SEMINAR ON THE SKIN NEOPLASMS & DERMATOSES

1954

SEMINAR ON THE SKIN NEOPLASMS AND DERMATOSES

American Society of Clinical Pathologists
International Congress of Clinical Pathology
Washington, D. C.

September 11, 1954

Given by

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Armed Forces Institute of Pathology
Washington, D. C.

Twentieth Seminar

Published by the
AMERICAN SOCIETY OF CLINICAL PATHOLOGISTS
1955

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Seminar on the Skin Neoplasms and Dermatoses

SATURDAY MORNING SESSION

September 11, 1954

The Annual Seminar of The American Society of Clinical Pathologists, held at the Shoreham Hotel, Washington, D. C., on Saturday, September 11, 1954, convened at 9 A.M., Dr. Lauren V. Ackerman presiding.

CHAIRMAN ACKERMAN: We have a long road to travel today, so we will begin promptly.

The same thing, of course, can be said at every one of these Seminars, because many of the same people and their organizations are involved. Dr. Culbertson, of course, does a tremendous amount of work, and we are always so much indebted to the Armed Forces Institute of Pathology. You have heard me speak of General DeCoursey and Hugh Grady and some of the others who are there. Perhaps we have been a little bit remiss in not speaking more about the people who get out the slides.

I am a rather farseeing person, and I know that if I asked Dr. Helwig to give this Seminar a year in advance he would refuse; but three years always seems so far away to anyone. As a matter of fact, it is just around the corner.

To prepare this Seminar was a Gargantuan task and, as you know, dermatologists and pathologists who know dermal pathology can write pages on a little wisp of tissue; but when you try to get enough wisps to make 1,400 slides, that is a little troublesome. I happen to know that the Institute cut about 50,000 slides on this particular Seminar, and discarded 15,000. So, if you complain to me that Slide 23 is not stained quite correctly, that it is a little on the hematoxylin, you will pardon me if I call you some names. (Laughter)

(Slide) I am not supposed to show you any slides, but this one I would like to show you. These are a few of the slides that went out. This fellow (Dr. Helwig) you will see more of today. He has a happy expression on his face because the slides are finished. You should have seen his expression before! (Laughter)

This lady is the one who is responsible for getting out the slides. Miss Mary Frances Gridley.

Dr. Davidsohn, would you mind coming up and saying, much better than I can, a few words about Miss Gridley?

DR. ISRAEL DAVIDSOHN (Chicago): Where is Miss Gridley?

CHAIRMAN ACKERMAN: She is in the audience. Make her come forward. (Applause)

DR. DAVIDSOHN: I asked Dr. Ackerman to give me the privilege of saying a few words to Miss Gridley on this occasion, because to me it is a little bit of unfinished business.

After all these years, since the AFIP began helping us in making this the most exciting educational experience of the year for American pathologists, I have always hoped to have an opportunity to express to Miss Gridley in the presence of all of you, the beneficiaries of her skill, our gratitude and appreciation for what she has done to help make this such a great success.

To us Miss Gridley is the flower of American technologists. (Applause)

This opportunity, for which I have been waiting for such a long time, has presented itself at this meeting, which is held in Miss Gridley's home town, and therefore I would like to present to you, Miss Gridley, these flowers. (Applause)

Always remember that you are the associate of all these American pathologists. Thank you, and God bless you. (Applause)

CHAIRMAN ACKERMAN: Now I should like to introduce Dr. Elson Helwig, who is going to conduct this Seminar. As you know, he is in charge of dermal pathology at the Armed Forces

Institute of Pathology, and he is a pathologist who knows all about those things that sometimes we call "chronic inflammation." (Laughter)

I would like to introduce Dr. Helwig without further ado, because we have so much to cover. Dr. Helwig. (Applause)

DR. ELSON B. HELWIG: Dr. Ackerman, Colleagues, Friends and Neighbors—and I Do Mean Neighbors: I am very happy to see so many of you here from other countries. I would like to tell you how deeply I feel about the honor of being invited to give this Seminar. It is a real pleasure. It is a privilege for me to be able to discuss with you some of my thoughts and problems concerning dermal pathology.

At the AFIP we have a conference in which the attitude of the audience toward the speaker is, "Don't speak while I am trying to interrupt you." (Laughter) I hope the same spirit prevails today.

My negotiations with Dr. Ackerman, your Chairman, up to this point, have been rather stiff and formal. I hope from now on he will relax a bit; in fact, I would like to tell you just how stiff and formal these negotiations have been.

One evening about three years ago, I wandered into one of those rooms where people were standing about, talking. I saw Dr. Ackerman over in a corner; I went over and said hello to him. Dr. Ackerman said, "You're It. Dermal Pathology, 1954. Congratulations." (Laughter)

I was still a bit skeptical. For all I knew, he was kidding. So to make sure I asked, "Will you put this invitation formally in writing, and, in addition, will you tell me what broad principles I should follow in that Seminar?"

He said, "I will be glad to."

I waited two years and I didn't hear from him. One day he came into the Institute and I said to him, "Are you really serious about my giving this Seminar?"

"I sure am," he replied.

"I have never received that letter and I have never received those broad principles. Here is my secretary. Why don't you dictate a letter to her right now?"

"That's not necessary. Give me a scrap of paper."

So I gave him a scrap of paper, and this is what he wrote:

"Dear Elson: I think the Seminar should be comprised of mostly tumors; that it should contain four or five sections of specific dermatoses to let the general pathologist know that he should not call everything 'chronic dermatitis,' and two stinkers." (Laughter)

I am sure we have several examples of tumors in the group and four or five very specific dermatoses, but I will have to wait until the day is over to find out about the "two stinkers." (Laughter)

Everyone at the Institute has been most cooperative in helping me prepare for this Seminar. You have already heard of the marvelous job that Miss Gridley and her assistants have done. The entire staff has helped me, and particularly some people who one seldom hears about. For instance, Mrs. Little and her assistants, to whom I could say, "I want a listing of 300 cases of discoid lupus erythematosus tomorrow night," and there was the list. Or to Mrs. Evans and her assistants, who showed no dismay when I said, "I want slides drawn on 500 senile keratoses for use over the week end," and I always got the slides. They have helped me a great deal in getting all this material together.

The staff in photography have been most cooperative. Chief Helwig, Sergeant Weber, and others have been patient with my wants and have taken many of these pictures.

General Elbert DeCoursey, the Director, Captain William Silliphant of the Navy, and Colonel Ralph Thompson of the Air Force, Deputy Directors, and Dr. Hugh Grady, Director of the American Registry of Pathology all have helped me in many ways in facilitating the preparation.

Much additional work has fallen on my assistants in the section of Dermal Pathology—Dr. Henry Grinvalsky in the early part, and more recently Dr. Clarence Denser and Dr. Joã Pereira.

Finally I would like to say thanks to the many pathologists of the Armed Forces, Veterans Administration, Public Health Service and civilian ranks for making the material available for this seminar.

In selecting material for the Seminar, we tried to choose examples that would be practical for the purpose of definitive microscopic diagnosis

and would also illustrate problems of differential diagnosis. We were governed somewhat by the material we could get. I have come to the conclusion, after looking over much material, that individual lesions vary just like people—no two look exactly alike. Many of the cases are recorded as Mr. or Miss Composite to indicate that the specimens from several patients were used on one case in your Seminar. It was quite a job to match these tissues and I hope you will not be too critical.

We should realize, as far as the diagnoses of dermatologic lesions are concerned, that in many instances the diagnosis has grown up on a clinical basis from the dermatologist, and it has been only within relatively recent years that the microscopic pathologic aspect has been brought in. Sometimes the two do not dovetail. The nomenclature seems out of line with the microscopic picture, but because of usage we frequently follow the nomenclature given long ago (e.g., mycosis fungoides).

I would like to say one other word about the skin. I do not believe skin is just skin—four layers which one can define. More and more I believe that skin is a very specialized organ, that it varies in function and in physiology in many different parts of the body. That, in turn, of course, is reflected in some diseases that we know now.

A few of you wrote in and asked that I give a very brief description of some of the changes in the skin. I think that is how we will begin. These first slides will show some of the anatomic and basic pathologic changes.

(Slide) This section shows *acanthosis.* By that we mean an increase in the amount of the prickle cell layer. The epithelial cells are increased and the rete ridges (they are not pegs) are greatly elongated, but they may be flat and broad. The connective tissue of the corium between the rete ridges (not the epithelium) makes up the papillae of the skin. At the surface there is an increase of the stratum corneum—*hyperkeratosis.*

(Slide) In this section we see an abnormality which is very important in the microscopic diagnosis of cutaneous diseases. At this zone in the epidermis there should be a distinct granular cell layer, but it is absent—*hypogranulosis.* As a result, above in the stratum corneum the nuclei are retained in the epithelial cells. This is called *parakeratosis.* (The epithelium of squamous mucous membrane normally shows parakeratosis.)

(Slide) In distinct contrast to the section we just saw, the granular cell layer in this section is quite distinct—*hypergranulosis.* This change is also important in diagnosis.

(Slide) Here is a change which is called *liquefaction degeneration.* As you can see, the junction of the basal layer of the epidermis and the corium is obscured in two ways: One, by edema of the lower cells of the epidermis; two, by the presence of inflammatory cells in the corium subjacent to the epidermis.

(Slide) This section shows prickle cells separated by clear spaces containing intercellular fluid. This is called *spongiosis.* If it becomes more marked, a vesicle may form.

(Slide) In this section, cells here and there in the epidermis exhibit an acidophilic (keratinized) appearance. These are dyskeratotic cells. The term "dyskeratosis" is not such an evil word as one might think. *Dyskeratosis* is simply maturation out of place and independent of the normal progression of maturation of the epidermal cells. This keratinized cell, instead of being up in the stratum corneum, is down deep, near the basal cell layer. There is faulty and individual cell keratinization.

The dyskeratotic cell alone does not indicate malignant or even premalignant change. It may be present in benign diseases (e.g., Darier's disease) as well as within malignant processes (Bowen's disease). It really is not pathognomonic unless it is in association with other cellular and structural changes to form a definite diagnostic pattern.

With this brief orientation we will begin the presentation of cases.

Presentation of Cases

CASE 1

History: (AFIP 67979, 317490, 493930, 536604, 598768, 510069, 88221, 186730, 279275 and 317309)

(Contributed by Hugh Gilmore, J. B. Hartney, Herbert Derman, D. L. Galindo, William C. Butz, C. J. Farinacci, F. H. Foucar, Robert L. Breckenridge, Joseph W. Lubitz and Joe M. Blumberg)

Miss Composite, a 25-year-old white woman, had a bluish white growth about ½ c. in diameter and sensitive to touch, on the left forearm, near the wrist, for 3 years. About 6 months ago she noticed that it had gradually enlarged and had become more tender. The lesion was located in the subcutaneous tissue and did not seem to be attached to any nerve. The specimen removed consists of a lobulated subcutaneous mass 1.8 x .5 c., gray-white, mottled with areas of purple-brown.

DR. HELWIG:

Diagnosis: Eccrine spiradenoma.

Discussion: What I have to tell you about eccrine spiradenoma represents work done by Dr. David Kersting and myself in a study of 136 tumors occurring in 134 patients. It is a painful adenoma of the eccrine sweat glands. As far as we know, this lesion never has been recognized before as an entity, either clinically or pathologically. Ninety-one per cent of these sweat gland tumors are painful—either painful to pressure or tender, and sometimes there is paroxysmal pain. The lesions vary from a few millimeters to 5 cm. in diameter and most often are flat. Usually they are skin colored, but may be bluish or reddish. In our series the tumors were usually single and distributed over the upper and lower extremities, trunk, neck and face. There were no lesions on the palms of the hands or soles of the feet, none in the axilla and none on the genitalia. There were lesions on the dorsum of the hands and fingers.

(Slide) This is a characteristic low-power picture. Notice that the lesion is in the subcutaneous tissue. Most tumors occur in the deeper corium or subcutaneous tissue. Generally there is little disposition to form a thick fibrous capsule. These lesions tend to separate from the surrounding connective tissue, and often the specimen you receive will not have corium around the lesion, but will be only the "shelled out" tumor. (Slide) This is from the wedge of connective tissue between those two lobules. These tumors are commonly lobulated, and here in the wedge-shaped area are a rich supply of nerves and blood vessels and tubules of a sweat gland. There is a mild inflammatory cell infiltrate. This may occur but is rather unusual. Again you will notice the rather sharp circumscription around the periphery of the nodule.

(Slide) There are several different histological patterns. The most frequent is a diffuse pattern in which there are fine trabeculae separating cells ranging from those with small, dark nuclei to larger cells with pale vesicular nuclei. (Slide) In this second pattern there is some disposition for the cells to line up in alveolar formation. (Slide) Here is still a third formation with distinct glandular structures lined near the central part with cells having pale nuclei and toward the external layer with cells having deep blue nuclei. There is evidence of secretion in the center of this tubule. Around the periphery of the tumor the cells appear essentially similar to those we saw a moment ago in the diffuse pattern.

(Slide) Here is a margin of one of the tumors, showing the eccrine sweat gland coming right down next to it. (Slide) This is the margin of the coil part of the eccrine gland, and I would

call your attention to the similarity between the cells of the coil gland and those of the tumor.

(Slide) These tumors also show some other variations. In this one section we have three different variations, but they may, and often do, occur separately. On the left there is a very edematous zone; in this area, a cystic zone, and in another, an intensely hyperemic and congested zone. (Slide) This section is from the margin of the cystic zone, showing cells in the diffuse and glandular patterns that we saw a moment ago. The cyst occupies a large part of the tumor and is filled with a protein-like material, perhaps secretion from some of these cells. (Slide) This section is from a tumor of the hyperemic vascular type, in which the tumor cells are greatly compressed by many dilated, blood-filled channels. This lesion is often misinterpreted as a form of glomus tumor or pericytoma. It is readily distinguished from the glomus tumor by the presence of endothelial-lined vessels which are separated by cells of two different types. (Slide) This section shows marked edema with the formation of spaces filled with fluid between tumor cells. This manifestation often is mistakenly called a lymphangioma.

Diagnoses Submitted by Participants:

CASE 1

Sweat gland tumor	42%
Glomus tumor	33%
Hemangiopericytoma	8%
Hidradenoma	7%
Benign tumor of skin adnexa	3%
Neuroblastoma	2%
Miscellaneous: Benign	1%
Malignant	4%

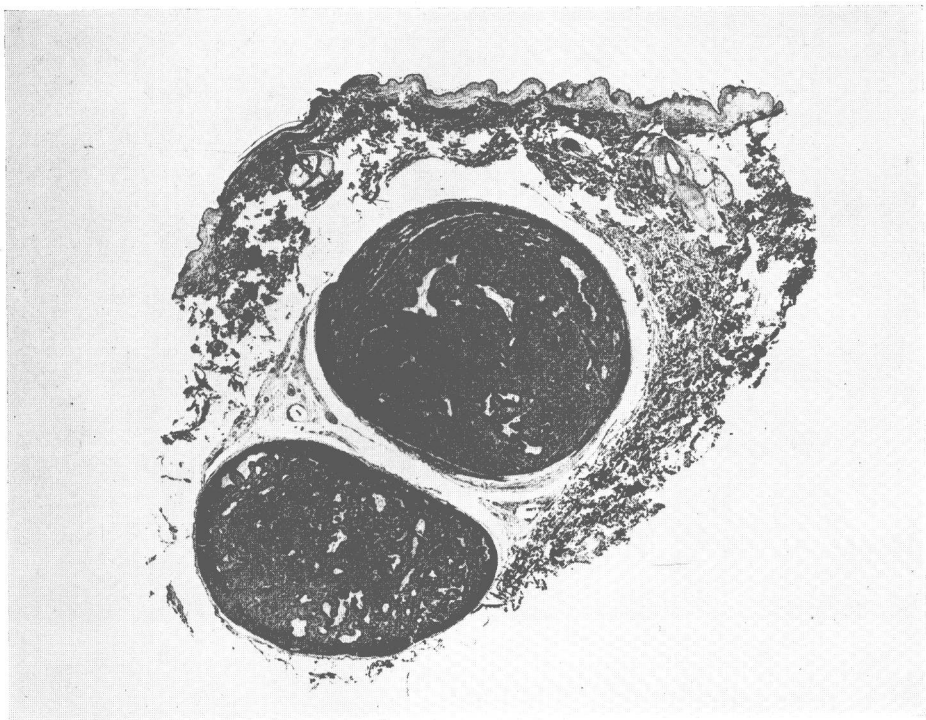


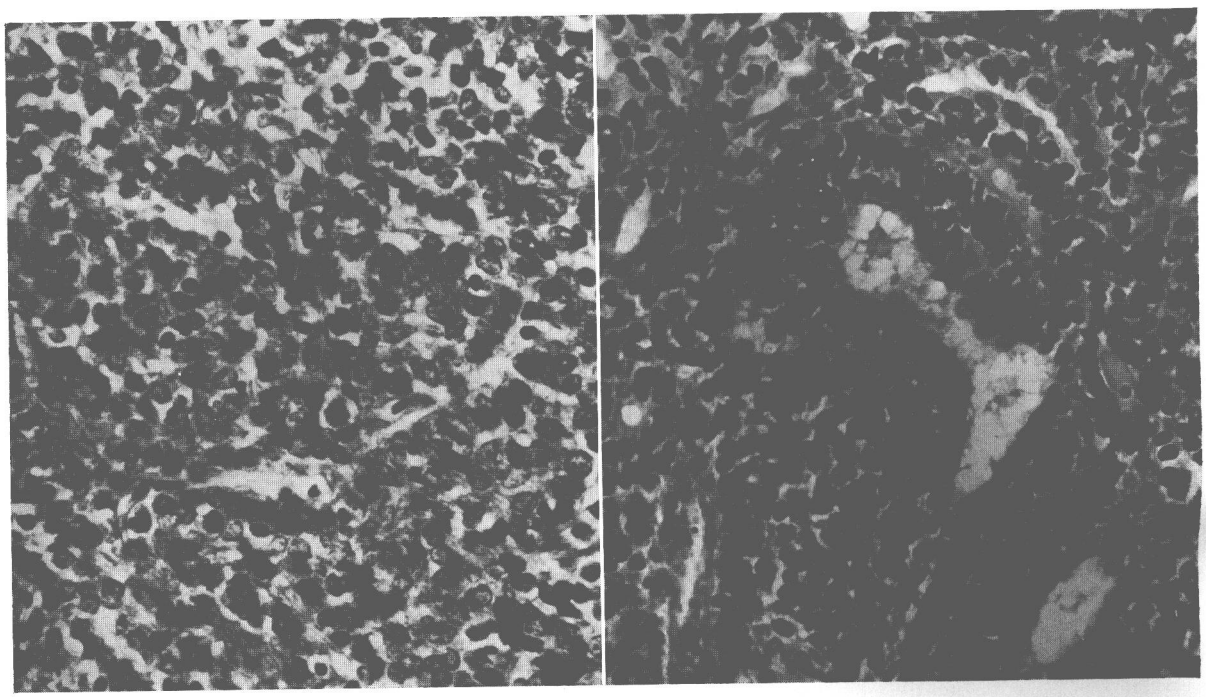
Figure 1. Eccrine spiradenoma.
The tumor is sharply circumscribed and lobulated. × 13¼. Case 1. (AFIP Neg. No. 53-12986, 3420.)

Most of you called the lesion some type of sweat gland tumor, which is a good diagnosis. About one third of you thought it was a glomus tumor, an example of which we shall see for comparison later in the day. A few believed the lesion to be malignant.

Now I would like to show you a few other lesions which sometimes are confused with eccrine spiradenoma. By the way, the spiradenoma was listed in our own files under 52 different diagnoses.

(Slide) This is the dermal cylindroma, or so-called turban tumor, which occurs chiefly on the scalp and is usually raised. There are small or large cylinders of cells in the corium extending nearly to the epidermis, which is elevated. (Slide) Under higher power, many of the cylinders of cells are surrounded by a hyaline membrane, and there is also hyalin between some of the epithelial cells—a picture distinctly different from that in the spiradenoma.

structures. In general the cells are uniform. (Slide) This is a closely related lesion and probably a variant of the "large cell type." The tumor here, though, shows vacuolization of the cells. This lesion has been called a papillary clear cell carcinoma and also a clear cell myoepithelioma. I agree with neither diagnosis. (Slide) This is a PAS preparation and the previously noted clear cells are filled with red material. If you applied diastase to this red-staining material, the color



A. B.

Figure 2. Eccrine spiradenoma.

A. The cells are arranged in a different pattern. Most nuclei are pale but some are small and dark. × 470. Case 1. (AFIP Neg. No. 53-12989.)

B. A disposition to glandular arrangement is apparent and there is evidence of secretion. × 315. Case 1. (AFIP Neg. No. 54-12494, 3561.)

(Slide) This next lesion we have called tentatively an adenoma of sweat gland of the "large cell type." We feel it is probably of eccrine sweat duct origin, but we still have considerable investigation to do on it. Even though the base appears irregular, this is a benign lesion. (Slide) The cell in this tumor is much larger than the cell of the eccrine spiradenoma and it has an acidophilic cytoplasm. In some areas a distinct tendency toward squamous differentiation may be observed. In other areas, there may be ductlike

would disappear, indicating that it is glycogen. I doubt that these are myoepithelial cells. On the rare occasions when this tumor undergoes malignant change, it is attended with cellular pleomorphism.

(Slide) Here is another example of sweat gland tumor—the syringoma. It develops more often in females and may begin at puberty as numerous small distinct papules. Much of the cutaneous surface may be involved. Microscopically it is comprised of small, cystic sweat duct structures

lined by two layers of epithelium. The inner layer is cuboidal and the outer flat. Sometimes the cysts have a tail-like process.

(Slide) This is a synovioma from the sole of the foot. It appears circumscribed and superficially simulates the eccrine spiradenoma. (Slide) Under higher power there is a distinct papillary structure with clefts lined by epithelial appearing synovial cells which cover tufts of spindle cells. (Slide) Here we can see a biphasic cellular pattern in the synovioma but not the mixture of two cell types noted in the spiradenoma. Several examples of the eccrine spiradenoma we studied had been diagnosed synovioma. This is particularly true of those tumors of the feet, hands, wrists and ankles close to tendons and joints, which have "shelled out" with a loss of the anatomic relationship. In fact, in one instance amputation of a hand was nearly carried out on the basis of a mistaken diagnosis of synovioma.

Reference:

Kersting, David W., and Helwig, Elson B.: Eccrine Spiradenoma, to be published. Archives of Dermatology. This paper was awarded first prize in the annual essay contest for research by the American Dermatologic Association.

DISCUSSION

CHAIRMAN ACKERMAN: You will notice that the diagnoses were listed in percentages. Last year I made a considerable plea to increase the number of people who submitted diagnoses. Last year we had 100, and this year, because of my plea, we got 125. I have concluded that people are people and I can't do anything about it, so all I will do is to continue to ask you to submit diagnoses.

As I have said before, we have 25 cases, and unfortunately we have very little time. As you can see, Dr. Helwig has several slides to show in each case, and I am going to limit the discussion. It isn't Dr. Helwig's fault if I am a little arbitrary at times, because he is a very sweet person and I am essentially very mean. (Laughter)

Are there any questions on this case?

DR. STRAUS (Los Angeles, California): Will Dr. Helwig discuss the malignant propensity of this tumor?

DR. HELWIG: I have not seen one of the eccrine spiradenomas undergo malignant change. Eleven of them have recurred. They recurred in essentially the same pattern, and there is a likely explanation for the recurrence—the tumor is often lobulated. The surgeon or dermatologist excised one lobule and left part of the tumor behind to recur.

CHAIRMAN ACKERMAN: We will go on to the next case.

CASE 2

History: (AFIP 128167, 163203, 164441, 505090, 481301, 526527, and 505676)

(Contributed by Joseph E. Flynn, Hans Popper, Rudolph F. Deutal, C. J. Lind, James N. Patterson, Joe M. Blumberg, Marcus R. Beck, and Roberto E. Benitez)

Mr. Composite, a 23-year-old man, first noted an oval red swelling over the angle of the right jaw about 6 months ago, which gradually increased in size. Recently there was some pain on shaving and washing. The mass was slightly movable and seemed to be attached to the deeper subcutaneous tissue.

The specimen is an irregularly shaped mass, measuring 1.8 x 1 x 1 cm. The cut surface is granular, partly gray-white and partly yellow.

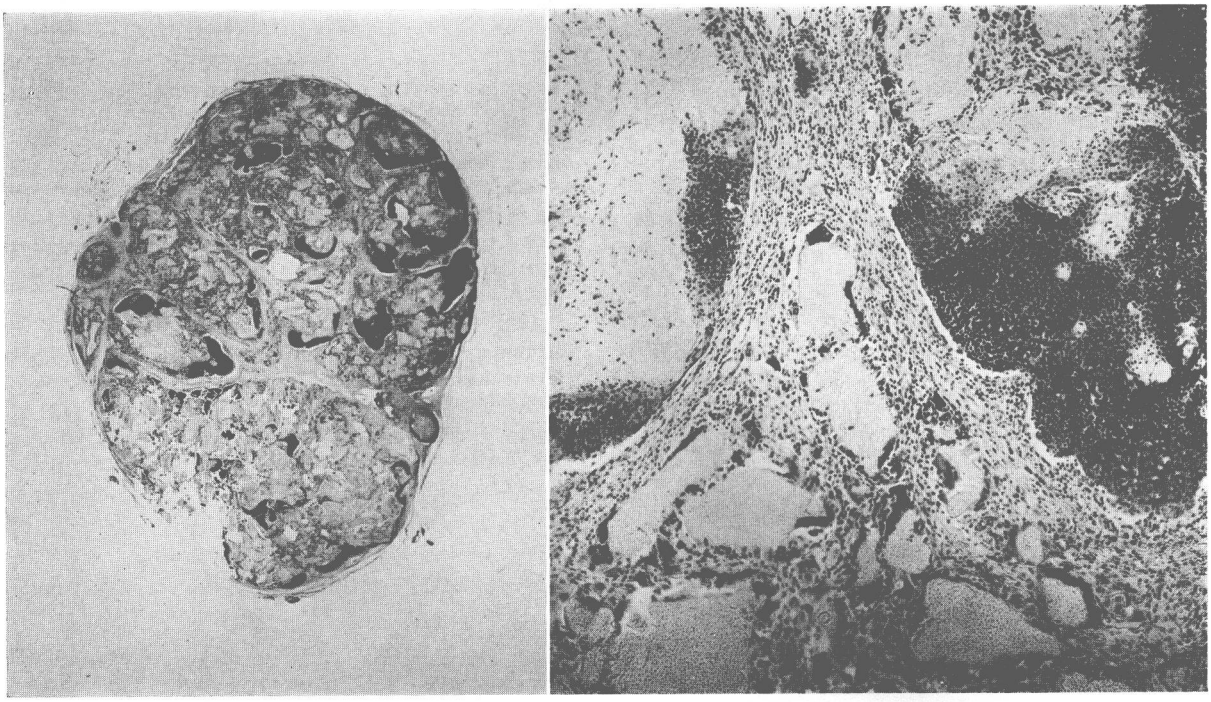
DR. HELWIG:

Diagnosis: Hair matrixoma (benign calcifying epithelioma).

Discussion: The hair matrixoma is, of course, more commonly called calcifying epithelioma. I will show you in a moment why I call it a hair matrixoma.

Clinically the lesion forms a nodule beneath the skin, which is sometimes, but not always, fixed to the skin. It commonly measures 1 or 2 cm. in diameter and occasionally up to 4 or 5 cm. It occurs on the scalp, face, trunk and extremities but not on the soles or palms. The clinical diagnosis is usually only presumptive and frequently is given as inclusion cyst.

(Slide) As you see from the low-power picture, this lesion is composed of two elements, one of

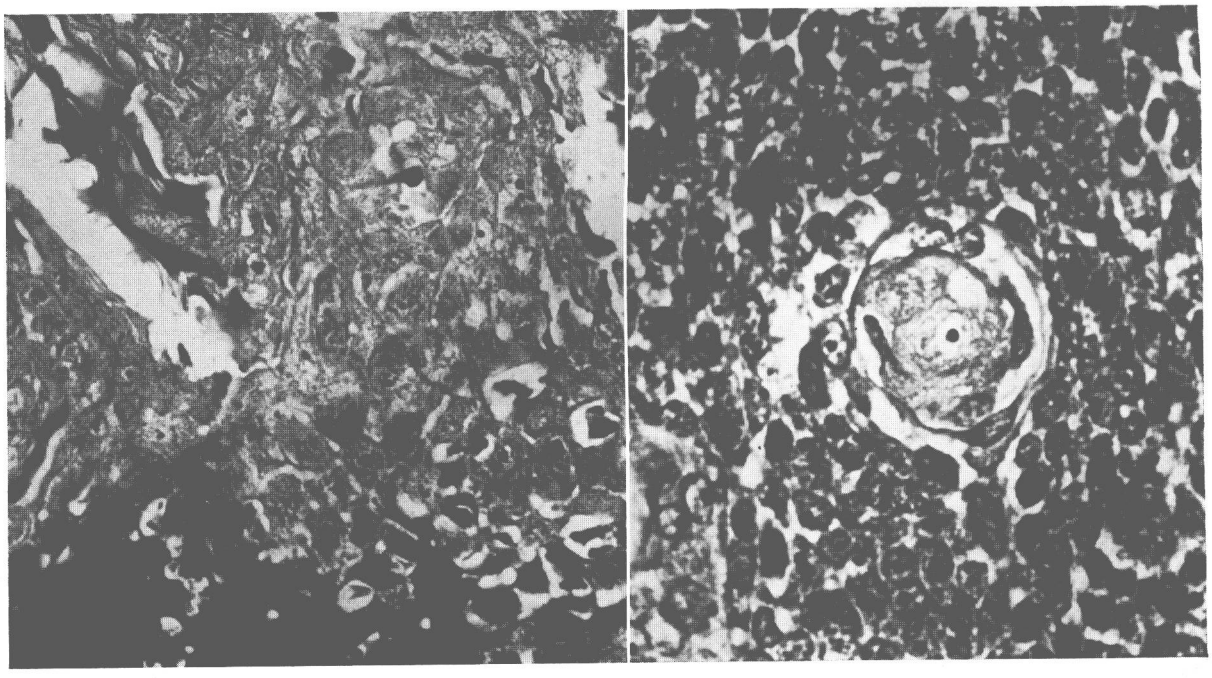


A. B.

Figure 3. Hair matrixoma.

A. It is not encapsulated. × 5¼. Case 2. (AFIP Neg. No. 54-11228, 3313.)

B. Clusters of basophilic cells are contiguous with pale shadow cells. Foci of shadow cells are surrounded by foreign body giant cell reaction. × 55. Case 2. (AFIP Neg. No. 54-11230, 3313.)



A. B.

Figure 4. Hair matrixoma.

A. Shadow cells with calcium deposition. × 475. Case 2. (AFIP Neg. No. 54-16626.)

B. Basophilic cells with central whorl of shadow cells. × 600. Case 2. (AFIP Neg. No. 54-17574.)

which stains blue, the other one pink. (Slide) In this higher power we see two types of cells —the deep blue staining cells, the shadowy pink cells with a foreign body giant cell reaction about them. (Slide) This shows an area of transition from the deep blue-staining cells in the upper left-hand corner to the shadow cells. The basophilic cells have fairly uniform nuclei and blue cytoplasm. As we follow the picture to the right the nuclei become pyknotic and then seem to break up into small fragments. The cell still exists, about the same size, with a distinct cell outline but with an indefinite nuclear outline, and finally the entire cell becomes granular, pink and shadowy.

(Slide) Many of these lesions have collars of basophilic cells surrounding or partially surrounding a central mass of shadow cells. Often there are several such masses, suggesting that the initial lesion has ruptured. There is no peripheral capsule and it is rare that a sharply circumscribed lesion is encountered. Occasionally one of these lesions recurs, simply because not all of the material was excised.

(Slide) In the basophilic areas are cells of moderate size with round and oval, fairly uniform chromatic nuclei and a rather scanty basophilic cytoplasm. Mitotic figures may be present but often are not. They do not indicate malignant change. Here is an interesting focus to speculate about. There is a rather sharp transition from basophilic cells to a minute circular collection of pink shadow cells. This possibly may represent pilar structure and a faulty attempt at hair formation.

(Slide) Many of these lesions (it depends a great deal on how carefully you examine them) contain calcium, which stains a very deep blue, as you can see here. There are fine granules of calcium in some of the shadow cells at the margin of the cell mass. Toward the center of the lesion the cells are densely filled with calcium and the structure is completely obscured.

(Slide) In addition, 15 per cent of our lesions showed the presence of bone. I have no doubt from my own experience that some lesions of the skin which are removed and diagnosed as osteoma, would, if examined carefully, exhibit shadowy outlines of epithelial cells between the bony trabeculae. Such lesions actually are hair matrixomas which have undergone ossification. Many of the tumors, and particularly those that appear to have been ruptured, exhibit a foreign body giant cell reaction in relation to the acidophilic shadow cells.

Diagnoses Submitted by Participants:

CASE 2

Calcifying epithelioma (Malherbe)	87%
Calcified epidermal inclusion cyst	5%
Epidermoid carcinoma in epidermal inclusion cyst	3%
Miscellaneous: Benign	4%
Malignant	1%

Most of you diagnosed the lesion correctly as a calcifying epithelioma. What I do not know is whether epithelioma means a benign or malignant tumor to you. In my experience the lesion we are discussing has always been benign.

Now I would like to show you why I call this a hair matrixoma.

(Slide) Here is a lesion much like the first one we saw, but which has been removed in toto with the covering skin. I would call your attention to the break in the epidermis which, I think, is a fairly typical follicular opening. The rest of the lesion is comprised of cells of two types—the pale shadowy cells and basophilic cells which I interpret as hair matrix cells. (Slide) On closer inspection of this follicular opening, we see that the squamous epithelium of the epidermis is continuous with that of the neck of the follicle. A short distance down the neck of the follicle the epithelium shows an abrupt transition. (Slide) Under higher power at the point of transition we see trichohyalin granules and basophilic cells. The squamous epithelium seen in the neck of the follicular opening probably corresponds to the external root sheath, the cells with keratohyalin granules to the internal root sheath, and the basophilic cells to the hair matrix cells. (Slide) This is another area from the same tumor, showing that it is a bona fide hair matrixoma—basophilic cells on the right with a gradual transition to pale shadow cells on the left.

(Slide) This is another bit of evidence of the relation of the lesion to the hair matrix. Here is a tumor which does not show any evidence of rupture. The cells around the periphery are baso-

philic, with shadowy cells in the center. Now I would like to show you this area from within the tumor. (Slide) This looks very much like an overgrown papilla of a hair and is comprised of connective tissue. As you can see, there is melanin pigment. (Slide) Under higher power, we can see melanin pigment in the connective tissue cells. At the junction of the connective tissue and the epithelium, melanin pigment is also present in the epithelial cells, which are basophilic in appearance. As the cells progress away from the junction they become pink shadow cells. To me, this pigment distribution looks very much like that which normally occurs at the hair papilla. These two anatomic changes, plus the facts that many of the lesions are not calcified and that epithelioma indicates or implies a carcinoma, I believe, are reasons enough for changing the diagnosis of this lesion to hair matrixoma.

(Slide) Now I shall show you a few other lesions. This is an epidermal inclusion. Calcification is also going on here, and the cyst or inclusion has ruptured. (Slide) This shows the epithelial wall of the inclusion and with strands of keratin along the inner margin. There is calcification. The cells, to be sure, become indistinct as they undergo keratinization, but their appearance is entirely different from that of the shadow cells of the hair matrixoma. Calcification does not make the diagnosis of hair matrixoma.

(Slide) This is another lesion which was called a squamous cell carcinoma, and I feel very strongly that it is nothing but an epidermal inclusion in which acanthosis or epithelial hyperplasia has occurred. It is partially circumscribed in some areas and elsewhere poorly defined peripherally. (Slide) There is somewhat of a lobulated pattern through the entire lesion. If we follow from the margin of one of the lobules toward the center area where there is keratin, there seems to be a normal progression of maturation of these epithelial cells—not a distorted pattern as would be expected in carcinoma. (Slide) There is no appreciable pleomorphism of these cells. They look very much alike throughout the entire lesion.

(Slide) Here is another epidermal inclusion which sometimes is called squamous cell carcinoma. In some areas there is piling up of the epithelium into the cyst lumen, but most of the lumen is filled with strands and flakes of keratin. (Slide) This section is taken through the nodular epithelial hyperplasia of the cyst wall. Under still higher power there is a certain epithelial pattern which we have called squamous eddies, and it is carried throughout all of the epithelial proliferation. These small repetitious squamous eddies are distinctly different from the pearls of squamous carcinoma. Again there is no appreciable pleomorphism. The cells in some places are aligned in different directions, but comparing cell for cell and nucleus for nucleus they all look about the same. Sometimes when a cyst ruptures and this material gets outside, it gives a really startling appearance, and you think, "This is really it—a squamous cell carcinoma." I have never seen one of these infiltrate. This lesion probably accounts for most of the reported examples of carcinoma arising in sebaceous cysts.

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DISCUSSION

CHAIRMAN ACKERMAN: Is there any discussion of this case? May I ask Dr. Helwig a question: What is the age incidence of these lesions? Have you often seen them in children?

DR. HELWIG: As far as the age incidence is concerned, out of 180 of these lesions which I studied, 15 per cent occurred in children under 10 years of age and the oldest patient was 73. Some infants apparently were born with them, and one lesion was removed at the age of 2 months. These tumors from the young age group are the ones that usually get the pathologist excited. They tend to have more of the basophilic cells and fewer shadow cells. Maybe this variation is only a matter of duration. The large amount of basophilic cells arouses the suspicion of carcinoma.

I might add that very early in the preparation of this Seminar I got out one of these lesions and thought I was very fortunate. It measured about 5 cm. in diameter and was large enough to make 1400 sections. The sample section showed some areas of calcification, some areas of the basophilic cells and some foci of shadow

cells. I cut seven or eight blocks, each of them 3 or 4 mm. in thickness and sent them to the laboratory. I thought, "Well, I have finished with this." When I got the samples back, most of them were comprised only of calcified material and a few shadowy cells. I did not find any, or only a few of the basophilic cells, which are the cells that may be disturbing. It would appear on the basis of this single experience that these lesions may grow more in one area than in others.

Although I have proposed the term hair matrixoma for this tumor, the concept that these tumors are derived from the hair matrix has also been advanced by other workers; Turhan and Krainer and Highman and Ogden have noticed the resemblance of the cells to those of the hair follicle.

CHAIRMAN ACKERMAN: We will go on to the next case, No. 3.

CASE 3

History: (AFIP 538494, 331097, and 525528)

(Contributed by Joseph Mendeloff, O. J. Wollenman, Jr., and C. J. Lind)

Mr. Composite, a man aged 59, hit his head on a door. Approximately 2 years later a lump appeared on the forehead near the midline and the bridge of the nose. The lump at first was red but gradually became paler and larger, reaching a diameter of 1.5 cm. in a 3 year period. It caused pain only when pressed, was firm and was fixed to the underlying soft tissue.

DR. HELWIG:

Diagnosis: Cutaneous mixed tumor (Salivary gland type).

Discussion: This low-power picture includes a slightly irregular and elevated, but otherwise normal, epidermis which covers the corium. Several normal hair follicles and sebaceous glands are present in the corium. In the deeper corium is a fairly well-circumscribed but non-encapsulated tumor compressing the surrounding stroma. A connective tissue septum separates the tumor into two lobules. There is quite a variation in the structural pattern in different areas. (Slide) Under higher power, in some areas there are irregular glandular structures, occasionally lined by a double layer of epithelium suggesting a sweat duct structure; in other areas, epithelial cells occur in solid sheets, and in still others, the epithelial cells appear continuous, with stellate shaped cells embedded in a mucoid stroma.

(Slide) Here is the same general pattern of cells and stroma, but the stroma appears more tenuous. A projection of epithelial cells streams into one of these tenuous, almost myxomatous, appearing areas. The epithelial cells seem to be proliferating into the stroma and become separated. Notice the close similarity between the cells in the stroma-like foci and those in the solid nests of epithelium. The point I am trying to make is that the cells in the loose tenuous foci are not all connective tissue cells; many are epithelial cells which become elongated and look like connective tissue cells. (Slide) In this section the same process is seen about the periphery of the myxoid area. But toward the center are some cells that have a nucleus surrounded by a clear halo, suggesting a cartilage cell. I do not believe they are cartilage cells. I believe they are epithelial cells.

Diagnoses Submitted by Participants:

CASE 3

Mixed tumor of skin	64%
Syringocystadenoma papilliferum	13%
Sweat gland tumor with chondral metaplasia	9%
Malignant sweat gland tumor	3%
Hidradenoma, mixed type	2%
Miscellaneous: Benign	4%
Malignant	5%

I would like to say a word about hidradenoma. I doubt that hidradenoma in itself without qualification has the integrity of an entity. The term has been used so often to include a number of entirely different tumors derived from the sweat glands that it no longer carries a specific meaning. Furthermore the various tumors to which it has been applied have different natural histories.

Several of you regarded this tumor as malignant. Of approximately 100 mixed tumors of the skin which we reviewed, all were of the salivary gland type and, like that which you just saw, all have followed a benign course. Most of the tumors occurred on the face (nose, lip, cheek) and on the scalp with a few elsewhere on the body.

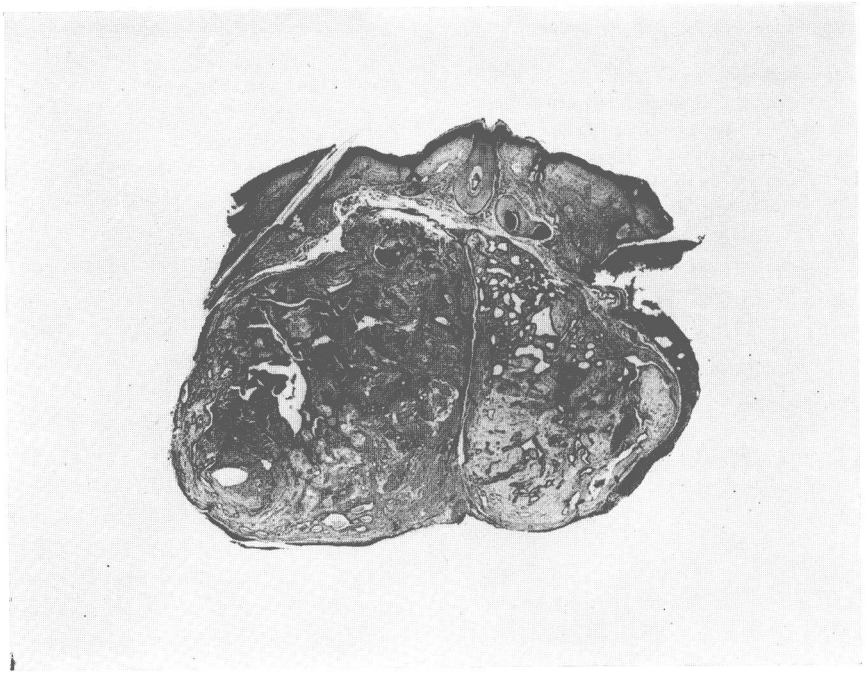
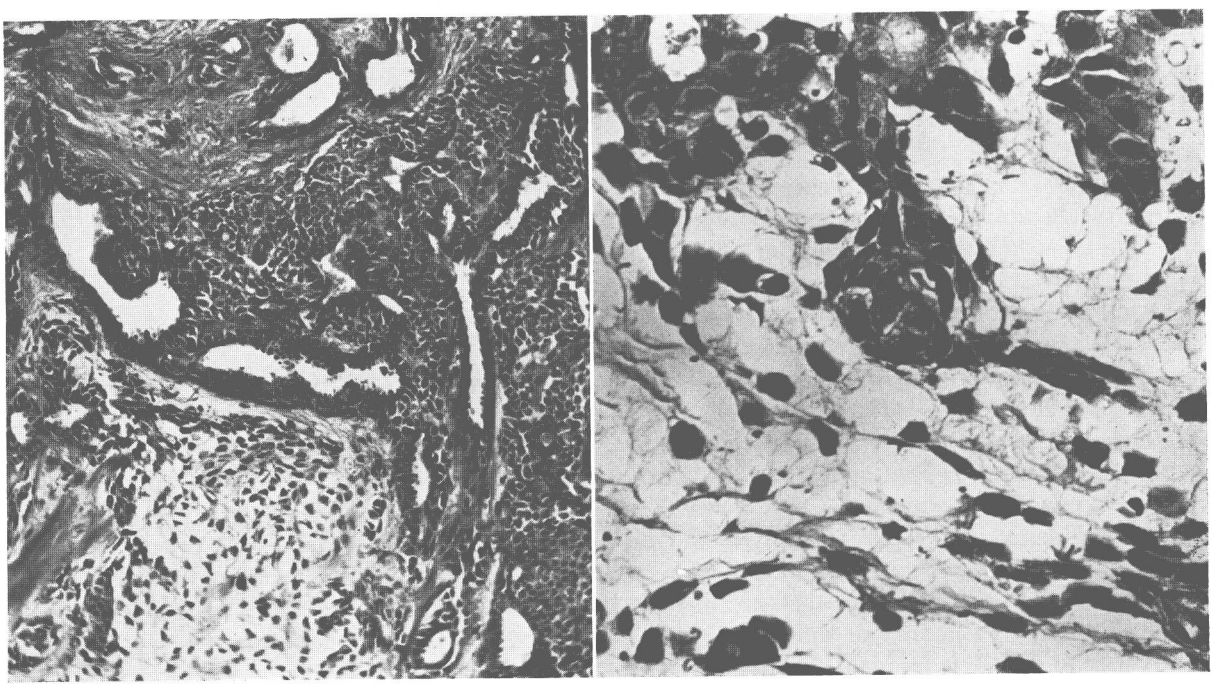


Figure 5. Mixed tumor of the skin, salivary gland type. × 12. Case 3. (AFIP Neg. No. 54-8649.)



A. B.

Figure 6. Mixed tumor of the skin, salivary gland type.

A. Glandular structures, some with a double layer of epithelium and myxoid area. × 118. Case 3. (AFIP Neg. No. 54-12655, 3648.)

B. Epithelial cells streaming into myxoid area. × 395. Case 3. (AFIP Neg. No. 54-12659, 3648.)