, in agreement with Petersdorf, the staphylococcus was resistant to penicillin and

sensitive to novobiocin.

Treatment of this disease varies in the literature, but the vast majority of authors agree that prophylaxis or prevention is most important (7, 8, 25, 11, 23, 13, 30, 22, 6). Listed as important in prevention are adequate fluid intake by mouth when possible, correction of nutritional and vitamin deficiencies, frequent gargles, correction of dental caries, pyorrhea, and pharygnitis, pre-operatively and good nursing care for debilitated patients. Minimal use of belladonna drugs for pre-operative medication is stressed by Lorhan et al. Trauma by the anesthesiologist should be kept to a minimum since it may play a more important role in the pathogenesis post-operative parotitis than is clinically proveable (19). Branson discourages the use of prophylactic antibiotics, and Petersdorf echoes this philosophy as does Goldman.

Once surgical parotitis develops, culture and sensitivity testing is mandatory in light of the resistant nature of most etiologic organisms. Appropriate antibiotic therapy should then follow.

Included also under non-operative care are warm moist packs, lemon or orange juice or mouth wash, and radiation. Stimulation of parotid secretion is the intent of sour fluids and mouth washes. Gum chewing is also recommended. In the 10 cases at UNH and OVAH, warm moist packs were used in eight of the 10 cases, while parotid secretion stimulation was employed in four of the 10. Isolation technique was followed with one of the 10 patients. Carlson et al reported three deaths (25%) with prompt use of irradiation within 24 hours of diagnosis while treatment without irradiation resulted in seven deaths (42%). Bowing and Friche (31) presented figures to show that treatment with radium gives a better diagnosis - a 23% mortality rate. Petersdorf

however, was not impressed by irradiation and did not advocate its use. Lorhan feels radiation should be considered an ancillary rather than a principle form of treatment. In contrast, Gilchrist et al felt X-ray therapy should be started as soon as the diagnosis of parotitis is made. He recommended a dosage of 75r to the gland daily. If the parotitis were clinically progressing however, he recommended incision and drainage. Two of the 10 patients at UNH and OVAH received radiation. One received 75r for three days while the others received 31.5r twice a day for six days. Both improved, but both were also getting antibiotics and warm moist packs to the gland area. In neither was an incision and drainage done.

Two operative techniques were employed in the patients at UNH and OVAH - incision and drainage, and tracheostomy. Lary recommended multiple incisions in the gland in order to prevent facial nerve paralysis. Coughlin and Gish feel a vertical incision made in front of the ear connecting with an oblique incision below and behind the ear will neither cause facial nerve paralysis nor sever a large vessel. In addition, a scarcely noticeable scar should result. Petersdorf notes, however, surgery offers little for two reasons: an avenue of drainage is already available through Stensen's duct; and the fibrous nature of the gland with its many septa interferes with proper external drainage. He holds a minority opinion. Carlson et al states incision and drainage should be done within three days if parotitis is not improved. Waiting for fluctuation resulted in death in two patients and necrosis of the gland in another. Four of 10 cases at UNH and OVAH received incision and drainage treatment and three (75%) survived. The other patient (#3) died of widespread

carcinoma. Tracheostomy becomes necessary if the airway is impaired. One patient at UNH and OVAH did have a tracheostomy performed, but this was not for parotitis and its obstruction of the airway.

Conclusions

1. Ten cases of surgical parotitis are summarized from the medical records of UNH and OVAH and the literature is reviewed on this disease.
2. Post-operative parotitis is generally a disease affecting elderly debilitated people with poor oral hygiene, malnutrition and dehydration.
3. Staphylococci is the most common etiologic organism, and it is generally penicillin resistant.
4. The disease is characterized by high fever, leukocytosis, warm fluctuant, tender gland and marked limitation of the temporo-mandibular joint.
5. Prevention of this disease by correction of poor oral hygiene, malnutrition and dehydration is highly recommended due to the high mortality associated with surgical parotitis.
6. Warm moist packs, stimulation of parotid secretion, irradiation, and antibiotics are non-operative modes of treatment.
7. Incision and drainage should be carried out if the disease progresses inspite of medical treatment. Tracheostomy to prevent airway obstruction may have to be performed.

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