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REVIEW OF URINARY TRACT INFECTIONS

ROBERT H. DILCHER, M.D.
and
FRANK CAPABIANCO, M.D.

THE supposition that infections were of animated origin was held by many of the so-called "thinking men" for ages; some of their thoughts were accepted and others laughed at. Many of the suppositions were boldly experimented with crudely and proved correct and others were found to be false. As early as the 5th century B.C. various learned men attributed the origin of animals to the soil.

Much of the original work in the genito-urinary infections dealt with venereal diseases. Gonorrhea was mentioned six times in the Bible. The Greeks had a lot of gonorrhea. Cleopatra recommended anterior urethral injections and also a cautery for penile lesions. At that time it was also recognized that these conditions were contagious. Catherine the Great of Russia died of syphilis. The term "clap" came from the French clapiere which referred to a house of prostitution, a low type frequented by sailors. The case of John Hunter is exemplary when in 1767 he innoculated himself with what he thought was a gonorrheal discharge, only to develop syphilis, which for over 50 years following this experimentation was accepted with gonorrhea to be a common disease. It was not until after the time of Lister in the 19th century and until early in the 20th century that urinary tract infections were more intensively investigated, and it was after this time that all other bacteria were found in the course of studying urines. As culture methods have been refined, various different bacteria were isolated and sub-groupings of these bacteria isolated.

There are certain etiological factors which are generally applicable to all urinary infections:

ETIOLOGY

(1) **Age:** There is a gradual increase of urinary infections in infancy which reaches its peak at 18 months of age to subside and again reach a peak from 30 to 50 years of age.

(2) **The female of the species** seems to have a greater incidence of urinary infection than the male, approximately 3 to 1. This may be attributed to the fact that the female baby may develop an

urethrocystitis due to fecal contamination of the diaper. This also may be the etiological factor in chronic urethritis in the adult woman. With reference to hematogenous infections the female is about equal to the male in incidence.

(3) **Heredity and environment:** Malnutrition predisposes to infection by lowering the general resistance of the individual.

(4) **Season:** During the summer gastro-intestinal upsets are much more frequent and therefore there is an increase of urinary infections due to the Colon Bacillus. During the winter, upper respiratory infections are much more common. Therefore, we see more urinary infections due to coccal organisms.

(5) **Mechanical factors:** Stasis and obstructions are the most important factors. Under this grouping we have—

(a) **Congenital anomalies and malformations.** Congenital anomalies and malformations occur in an incidence of 2–13 percent. A recent paper by Barns demonstrated common anomalies at the ureteropelvic juction with high insertion of the ureter above the most dependent portion of the renal pelvis and intrinsic narrowing of the ureteropelvic junction. A third abnormality was that of aberrant vessels and fibrous bands compressing the ureter just below the ureteropelvic junction. Wharton and his group studied a large group of patients in follow-up studies, patients who had been former admissions of the Pediatric Department at Johns Hopkins. They divided the groups into those who had had only one attack of pelitis in childhood and those who had had more than one attack. In the first group approximately 13 years later, they found three with normal urinary tracts and six with slight or definite pathological changes in the urinary tract which included dilated and redundant ureters with ptosis, renal calculus and slight hydronephrosis. In the second group, followed from 3-14 years after their original visit, about 50 percent still had urinary tract pathology, including infantile shrunken kidney, infantile kidney, ureterectasis, pyelectasis and frank hydronephrosis. In an over-all picture in persistent pyuria, anomalies are found in about 40 percent of the cases.

(b) **Urethral pathology:** As we shall see by diagram and in later discussion, there are many points of possible obstruction to drainage along the entire urethra beginning with the meatus and constriction at the fossa navicularis to the posterior urethra and internal sphincter.

(c) **Bladder:** (1) The prostate is obviously a point of obstruction to adequate drainage to the bladder in men past 40 years of age to a varying degree. In the fifth decade we see those cases with early median bar formation and contracture of the vesical neck. Later we see benign and malignant enlargement of the prostate obstructing the bladder neck. (2) Urethritis with stricture formation is common in the female adult. Involvement of the posterior urethra with chronic irritation and polypoid formation with edema around the internal sphincter leads to fixation of the urethra and narrowing of the urethral lumen. Also in female patients the stasis produced by a cystocele or undermining of the trigon leads to chronic exascerbation of existing infections and an ideal nidus for new infections. (3) Foreign bodies: The presence of any foreign body in the bladder such as a calculus or blood clot or introduced foreign body in perverted sexual practices leads to infection of the bladder mucosa. (4) Congenital or acquired diverticula and tumors of the bladder are ideal points for infection. (5) Trauma: Trauma may be due to external blows to the bladder region or may occur after instrumentation of the urethra and bladder. (6) Adynamic bladder: Certian trauma may contribute to an adynamic bladder directly or after operation on the pelvis or in dissecting out the rectosigmoid. There will be enough reflex involvement of the sympathetic nervous system to result in a temporary or permanent adynamic bladder. Involvement of the central nervous system may occur and produce the adynamic bladder from trauma to the vertebral column or by diabetes, tabes dorsalis, pernicious anemia, paralysis agitans, poliomyelitis, meningitis, and congenitally with the spina bifida occulta. An adynamic bladder may also be produced from chronic long-standing obstruction due to bladder neck obstruction.

(d) **Ureteral obstruction.** This may result in simple atony or may be due to an inherent neuro-muscular disease. It is usually unilateral. It may essentially be due to ureterovesical obstruction at the ureteral meatus or intra-mural obstruction of the ureter.

(e) **Renal obstruction.** Renal obstruction may be in the form of ureteropelvic obstruction by adhesive bands, aberrant vessels or high implantation of the ureter on the pelvis. Also important in renal obstruction is ptosis of the kidney which in its dependency may cause angulation of the ureteropelvic junction and stasis of the urine.

(6) **Toxemia.** Naturally any systemic infection may contribute to urinary tract infection. Any acute upper respiratory, pulmonary, gastro-intestinal, or skin infection may produce a temporary hematogenous invasion and thereby produce renal infection.

ROUTES OF INVASION

(1) **Hematogenous.** The most likely method of urinary tract invasion by bacteria is through the hematogenous route. There are many possible foci in the body such as teeth, tonsils, ears, sinuses, osteomyelitis, boils and carbuncles, etc. which may produce temporary bacteremia or septicemia and lead to renal invasion. The vascular supply of the bladder is closely associated with the ureteral, ovarian and renal blood supply by numerous anastamoses of vessels, thereby infections which are prevalent in any one area may readily be spread to the bladder. There are five well recognized vascular invasions of the ureter from its ureteropelvic junction to the bladder, all of which anastomose and all of which are capable of spreading infection.

(2) **Ascending.** Ascending infections are not common, unless by direct extension from the urethra to the bladder by instrumenation or in the case of a neurogenic, adynamic bladder or ureter with a patent ureterovesical orifice, back pressure may cause ascending infection. There is epithelial contiguity which may allow spread of infection.

(3) **Lymphatics.** Lymphatic spread is also uncommon. The lymphatics of the lower ureter, bladder and posterior urethra all empty into the hypogastric nodes. Those of the kidney, upper ureter and the perirenal fat enter into the peri-aortic nodes. There are also a group of intra-mural lymphatics which are capable of spreading infection.

PATHOLOGY

In considering infections of the penis and the urethra it is essential that we recognize the fact that there is squamous epithelium covering the glans penis and the first portion of the urethra to the fossa navicularis, after which the epithelium becomes a columnar type. The squamous epithelium is quite resistant to infections including the gonococcus. In the case of uncircumcised males where there is a long redundant foreskin the epithelium of the glans is not of the squamous type and may very easily become involved in a development of a balanitis or a meatitis at the urethral meatus. There are several glands on the outer surface of the penis which may be involved, such as the gland of Tyson or the parafrenal glands. There are also the paraurethral sinuses or dimpling about the urethral orifice on each side. These are more commonly seen in the hypospadiac urethras. These glands are lined with columnar epithelium and are prone to pick up infection. Along the course of the urethra are the glands of Littre which very readily become involved both in specific and non-specific

urethritidies. As we go back through the urethra we come to the opening of Cowper's glands and then into the posterior urethra where the many glandular openings of the prostatic ducts exist. Involvement of the posterior urethra very readily causes extension of the infection into the prostatic ducts and results in a prostatic infection. The verumontanum and utricle lie in the posterior urethra and very easily become infected. Infection may extend from the verumontanum along the ejaculatory ducts into the seminal vesicles or down the vas into the epididymis and indirectly may involve the testicle with any acute infection. These infections are all of a descending type extending from the posterior urethra. We may also have hematogenous invasions of the prostate, epididymis and testicle. Infections involving the bladder are usually limited early to the trigon and interureteric ridge. The trigon of course is the pushbutton mechanism of the bladder and any irritation of this area results in dysuria, frequency, or urgency. Involvement of the trigon or seminal vesicles which closely approximate the ureterovesical junction extra-vesically may result in chronic obstruction at the ureterovesical junction and produce back pressure to the upper urinary tract.

In pyelonephritis the earliest changes occur in the cortex in the (1) hematogenous infections with acute involvement of the glomeruli where the blood vessels are filled with bacteria. The infection soon extends to the medulla causing suppuration in the tubules and peritubular spaces. The infection spreads to the pelvis and subsides under treatment or may localize in either pole with abscess formation. In (2) the ascending infections which involve the pelvis in static urine, the infection may extend up the tubules or venous channels. The end picture is the same as that of the hematogenous route. (3) The carbuncle is a cortical localized abscess usually arising from coccal infections. (4) Septic infarcts may occur when infected emboli lodge in the kidney and lead to later abscess formation.

All of these infectious processes produce some damage. Early it may completely regress with adequate therapy, however, longstanding cases or repeated insults may result in chronic pyelonephritis, atrophic pyelonephritis, infected hydronephrosis, pyonephrosis or calculous pyelonephritis or pyonephrosis.

BACTERIOLOGY—TREATMENT

1. Prophylactic—Precystoscopic therapy, whenever possible, is essential.
2. Active

With the advent of a variety of new, powerful and sometimes debilitating and damaging drugs, it behooves us to have a systematic and logical as well as a scientific approach to the treatment of urinary infections. To go ahead and treat the patient on the history and symptoms or by telephone without making certain infection actually exists is wrong and possibly harmful from a drug standpoint because of possibly sensitizing the patient to the particular drug or producing toxic effects of the drugs themselves. There are many urological conditions which symptomatically simulate infection. Therefore, after satisfying ourselves by urine examination that pus or bacteria is present, a culture should be done to identify the organism. A gram stain may give valuable aid but is not as satisfactory as the culture. The patient may then be started on the drug of choice, usually a triple sulfonamide in gram dosages, q.i.d. After the report of the culture is received, the medication may be changed, if indicated, or maintained if improvement has occured. The new triple sulfonamides are still cheaper than the antibiotics, tolerated fairly well and have a low crystalluria index. They are effective against coccal and coliform infections and work best in an alkaline urine. Toxic reactions must be watched for, particularly in the aged and debilitated.

The following is a list of some of our present day armamentarium and its indications:

DRUGS

	Penicillin	Sulfonamides	Mandelic Acid	Streptomycin	Aureomycin	Chloromycin	Terramycin	Gantrisin	Mapharsen	
Gonoccus	+	+		±	+	±	+			Gonococcus
Streptoccus	+	+		+	+		+			Streptococcus
Staphyloccus	+	+			+	+	+			Staphyloccus
Strep Faecalis			+		+	+		±		Strep Faecalis
B. Coli		+	+	+	+	+	+	+		B. Coli
Proteus Vulgaris		±				+ Gantrisin		+		Proteus Vulgaris
Pyocyaneus			±	+			±	+		Pyocyaneus
T B				+ Pasa						T B
Abacterial Puria									+	Abacterial Puria

+ Good
± Variable
Pasa Paramino Salicylic acid

In the event of recurrent or protracted cases, cultures should be repeated and sensitivity tests done, in order that the drug which appears more specific may be resorted to. It must be understood that bacteria which are highly susceptible to a particular drug will not necessarily work that way in the human body.

The wide spectrum antibiotics have a rather generalized usefulness but are expensive and do have the side effects of pruritus, dermatitis and gastro-intestinal upsets.

The following three cases are examples of urinary tract infections which did not respond to the usual therapy and after complete urological investigation, satisfactory explanation of inadequate response was found:

D.G. Age: 12 weeks, No.266,573, Admitted: 5-16-51, Discharged: 6-16-51

Diagnosis: Hydronephrosis, pyonephrosis and hydro-ureter with non-functioning kidney, right.

HPI: Since birth a gelatinous discharge was noticed following voiding with one episode of bloody discharge. The child cried on voiding.

Phys. Exam: Negative.

X-Ray Studies: A cystogram revealed an excentric abnormality of the bladder contour and a ureteral reflux on the right side with a large dilated ureter and questionably dilated pelvis. I.V. urogram showed good function of the left in 10 minutes with good emptying on the left in the 60 minutes and no function on the right. Cystoscopy revealed a right ureteral orifice larger than normal, sclerotic in appearance and more laterally placed. No urine was seen to spurt from the orifice.

Laboratory: The urine showed a trace of albumin with a positive culture for E. coli, microscopically negative, B.U.N. 25 mgm%.

Operation: The patient was assumed to have a congenital anomaly of the right upper urinary tract and under ether anesthesia a right nephro-ureterectomy was done because of the operative findings of an atrophic hydronephrosis and hydroureter.

R.S. Age: 14, No.217,390, Admitted: 11-15-46, Discharged: 11-17-46

Diagnosis: Stricture, ureterovesical orifice, congenital with hydro-ureter and hydronephrosis.

HPI: The patient was seen in May of 1946 with left sided lumbar pain, chills, fever and microscopic hematuria and pyuria. Two other attacks occured before admission to the hospital.

Phys. Exam: Entirely negative.

X-Ray Studies: The urogram showed a dilated tortuous, left lower ureter. Retrograde pyelograms showed extensive dilations of the lower third of the left ureter, constriction at the lower and mid portion of the ureter. The upper third of the ureter and the pyelogram showed only slight dilation.

Cystoscopy: Cystoscopy revealed an enlarged left ureteral ridge and edema around the left ureteral orifice.

Subsequent ureteral dilations left the patient asymptomatic.

W.W. Age: 15 years, No.218,908, Admitted: 1-19-48, Discharged: 3-22-48

Diagnosis: Kidney tuberculous, left.

HPI: The patient was in good health until just before admission when he complained of burning in the urethra and suprapubic pain. There was no response of pyuria to sulfonamides and penicillin.

Phys. Exam. The patient apparently well developed and nourished. There was slight suprapubic tenderness.

X-Ray Studies: Pyelograms showed a moderate degree of hydronephrosis of the left kidney with hydro-ureter.

Laboratory: Urine culture and guinea pig positive for tuberculosis.

Operation: The patient was prepared with streptomycin and chalmoogra oil for 45 days after which a left nephrectomy was done.

SUMMARY

The cases just presented are cases where apparently simple urinary infection exists but the expected response is not forthcoming under supposedly adequate treatment. Again we wish to emphasize that patients who do not respond promptly on the accepted adequate therapy, be investigated by culture urography and cystoscopy to rule out other causes of indequate response.

CANCER OF THE LARGE BOWEL; EARLY DIAGNOSIS AND TREATMENT

MARTIN SELER KLECKNER, M.D., F.A.C.S.

CARCINOMATOUS lesions of the large bowel include all malignancies from the ileocecal junction to the anal orifice. As a proctologist trained primarily in general surgery, I think the management of colon cancer is being fairly well treated, but it is capable of improvement.

In presenting personal data showing the distribution of cancer in the various parts of the large bowel, it is interesting to note that the vast majority lie in the distal half; that is between the splenic flexure and anus. Fortunately most of the cancers of the colon and rectum produce early symptoms such as change in normal bowel habit, the presence of blood and mucus in the stool, and if the lesion is higher in the colon, oftimes abdominal cramps or pain. The presence of marked anemia and loss of weight (while terminal symptoms) are ofttimes due to right colon involvement. These symptoms are frequently diagnostic at times, but demand your further investigation.

In a series of (565) malignancies of the large bowel taken from my own private practice and covering a period of 15 years, I have noted some interesting findings which may prove of vital importance to you. My results compare most favorably with those obtained at other large clinics throughout the country and I wish to submit some of these facts.

Because carcinoma of the colon and rectum invariably produces early symptoms, I urge you to investigate all patients earlier, particularly when they show some of the above mentioned symptomatology. I want to plead with you that you regard every one of these cases with almost convincing suspicion until you have diagnostically ruled out cancer. Your best and foremost thought is to regard each patient as being one of your most intimate friends or a very dear member of your family, and that his or her life may be at stake.

A thorough history taken of these patients will prove most helpful. We must be cognizant of the fact that cancer is not a respector of age but is found in all ages, though most commonly in the 5th and 6th decades. It has been my experience that change in the normal habit of the bowels is the earliest and most frequent diagnostic sign, in which a variation from the usual daily bowel function occurs,—slight though it may be at first, but elicited on questioning the patient. When the lesion is well within the reach of the gloved finger, more or less tenesmus may be present and then the patient may have many calls to stool which he or she erroneously believes to be a diarrhea. The elimination usually

consists of mucus, blood, pus and very little feces combined with more or less flatus. Other patients may show a tendency toward a progressive tightening of the bowels which they call a constipation. Such complaints should put the examining physician (which is usually the general practitioner) on his guard. When they tell you that they have a diarrhea, you must remember that Amoebic and Bacillary Dysentery, Malarial presence, Helminth infestation, chronic Ulcerative Colitis, etc. show like symptoms. Other patients may note an increasing constipation or they may tire easily. Loss of weight and strength are invariably late symptoms in cancer of the large bowel and make the prognosis more hazardous.

When blood in the stool is the predominant sign, always remember that internal hemorrhoids and anal ulcer (fissure in ano) are a common cause but do not forget this fact—that hemorrhoids or piles (as they are known to the laity) are invariably present when the cancer is located within the terminal 10 inches of the G.I. tract, whereas cancer is only occasionally present when hemorrhoidal pathology exists. It is by digital examination, the use of the proctoscope, and the sigmoidoscope, that these lesions can be determined and specimens taken for biopsy and pathological examination properly interpreted. Beyond the range of the scope, roentgenologic study is necessary to discover possible bowel lesion. The use of the double contrast barium enema is the best method to determine the presence of any bowel lesion beyond the range of the sigmoidoscope. Sigmoidoscopic study is far more effective and dependable than any X-ray taken in these terminal 10-12 inches because of the ability to see these lesions with the naked eye through the lighted scope. It is frequently advisable to repeat these studies if any doubt exists and in the hands of the competent roentgenologist such consultation is invaluable. If possible and for most accurate study, thorough cleansing of the bowels by castor oil and enema is necessary. When polyp is present (and it is usually the precursor of cancer), roentgen study by the above method may be required at weekly intervals to definitely mark its exact location.

Where its possible that every patient having the above-mentioned symptomatology be subjected to proper study and investigation, I am certain that the operability of cancer of the rectum and colon would be materially improved, the mortality rate would be decreased and the non-recurrence rate would similarly be raised.

The question is frequently asked, "How long has this cancer been present?" Those of you capable of sigmoidoscopic study realize and know that polypoid formation may occur within a very few months and that

its rate of growth varies. In general, the younger the individual, the more rapid the growth and the quicker the metastasis. Cancers of the rectum and colon, as a general rule, bear a common resemblance. They are rather hard, with an excavated center and their edges give it a cauliflower feel and appearance, which is almost diagnostic. Usually 9 to 12 months elapse before the cancerous growth reaches the doctor's office. If all these growths could be found prior to this period, the prognosis would be particularly gratifying to the surgeon and more hopeful for the patient. There are instances where the passage of blood has been noted for as much as two years before the doctor is consulted and malignancy is diagnosed. When other rectal pathology may be minimized or ruled out, we may assume that the bleeding came from a polyp which later under went malignant degenerative change. Otherwise such a lesion could not have remained operable over such a long period of time with the presence of blood in the stools.

Cancer of the right bowel, as compared to lesions on the left side, is characterized by greater anemia, earlier loss of weight and strength, and if located in the cecum or hepatic flexure may simulate appendicitis or gall bladder lesion. In spite of these symptoms, operability of right sided lesions oftimes offers better prognosis than will similar signs noted on the left side when such symptomatology is present but exists in more advanced conditions.

Cancer of the large bowel presents its greatest problem in that the malignant growth has been permitted to advance "too far" in too many patients to permit of good prognosis. No one disputes the fact that a good surgeon with the aid of competently trained assistants and experienced anesthetists cannot help but get the best results when doing colonic surgery whether the malignancy is fairly well localized or even far advanced. Good percentages of cure and a low death rate can readily be assured if we draw "a definite line of operability". We have improved and modified our pre-and-post operative care so that today we can help those whom we regarded as hopeless 15 years ago.

While the truth still prevails that curability of cancer varies inversely with the duration of the disease, we have experienced greater success in surgical accessibility to all tumors. Radiation treatment of large bowel lesions is questionable and but for its palliative and psychologic effect on the patient, its use has been limited by me. Most colonic cancers are fairly accessible to surgery but as we approach the rectum their removal becomes a more difficult problem requiring extensive and special surgical skill. Operation for cancer demands the excision of the lesion together with a generous portion of normal tissue surrounding the

growth and all the lymphatic involvement which is present in variable degree. Lymphatic spread is definitely upward in rectal cancer with a very small percentage extending distally, until blockage occurs above the cancer and then the retrograde downward spread may be rapid. Because lymphatic drainage in all large bowel cancers is of such vital importance, it is urgent that we remove sufficient tissue to obtain a successful result. The preservation of the sphincter mechanism to my mind is only justifiable if the lesion in the rectum is so high that all the involved malignant tissue may be completely removed and proper anastomosis performed. I have found the modern Miles one stage technique where the combined abdomino-perineal operative procedure is done, to be the most satisfactory operation for rectal cancer.

It must not be forgotten that the presence of obstruction of the large bowel may demand immediate and palliative measures, such as an ileostomy, cecostomy or a transverse colostomy—dependent on the location of the obstructing lesion. This is often a life-saving procedure but will permit of later operation when the acute condition has subsided and the patient becomes a better risk for more radical abdominal approach.

A well placed and trained abdominal colostomy is more readily handled and more comfortable to the patient than most perineal colostomies or sphincter preserved operations which I have had occasion to see. My patients are taught to irrigate their colostomies at regular times and never wear any bag or appliance. They are absolutely comfortable and there is no offensive odor associated with the patient if the established routine is followed.

I wish to mention briefly the important pre-operative stages in these cases. Complete serologic study and "build up" of the patient by transfusions of whole blood, plasma, glucose, amino acids, etc. are mandatory if one wishes to have his patients in the best possible condition for these operative procedures. This requires a minimum of five to seven days. There is still some hesitancy on the part of patients to accept abdominal colostomy, and ofttimes considerable diplomacy and persuasion are necessary to get the patient's approval to permit this radical operation, and the placing of the colostomy which in the Miles procedure serves best in the line of incision and near the midline. The surgeon himself must assume this obligation and either he or some one competent on his staff must teach these patients that the well placed artifical stoma will help them to live normal lives. Never apologize for colostomies but remember this good criterion—"the more we know and teach our patients about well regulated colostomies, the less will be their objections to its presence".

CUTANEOUS ANTHRAX

BEN C. BARNES, M.D.

HUMAN anthrax infections are encountered only rarely today in average medical practice. Prior to the advent of specific therapeutic agents, the mortality of the cutaneous form was 20-25 percent. Recovery from the pulmonary and septicemic types seldom, if ever, occurred. Highly effective measures are available to the modern physician. However, bacteriological confirmation, choice of drug, care of the cutaneous lesion and release of the patient from isolation evoke certain questions in the management of anthrax. The following care of cutaneous anthrax is presented as illustrative of these points.

Report of Case

J.R., white male, age 24 years, was admitted to the Allentown Hospital, January 23, 1951, because of a painful, swollen right upper extremity, chills and fever. Approximately 36 hours prior to admission he had sustained a puncture wound in the right forearm with a wire. He was employed in a local paper factory where his job was feeding raw materials into a chopper. These materials were baled with wire and consisted in part of hides, scraps of fur, old fabrics, and the like. Within 18 hours the right forearm became painful, swollen, and reddened. He was seen by a physician who treated the puncture wound with a dressing of ammoniated mercury ointment and administered 300,000 units of penicillin intramuscularly. The entire right upper extremity became swollen and chills and fever ensued. A black crusting lesion appeared at the site of the puncture wound. He was seen again by the physician, the same dose of penicillin repeated, and was referred to the isolation ward of the hospital with a provisional diagnosis of anthrax.

A general physical examination revealed an acutely ill, flushed, somewhat lethargic young white male. The general nutrition and development were normal. The temperature was 103.4 degrees, pulse 108, respiration 24, and blood pressure 114/74. A black, circular, raised, sharply demarcated lesion was present on the flexor surface of the lower third of the right forearm. It measured 17 x 22 mm. The center of the lesion was umbilicated and the periphery ringed with vesicles containing a serosanguinous fluid. The entire right upper extremity was tensely swollen and diffusely reddened. The superficial lymphatics were infected. The regional epitrochlear and axillary lymph nodes were enlarged and tender. The remainder of the general physical examination revealed no findings of significance.

His blood count revealed the following: hemoglobin 83%; erythrocyte count 4.22 millions; leucocyte count 11,200; and a differential leucocyte count of neutrophiles 76%, lymphocytes 22%, and monocytes 2%. The urinalysis was pH 7.5, specific gravity 1.028, albumin negative, sugar negative and microscopically negative. A blood culture taken shortly after admission was sterile. Blood Kolmer, Kahn, and Mazzini tests were negative. X-ray examination of the chest was essentially negative. Material was obtained from the cutaneous lesion for smear and culture. The smear was negative for organisms morphologically resembling bacillus anthracis.

Aqueous penicillin, 50,000 units, was administered intramuscularly every three hours. Warm moist saline packs were applied on admission. These were discontinued and the lesion was covered with sterile dry gauze held in place with a roller bandage. Tetanus toxoid, 1.0 c.c., was given intramuscularly as he had previously been immunized in the Army. He showed no improvement after twenty-four hours. His temperature remained sustained at 103 degrees, the pulse was 120, and respirations were 24. Penicillin was increased to 100,000 units every three hours and streptomycin, 0.5 gram, intramuscularly, was given every six hours. Twenty-four hours later, his temperature became normal for the first time. He felt generally improved and the swelling of his right upper extremity had decreased considerably. Cultures from the skin lesion revealed large bacilli resembling bacillus anthracis or bacillus subtilis. Bacteriological confirmation of anthrax was secured by injection of the culture into a guinea pig who rapidly succumbed and the organisms were again cultured from the animal.

At the end of the first week of hospitalization, the cellulitis and lymphadenitis of the right upper extremity had improved a great deal. The local lesion consisted of a firmly adherent and round black crust without any appreciable change in size. Streptomycin was discontinued after eight days of administration. Penicillin dosage was gradually decreased and stopped entirely on the 19th hospital day. After five days without specific therapy, cultures were obtained from beneath the eschar and were negative for bacillus anthracis. The patient was discharged on the 28th hospital day with a dry dressing on the still adherent eschar.

Comment

The occupational history and appearance of the malignant pustule lead, as a rule, to prompt recognition of cutaneous anthrax. Bacteriological diagnosis by smear and culture is possible in over 80 % of cases.[1] Bacillus anthracis may be confused with bacillus subtilis and can be distinguished from the latter by its virulence in the guinea pig.

The clinician has a choice of several effective agents against anthrax. The successful use of sulfathiazole, sulfapyridine, and sulfadiazene has been reported. [2] Penicillin has been employed more extensively than any of the other antibiotic agents and until recently was the drug of choice.[3] No clinical improvement was noted with penicillin in the above case after forty-eight hours. Streptomycin combined with penicillin was then given and a prompt response obtained.[4] Shortly after this case was treated Gold and Boger reported a small series of patients with cutaneous anthrax effectively treated with aureomycin, chloromycetin, and terramycin.[5] The oral route was employed in all these cases and the majority were treated at home. If these results can be confirmed satisfactorily, the advantages of such therapy are obvious. Anti-anthrax serum has apparently become obsolete.

Incision and drainage of the cutaneous lesion are unnecessary and dangerous. A sterile dry dressing protects the malignant pustule, is easily disposed of by burning, and is probably more effective in the reducing risk of dissemination of the anthrax organism than are wet dressings.

The majority of anthrax skin lesions are reported as sterile after seventy-two hours' treatment with penicillin.[1] Organisims may still be present after a week's treatment. There was a good deal of uncertainty as to when the patient in this report could be safely released from the isolation ward. At the suggestion of Dr. LaBocetta[6] treatment was discontinued for several days and cultures were obtained from beneath the eschar on two successive days. The cultures were negative for bacillus anthracis and the patient was released from the isolation ward. Such precautions seem reasonable and warranted in view of the organism's marked resistance.

Summary

A case of cutaneous anthrax is reported which responded successfully to combined treatment with penicillin and streptomycin.

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OCULAR MANIFESTATIONS OF THE DERMATOSES

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A considerable number of skin diseases have major ocular complications. In this paper the eye manifestations of Rosacea, Epidemic Herpes Zoster, Herpes Febriles and Contact Dermatitis will be described in detail.

Rosacea is essentially a disease of adult life, starting usually between 20 and 30 years of age and tending to die out in old age. It is a disease of the skin of the face, characterized in its early stages by transient attacks of vasodilitation on the nose and cheeks which ultimately become permanent. The dilitation is most pronounced in the small vessels of the upper horizontal network in the cutis, and is associated with a spasmotic constriction of the capillaries in the papillary body. The state of chronic stasis and congestion leads to a disturbance of secretion and hypertrophy of the sebaceous glands, resulting in the development of erythema with papules which may eventually be converted into pustules. Finally, the continued stasis and low-grade inflammation produce a tendency to hypertrophy, resulting in the formation of telangiectases and the development of rhinophyma. The disease tends to run an extremely chronic course, characterized by irregular exacerbations, some patients showing hardly any trace in the intervals between attacks, but eventually the skin usually takes on a permanently thickened and purplish appearance.

Ocular Rosacea may assume three forms, a blepharo-conjunctivitis which is common and usually of slight import; a keratitis, more rare and frequently involving serious consequences; and more rarely still, an episcleritis. They all tend to become bilateral, although one eye is usually affected first.

(a) **Rosacea Blepharo-Conjunctivitis.** Almost every case of rosacea eventually develops a blepharitis, fortunately usually of a mild nature. It involves a scaly desquamation of the superficial layers of the skin of the lid margins without scarring or distortion of the lashes, and, being very resistant to treatment, results in little more than a chronically red and unsightly appearance of the eyes. Sometimes, but not invariably, the condition spreads to the conjuctiva. Here it may assume two types: more commonly a diffuse hyperaemia, and more rarely a nodular conjuctivitis. The diffuse hyperaemic conjunctivitis of rosacea is characterized by an engorgement of the vessels of the tarsa and ocular conjunctiva, which

is usually especially marked in the exposed inter-palpebral region. The secretion is usually scanty and watery, and there may be considerable irritation and some photophobia. The importance of the condition lies in the fact that it may precede an invasion of the cornea and be the precursor of a keratitis. The condition also predisposes to the development of chalazia.

The nodular conjunctivitis of rosacea is much less common. Without exciting subjective symptoms, small grey nodules appear on the bulbar conjunctiva preferentially near the limbus in the inter-palpebral area. They appear quickly, undergo superficial ulceration, and may disappear as quickly. They are characterized by the extent of their vascularization, being richly supplied by large varicose vessels which give off fine convoluted twigs surrounding the nodule; and on the disappearance of the latter the peculiar type of vascularization persists, allowing a diagnosis to be made with certainty.

(b) **Rosacea Episcleritis.** The appearance of similar nodules in the episcleral tissue is rare. The nodules are of the same type as occur in the conjunctiva near the limbus. They are highly vascular and tend to rapidly appear and disappear.

(c) **Rosacea Keritis.** When rosacea spreads to the cornea it becomes a serious disease. The first appearance of rosacea keratitis is a marginal vascular infiltration, an extension of a rosacea conjunctivitis. The vessels of the perilimbal plexus become dilated and advance into the cornea quite superficially in a zone of grey infiltrating tissue for a distance of about a millimeter. The vascular loops are small and show little branching, and the infiltrated area, which is always sharply delimited, may extend all round the limbus, but is frequently accentuated in the upper quadrants. The next stage is ushered in by the development of sub-epithelial infiltrates. At first usually as small, round or oval, sharply delimited areas near the limbus, and then as larger more ill-defined areas nearer the center of the cornea, and usually in its lower half, these greyish white infiltrates occupy the more superficial layers of the parenchyma, and slowly progress. They are heavily vascularized by large freely anastomosing vessels running into the cornea continuously with the conjunctival vessels. Eventually the epithelium over them trend to become eroded, it becomes uneven and stippled and takes on a fluorescein stain, and eventually breaks down with the formation of an ulcer either at the margin or in the central region of the cornea. As a rule the ulcers are resistant to treatment, their floor becomes

chalky-white, and vascularization of the fascicular type develops. When they do heal, they tend to break down again with a renewal of activity; and with each successive attack the infiltration pushes further towards the center with a resulting grave impairment of vision.

Epidemic Herpes Zoster is part of a definite infection which produces constitutional disturbances and fever, and runs a more or less similar course in all cases from its onset to its subsidence. Although it does occur sporadically, it is usually seen in mild epidemic form particularly in the spring; the epidemics being frequently associated with chicken-pox. It attacks almost entirely adults and the aged. The disease is of sudden onset accompanied by fever and prostration, and neuralgic pain along the first division of the trigeminal which at the beginning may to some extent be masked by general discomfort, nausea and vomiting. Sometimes simultaneously with the pain, but usually 3 to 4 days after its onset, a blushing of the skin over some part or the whole of the area of the distribution of the nerve is followed by a marked edema and the appearance of vesicles. The flushing and redness may be so marked as to suggest erysipelas, and is sufficient to raise the skin temperature on the affected side considerably above the other. The vesicles are first filled with clear fluid, which rapidly becomes turbid and yellow; in a short time they burst, forming scabs which on separating leave deep permanent pitted scars showing that the dermis has been affected by the necrotic process. The whole cycle occupies some 3 to 6 weeks, and throughout this time, the most distressing symptom is usually the severe neuralgic pain.

The distribution of the lesion varies, but the frontal branch of the opthalmic is always involved; sometimes it is the only one. The lacrimal and naso-ciliary branches frequently escape, but when they are affected ocular complications are more common. The terminal branch of the nasocilliary nerve is the external nasal nerve which issues between the nasal bone and the lateral cartilage to supply the skin of the lower part of the nose. Therefore if the herpes involves the side of the tip of the nose, you can be quite sure that there will be severe ocular complications, and these complications are keratitis, scleritis, iridocyclitis, ocular palsies, and optic neuritis.

(a) **Herpetic Keratitis** may assume several forms, but the essential lesion appears to be a sub-epithelial infiltrate composed of minute dots of opacity aggregated into large discrete round spots. One or two, or many of these lesions may occur in the more superficial layers

of the substantia propria, probably at the points where the nerve fibrils pass from the deeper corneal plexus to the more superficial. While such spots in the superficial layers of the substantia propria form the characteristic feature of the keratitis, similar spots may be met with at deeper levels. More particularly in the early stages of the disease; also, irregularly-shaped opacities may occur, together with edematous striae and general haziness, with, in the more severe cases, the formation of folds in Descemet's membrane. These spots may form the only lesion, but it is the rule for epithelial vesiculation to appear over them; exfoliation and secondary infection may occur, leading to the development of ulcers which are slow to heal and troublesome to treat, or alternatively a sluggish and recalcitrant keratitis profunda may develop.

The punctate infiltrate with acute symptoms may settle down in 2 to 3 weeks, or may drag on unaffected by treatment for months. Some opacities always remain permanently, and in the more severe cases new vessels may invade the cornea. Sensation recovers slowly, usually after several months, sometimes after years, sometimes not at all, the recovery depending presumably on the damage done to the cells of the Gasserian gangliao. In most cases the recovery of sensation is permanently imperfect, and in the very worst cases a complete picture of neuroparalytic keratitis may develop with the usual disastrous consequences.

(b) **Scleritis** is a relatively rare complication and usually occurs late, in association with an iridocyclitis and frequently with a paralytic mydriasis. Round nodules about the size of a lentil, painful and irritable, appear usually about 2 to 3 months after the cutaneous eruption has gone; the conjunctiva over them is glossy, smooth and hyperaemic, and after remaining active for several months, they fade away gradually to leave a permanent, pigmented, slate-colored scar.

(c) **Iridocyclitis.** With the exception of those cases complicared by a paralytic mydriasis, every case of herpes opthalmicus is characterized by a small pupil, partially the result of an irritative reflex miosis and partially of a congestive hyperaemia. When keratitis supervenes iridocyclitis is practically invariable, coming on usually about a week after the skin eruption, and it may also appear when no keratitis has existed. The deposition of keratic precipitates is common, and a profusely exudative plastic inflammation may result.

(d) **Ocular Palsies** are not uncommon affecting the IIIrd, IVth and VI nerves. In the eruptive stage of the disease a Ptosis is constant, largely determined mechanically by swelling of the upper lid, but in many cases a paretic ptosis exists, sometimes accompanied by a mydriasis and a paresis of accommodation or a paralysis of the internal rectus. Aparesis of the IVth and VIth nerves is rare, and of the VII rarer still. The prognosis is good, for although some of these palsies may persist for long, they usually disappear in less than 6 weeks.

(e) **Optic Neuritis** is fortunately a rare complication, but when it does occur it may be followed by atrophy to the extent of abolition of perception of light.

Herpes Simplex may occur as a local corneal reaction to the herpes virus without other systemic affection, sometimes accompanied by herpetic lesions elsewhere, or it may occur in association with some other febrile systemic disease. It is almost certainly the case that a considerable proportion of the population are herpes carriers and the effect of the accompanying systemic infection is to allow the virus to establish a foot-hold in tissues, the resistance of which has been lowered. Such an associated infection precedes about 60% of the cases of Herpes. Herpes febrilis is a common disease consitituting one of the most frequent affections of the cornea. It occurs at all ages, and very frequently in children. It is usually unilateral, but bilateral cases have been recorded, although rarely; but once an eye has been affected there is a definite tendency to recurrences. Both sexes are attacked about equally in childhood, but in adult life males predominate. The clinical picture varies enormously, but usually opens acutely with a sharply painful eye associated with the appearance of a haze of fine granular white spots in the epithelium, so fine that they may not be visible to the naked eye except when agglutinated into clumps. This is associated with a fine epithelial bedewing and frequently with the development of cracks and fissures in the epithelium, round which the punctate opacities are frequently closely packed. The granular changes or the fissures may predominate, both the spots and the fissures stain with fluorescein. A more rare appearance is the arrangement of the minute epithelial dots into lines, frequently with a complicated criss-cross pattern. It is typical in all cases that for a considerable area around the visibly affected region the epithelium lies loosely on Bowman's membrane can be readily sucked up or detached.

The appearance of the fine epithelial opacities may be followed by the classical picture of tiny clear vesicles in the epithelium coming up

in a crop, arranged in bunches or rows associated presumably with the terminations of nerve filaments, while underneath them is an area of fine haze in the superficial stroma. The frail vesicles are rarely seen, for they soon break leaving behind dequamated spots, the bases of which are delicately clouded. Their appearance is associated with considerable pain owing to the exposure of the inter-opithelial nerve fibriallae. In complicated cases, after persisting from 2 to 3 days to 2 to 3 weeks, these clear up leaving no trace, although there is a definite tendency for recurrect crops to appear either when the original lesion is still active or after it has healed, until the epithelium has again become consolidated and the condition of epitheliolysis has disappeared. Alternatively, however, they may spread and coalesce to form an irregular ulcer. On the other hand the fissures extend in breadth and depth and their edges become infiltrated to form bizarre-shaped dendritic ulcers; while at other times, without the appearance of vesicles, small grey ulcers develop.

The dendritic ulcer presents a very typical appearance. Formed as it is of a confluence of minute herpetic efflorescenses, it is of an irregular zig-zag linear shape with numerous side-branchings, forming a complicated arborescent figure with beadlike nodes at the ends of the branchings. The ulcerated area is barely 1 mm. broad and show fine furrows and is surrounded by a swollen overhanging edge and a hazy area of infiltration. The ulcer stains with fluorescein, but the stain diffuses rapidly under the adjacent epithelium demonstrating its loose attachment and its participation in the infective process. Such a picture, accompanied by photophobia lacrimation and pain, may persist for a long time—sometimes for several months in the absence of adequate treatment, breaking down and healing in an irregular and sluggish manner, so that now one part and now another of the linear figure stains, the expanded nodes persisting the longest. Many cases run on in a quiet unobtrusive way, causing little discomfort for months; but eventually the furrows become epithelialized leaving the nodes as small grey flecks, until these, too, finally disappear, usually without forming real scars: vascularization does not occur.

In addition to the usual superficial spread in either linear or aroborescent form, even in cases uncomplicated by secondary infection, the process may spread into the substance of the cornea producing a parenchymatous keratitis of varying severity. This may be diffuse, the stroma becoming hazy and intersected with rifts, or, more frequently, a circumscribed Disciform Keratitis may develop.

An herpitic iritis is a frequent accompaniment, sometimes of a hemorrhagic type, and occasionally showing dilated and tortuous

vessels and multiple extravasations of recurrences may be without any corneal involvement.

Contact Dermatitis of the skin of the lids is characterized in the acute stage by redness and edema which is followed by vesicle formation and later by oozing and crusting. If the sensitizing agent is not removed, the acute stage passes into a chronic stage which is characterized by continued redness, and the skin becomes thickened, scaly and covered with excoriations.

The eye findings in a contact dermatitis consist chiefly of marked congestion and edema of the conjunctiva. The congestion is more marked in the palpebral conjunctiva while the edema is more marked in the area of the fornicies and the caruncle. The cornea is rarely involved. The chief complaint is that of intense itching.

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