Mammary Duct Ectasia, Mammillary Fistula and Subareolar Sinuses

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RARE, specific causes of inflammation of the breast include tuberculosis, syphilis and fungus infections. In this country, however, mammary duct ectasia, mammillary fistulas and nonspecific inflammation are seen more commonly. These sub- and para-areolar infections are frequently confused with carcinoma and unnecessary mastectomies have been done on the mistaken premise that the lesion was carcinoma. It is important, therefore, to obtain microscopic confirmation prior to the performance of mastectomy. Not all inflammatory lesions are benign, however. Inflammatory carcinoma can be present and mammary duct ectasia can coexist with carcinoma. The clinical features of mammary duct ectasia and mammillary fistulas will be discussed in the light of our present knowledge.

Mammary Duct Ectasia

Mammary duct ectasia is characterized by dilatation of the large ducts beneath the nipple and areola, fibrosis of the wall and periductal inflammation. It has been designated in the earlier literature by a wide variety of terms including plasma cell mastitis,⁶ varicocele tumor,⁴ comedomastitis,⁷ mastitis obliterans,¹⁶ and peri-

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ductal mastitis.¹² Haagensen ¹³ concluded that these terms were all expressions of the same disease process during various stages.

Transient ectasia of the mammary ducts has been reported in newborn infants, which Sandison²⁴ believes is due to stimulation of the neo-natal breast by maternal hormones. Involution soon follows. In adult women, Walker and Sandison²⁴ found 34 patients with ectasia in 283 breast operations (12 per cent incidence). Foote and Stewart¹⁰ reported periductal mastitis in 44 of 200 non-cancerous breasts (22 per cent), and in 44 of 300 cancerous breasts (15 per cent). None of the carcinomas were traced to periductal mastitis. While mammary duct ectasia is considered a disease of the involuting breast, it has been reported to occur in young parous women and young virgins. The lesion may coexist with cystic mastopathy, or benign and malignant tumors. Haagensen 13 found no relationship between mammary duct ectasia, and mammary function or the menopause.

Etiology has been attributed to (1) mechanical factors due to intrinsic or extrinsic pressure ^{2, 10, 14, 17} such as abnormalities of the nipple, local growth of the duct lining, epithelial tumors, cysts, etc. (2) Secretion too profuse for elimination through ducts in the absence of mechanical factors.²³ Others have been unable to demonstrate obstruction but implicate hormonal imbalance.^{6, 22} Rodman and Ingleby ²⁰ found colostrum-like cells and fatty acid crystals and state that the disease could be due to the action of enzymes which split milk-like substances secreted under certain conditions in the breasts of non-pregnant women.

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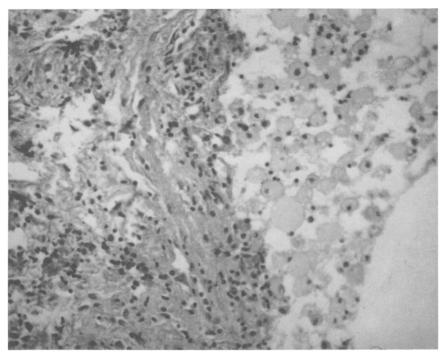


FIG. 1. Mammary Duct Ectasia. Intraductal foam or colostrum cells, disappearance of lining cells, periductal inflammation predominantly lymphocytes. (H & E ×200.)

Tedeschi *et al.*²² believe that the histologic picture is consistent with that of chemical irritation. They report, in a histochemical study, severe alterations in the system of fibrous collagenous fibers, reticulin fibers, elastic tissue, and ground substance.

The mechanism of escape of secretion into surrounding tissues could be (1) degeneration of the epithelium by retained secretion,⁸ (2) focal infection ¹⁸ or (3) local trauma,⁵ including surgical incision.

Clinico-Pathologic Correlation

In the early stages, the lesion may be asymptomatic. The terminal ducts beneath the nipple and areola are dilated and the lumen contains lipoid material and cellular debris. Duct epithelium is not abnormal, nor is there evidence of inflammation. Frantz and associates ¹¹ in an autopsy series found such changes in 25 per cent of women who supposedly had normal breasts. The average age in their series was 61.7 years. Sandison ²¹ in another autopsy study of 800 patients found that duct dilatation was present in 72 per cent, and 11 per cent had gross duct ectasia.

The condition becomes symptomatic in a small per cent of patients, and pain and tenderness are frequent. The symptomatic group can be divided into stages (a) nipple discharge (b) nipple retraction (c) tumor (d) abscesses, fistulous tracts, and fibrotic changes.

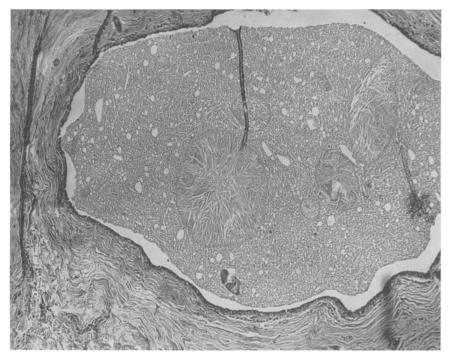
Nipple Discharge: Nipple discharge, when present, can occur spontaneously, is usually intermittent and may exist for long periods. The secretion varies from yellow to dark green in color, and the consistency is watery to pasty. The discharge is not related to the menses. Histologic examination of the secretion shows amorphous debris and colostrum (or foam) cells. If an operation is done, the ducts are dilated to from 2 to 5 mm., appear bluish in color and contain lipoid material. The majority of large ducts are involved, but the process may extend to involve smaller ducts.

Nipple Retraction: As the disease progresses, the duct dilatation is accompanied

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FIG. 2. Mammary Duct Ectasia. Dilated duct with atrophic epithelium —contents are crystals with radiate structure, fat bodies and debris. (H & E $\times 56.$)



by inflammatory changes and infiltration with lymphocytes. The duct wall becomes thickened by fibrosis leading to shortening and retraction of the nipple. If edema is present, a peau d'orange appearance results. Haagensen¹³ states that the epithelium lining of the ducts is thinned and there is absence of epithelial proliferation. Tedeschi et al.22 believe that although atrophy is the basic alteration of the epithelium, proliferative changes can occur in an abortive attempt at secretion. Frantz¹¹ found proliferative or apocrine changes in the ductal epithelium in 50 per cent and foam or colostrum cells in 35.2 per cent of patients with ectasia (Fig. 1). The ductal contents are amorphous debris (lipoid) and contain scattered crystalline bodies (Fig. 2), which are round or oval and have a radiate structure.

Tumor Mass: The continuity of the duct epithelium is broken and lipoid material escapes through the duct wall, leading to a marked yellowish-gray induration of the surrounding tissue. The tumor mass, with or without obvious inflammation, is firm or poorly circumscribed. Nipple retraction and dimpling are often present; therefore, the lesion is frequently diagnosed as carcinoma. Pain and tenderness, evanescent inflammatory changes, both occurring over a long period, tend to rule out such a diagnosis. Biopsy, however, is mandatory to exclude carcinoma.

Histologically, at this stage, the inflammatory reaction is intense. In addition to the usual atrophic epithelial changes in the duct epithelium and inflammatory reaction in the duct wall, there is a marked cellular inflammatory response (Fig. 3). The inflammatory cells consist chiefly of lymphocytes and plasma cells, or almost entirely of plasma cells (plasma cell mastitis). Phagocytic giant cells surround the lipoid material, and infiltration of histiocytes, lymphocytes, and polymorphonuclear cells occur in the breast stroma.

Abscesses, Fistulas and Fibrosis: An apparent abscess develops. However, when this is incised, the material is usually



FIG. 3. Mammary Duct Ectasia. Duct with marked lymphocytic periductal inflammation. A fibrosed duct is also seen. (H & E $\times 110.$)

sterile. Recurrent abscesses, and fistulization then occur. Although the duct epithelium is usually atrophic, proliferative and apocrine changes, metaplasia of the squamous cell type (Fig. 4) and mammillary fistula can be present. With repeated infections, scarring and fibrosis ensue. Destruction of the duct by inflammation, and



FIG. 4. Mammary Duct Ectasia. Sinus tract with squamous metaplasia of a duct. Diffuse infl a m m a tory c h a n g e s in breast stroma. (H & E $\times 110$.)

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FIG. 5. Mammillary Fistula. Two large ducts lined by squamous epithelium. One of the ducts has ruptured, liberating keratin. (H & E $\times 40$.)



fibrous obliteration of the duct are also frequently observed on histologic examination.

Treatment: When there is nipple discharge alone, the secretion should be examined for the presence of foamy colostrum cells. In such instances, observation only is indicated. Operation, however, may be advisable to rule out a possible tumor, particularly if secretion can be expressed from a segmental area. In such instances, when operation is carried out, the enlarged bluish ducts will be found. The ducts are severed at the base of the nipple and a segment of tissue is removed.

In the presence of retraction or tumor, excisional biopsy and frozen section is required. Mastectomy is not necessary for this lesion. When the lesion presents as an abscess, incision and drainage with biopsy of the abscess wall is accomplished. Wide excision and primary closure is the procedure of choice for fistulous tracts. Excision with delayed closure, and excision with marsupialization has also been performed. In isolated instances, sinus tracts have successfully been treated with estrogens.^{15, 24}

Mammillary Duct Fistula

Mammillary duct fistula (Figs. 5, 6, 7) is frequently confused with mammary duct ectasia. Zuska et al.25 in 1951 reported five cases of comedo-mastitis in which fistulas developed. In several cases the ducts were lined by squamous epithelium. In 1955, Atkins³ described the entity as mammillary duct fistula. The lesion was found in younger women and was most commonly seen during pregnancy and lactation. The clinical features are a small subareolar abscess, which eventually drains, heals and later recurs. Atkins described the disease as a subareolar sinus lined by nonspecific granulation tissue that communicates with a duct. He suggested that blockage of the duct system was due to congenital retraction of the nipple and recommended as the treatment, exteriorization of the fistula. Zuska's ²⁵ demonstration of large ducts lined by squamous epithelium was con-

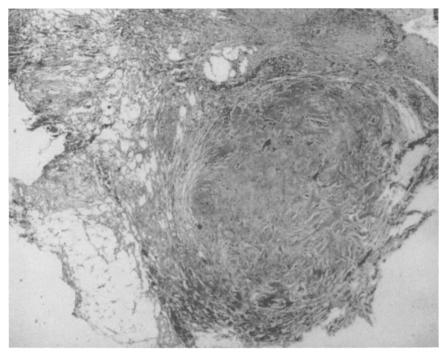


FIG. 6. Mammillary Fistula. Complete obliteration of a large duct. (H & E ×40.)

firmed by Patey and Thackray,¹⁹ who in turn recommended excision. They observed blockage of a duct by a plug of cornified cells. They postulated that mamillary duct fistula was due to a congenital abnormality of one of the main ducts since in two in-

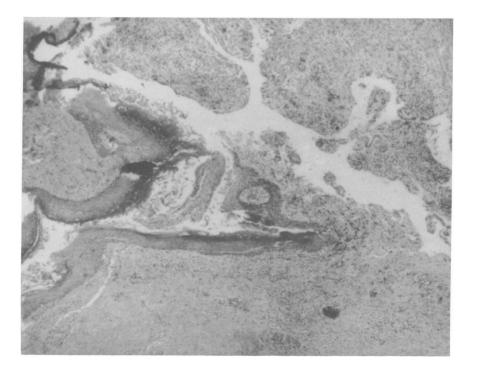


FIG. 7. Mammillary Fistula. A sinus tract lined by squamous epithelium. Marked inflammation is present. (H & $E \times 40.$)

TABLE 1. Summary of Cases of Mammary Duct Ectasia, Mammillary Fistulas, and Subareolar Sinuses

Group 1: Mammary	duct ectasia	without	sinuses-Cases 1-7	
Group 2: Subareolar	sinuses			

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	Case	Age	Race	History	Clinical Features	Operation		Pathology
Group II Group I	1. MW	26	С	Abscess with preg., subareolar mass	Tumor	Excision	1X	MDE
	2. MY	43	С	Subareolar mass	Tumor	Excision	1X	MDE
	3. LS	45	С	Nipple discharge	ThickenIng	Excision	1X	MDE
	4. QW	39	Ν	Subareolar mass	Tumor	Excision	1X	MDE
	5. EC	42	С	Subareolar mass	Tumor	Excision	1X	MDE
	6. OD	40	Ν	Subareolar mass	Tumor	Excision	1X	MDE
	7. MN	32	Ν	Subareolar mass	Tumor, inflammation,	I and D Excision	1X 1X	MDE
	8. FC	35	С	Abscesses 6×, (I & D); breast biop., bilat.	Fistula, inflammation, tumor	I and D Excision	$^{4\times}_{1\times}$	MDE
	9. AG	41	С	Abscess, I and D	Fistula, inflammation, tumor; clin. ca.	Excision	3×	MDE
	10. MD	38	С	Breast biop., p.o. sinus	Fistula, drainage	Curettage I & D 1×, Exc.	$2 \times 3 \times$	MDE
	11. EB	46	С	Subareolar mass, discharge, pain	Fistula, tumor	Excision	1X	MDE
	12. ML	35	С	Subareolar mass, discharge	(a) Tumor, inflammation (b) Fistula	(a) I and D (b) Excision	1X 1X	MDE
	13. EB	40	Ν	Abscesses and sinus 8 years duration	Fistula, tumor, inflammation	Exc. 1×; Exc. and Marsupial.	1×	MDE., Sq. Meta.
	14. MG	35	С	Abscesses, recur.; many I and D's	Fistula, inflammation	Excision	$4 \times$	MDE., Sq. Meta.
G	15. MB	40	С	Abscess, I and D, biop. wall, drain.	Fistula	Excision	$2\times$	MDE., Sq. Meta., MF
	16. KK	43	С	Breast biop., p.o. sinus	Fistula, inflammation	Curettage Excision	1X 1X	MDE MF
	17. HR	32	С	Recur. abscesses & sinuses $8 \times -1\frac{1}{2}$ yrs.	Fistula	Excision	1X	MDE MF
	18. HH	35	С	Subareolar mass, discharge	Fistula, tumor, inflammation	Excision	1×	MDE MF
	19. AY	33	Ν	Abscesses with preg., I and D $-3\times$	Fistula, tumor, inflammation	I and D Excision	$1 \times 2 \times$	MF
	20. MH	41	С	Subareolar mass	(a) Tumor, inflammation (b) Fistula	(a) Excision (b) Excision	1X 1X	MF
	21. MG	24	С	Drainage	Fistula	Excision	1×	Non-spec inflam.

Note: C., Caucasian: N., Negro: I and D, Incision and Drainage: MDE, Mammary Duct Ectasia: MF., Mammillary Fistula: Sq. Meta., Squamous Metaplasia.

stances they found sebaceous glands and in another hair lying free in a duct. As an alternative theory Patey and Thackray suggested that metaplasia of the main ducts could be a cause. Squamous metaplasia of the sinus tract itself has been reported by Hadfield ¹⁴ and by Sandison and Walker.²¹

Material and Methods

In a previously reported series of breast biopsies ¹ performed at the Outpatient Clinic, Walter Reed Army Medical Center, Washington, D. C., mammary duct ectasia was found in 18 patients (2.1 per cent incidence).

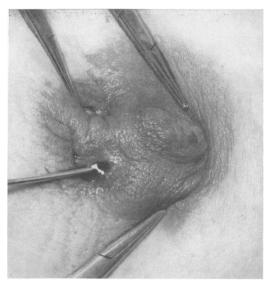


FIG. 8. Preoperative photograph of a patient with draining sinuses. Two sinus openings are seen and extent of induration is demarcated by the hemostats and probe.

Seven patients had mammary duct ectasia without sinuses (Table 1). Four were Caucasian and 3 were Negroes and average age was 38 years. Nipple discharge alone was found in one patient. A subareolar mass was present in five; one had an abscess previously. An inflammatory mass was present in the seventh patient; incision and drainage preceded excision. Excision of the mass resulted in cure in all patients—in contrast to the multiple procedures required in patients with sinuses.

Subareolar sinuses were present in 14 patients (Table 1), seven had mammary duct ectasia—two of whom also had squamous metaplasia of small ducts. Four additional patients had elements of both mammary duct ectasia and mammillary fistula (one of these also had squamous metaplasia of a duct on histologic study). Two patients had mammillary duct fistula alone, and nonspecific mastitis with a sinus tract occurred in another. Squamous cell changes were seen in the lining of the sinus tract itself in three patients with mammillary fistula.

In 14 patients with sinuses or fistulas,

there were 11 (10 Caucasian, one Negro) who had mammary duct ectasia alone, or combined with mammillary duct fistulas. Their average age was 38 years. In three remaining patients, two had mammillary fistulas alone and one had nonspecific mastitis. Their age and race are listed in Table 1.

Of 14 patients with sinuses, regardless of etiology, five had had no prior operation. In the remaining nine patients, four had single operations and five had multiple procedures.

Of operative procedures performed in the Surgical Clinic on the sinuses, incision and drainage was performed seven times (in four patients) and curettage three times (in two patients). Neither of these methods resulted in cure. Single excisions were successful in seven patients. Multiple excisional procedures were required in seven other patients (four times in one, three times in two, and two times in four patients). Twenty-five excisional procedures were therefore required in fourteen patients with sinus tracts to effect healing.

The type of excisional technic depends upon the findings at operation. When a simple sinus tract communicates with the nipple, excision of the sinus tract and reconstruction of the nipple is necessary (two patients). With diffuse induration, involvement of the nipple and fistulization, complete subareolar excision including the tissue beneath the nipple, is necessary (Figs. 8, 9, 10) and was performed in four patients. In three patients, the sinus tracts were probed from one side of the areola to the other, beneath the nipple. Excision of all subareolar tissue was required, including reconstruction of the nipple in one. When the sub-nipple area has been excised, several "tacking" sutures are placed on each side of the nipple to prevent deformity and to ensure better healing by eliminating dead space. There were no adverse effects from these procedures, although none of the patients subsequently Volume 169 Number 2



FIG. 9. Postoperative appearance. The entire involved area has been excised including the subnipple area. The nipple has been "tacked" down with several chromic catgut sutures.

became pregnant. Simple excision of the subareolar thickening and sinus, not including the nipple area, was required in the remaining five patients. Excision and delayed closure, and excision and marsupialization were also performed in two patients. Follow up in these patients with sinus tracts ranged from 1 to 6 years. One patient continued to have difficulty.

Five other patients, not included in Table 1, had chronic nonspecific subareolar inflammation. Biopsies were performed on each breast of a young girl at the ages of 13 and 15 years. Periductal mastitis, consisting chiefly of lymphocytes, was found. The possibility of periductal mastitis occurring as a manifestation of an autoimmune process is suggested not only in this patient, but also in many instances of mammary duct ectasia.

Discussion

The basic features of mammary duct ectasia and mammillary fistula have been described; however, there are gaps in knowledge of these lesions. While Haagensen ¹³ divided mammary duct ectasia into clinical stages, it is evident that varying histologic features, including overlapping changes and early and late changes can be found in a single specimen. Haagensen emphasized the absence of epithelial proliferation of the duct lining and stated that atrophy and flattening of the epithelium was

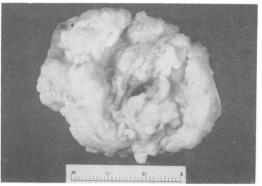


FIG. 10. Gross appearance of the resected specimen.

the rule. In all 20 cases, he found crystalline bodies in the dilated ducts; however, these crystals may be difficult to demonstrate in the usual, single paraffin section. Colostrum cells, squamous metaplasia, apocrine changes and fistula formation were not described in his paper, but have been mentioned by others ^{20, 23, 24} as an integral part of this lesion. Proliferative duct changes have also been described.^{10, 22, 23} In addition, marked destruction of the lining epithelium, fibrous obliteration of the ducts, and a diffuse fibrosis can occur.

Fistula formation is a frequent finding in later stages of mammary duct ectasia; and may occur either spontaneously, or after incisional and excisional procedures. Fistulization occurred in five of six patients in Tedeschi's series,²² and in six patients in Sandison and Walker's series.²¹ The latter also called attention to the association of mammillary fistula and mammary duct ectasia. In the presently reported series, fistulous tracts were found in 13 patients: one with fistula due to nonspecific mastitis was excluded. Mammary duct ectasia and associated mammillary fistulas were found in four patients. Sinuses or fistulas with mammary duct ectasia also were present in seven patients and two patients had mammillary fistula alone.

In the two lesions there are many common pathologic features. The sinuses are usually lined by nonspecific granulation

tissue. The duct walls are thickened and periductal inflammation is present. Obliterative duct changes, destruction of the ducts and diffuse fibrosis can occur in both. The essential difference seems to lie in the dilated duct lining and contents of the lumen of the duct. In mammillary fistula the duct lining is replaced by squamous cells and the lumen contains amorphous material, keratin and at times cornified plugs. In mammary duct ectasia, the duct lining is usually atrophic (however, apocrine or proliferative changes can be present) and the duct contents are debris, lipoid material and crystalline bodies.

The diverse clinical and pathologic findings associated with mammary duct ectasia, the similarity of the changes in sinuses associated with mammary duct ectasia and mammillary fistula, and their not infrequent coexistence indicate that these two lesions are variants of the same disease process. As suggested by Ewing⁹ the basic etiology is duct stagnation.

Summary

The basic features, including treatment, of mammary duct ectasia, and mammillary fistula have been reviewed. While these entities may be distinct and separate, there is increasing evidence that indicates they are variants of the same disease process caused by duct stagnation.

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