医学专业英语

（供医学 预防 检验 口腔 麻醉 精卫 护理 专业用）

湖南医科大学教务处

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How to write a case report

Yang ××, 30-year-old, married, male, teacher, was admitted to our hospital on November 1, 1990, because of pain in the right lower abdomen for two days.

On the morning, two days prior to his admission, the patient started having pain in the upper abdomen. The pain gradually increased in severity and was accompained with nausea and vomiting, the vomitus being undigested food and acid fluid, He had a poor appetite. About six hours later, the pain shifted and was confined to the right lower abdomen. The patient described it as a prickly sensation and said it was intolerable. The patient went to a nenrby hospital for treatment, He took some medicines but without any effect. The pain became more intense early this morning. so he came to our Hospital for emergency treatment. Since the illness, he has had a slight fever, which ranges from 38℃ to 38.5℃ and has had normal urine. He has been constipated for three days.

Past history: In January, 1989, he had similar abdominal pain for three days and the pain subsided after antibiotic treatment. No history of TB, hepatitis or other major infections diseases.

PE:

T: 38℃ P: 90/min R: 22/min BP: 120/80mmHg.

Well developed and nourished. Mentally clear but acute distress. Head and sense organs normal. Sclerae not jaundiced. Neck soft and freely moveable fromside to side. Thrachea in midline, Thyroid not enlarged. Chest normal on inpection and symmetrical with equal movement during respiration. Breathing sonds over both lungs clear, Heart beat regular, with a rate of 90/min, and no murmurs heard. Abdomen examination presented in the "Surgical Condition" below. No deformity or disturbance of function, in spine and extremities. No pathological reflexes detected. Anus and acternal genitalia are normal.

Surgical Condition.

Abdomen flat, Liver and spleen are not palpable below the costal margin. Marked tenderness present in right lower quadrant, especially over Mcburney's point, with muscle guarding and rebound tenderness but no mass can be felt, Left lower abdomen soft and free from tenderness. Rovsing's sign postive. No shifting dullness heard on percusion. Bowel sounds audible without high pitch.

Laboratory findings.

W.b.c. 10,800 N: 88% M: 10% E: 5%

Urine: Normal

Diagnosis:

Acute Appendicitis

Signature. Wen ××

Syndrome of hypertenalon and hypokalemia

(Lacture outilne)

Hypertension associated with hypokalemia is a rather common syndrome. In my lecture, I shall emphasize to talk about the differential diagnosis and the mechanism of inducing hypertension and hypokalemia in each disease.

一、Clinical manifestation

There are many diseases that can induce this syndrome, no matter in the causes this syndrome must include two-groups symptoms and signs of hyperte—nsion and hypokalemia.

(1). Hypertensive symptoms and signs: Headache. dizziness. palpitation. blurred vision, weakness, and insomia. In long-duration patients, hypertensive complications may occur, such as angina-pectoris, hypertensive heart disease, hypertensive encephopathy, apoplexy and giomeruler angiosclerosis.

(2). Hypokalemic symptoms and signs: palyuris especially at night, polydepsia Paralysis of extremisies. even respiratory mascles to indure dyspnea, extrasystoles, hypokalemic nephropathy with mild proteinuria depressed ST segment and abnormal U wave in EKG.

二、Disease associated with this syndrome

(1). Essential hypertension: If hypertensive patients have treated with antihypertensive drugs containing DHCT for a long time, without potassia complement, this syndrome may developed, After thses durgs withdram Hypokalemia disappears rapidly only keeping the blood pressure elevated.

(2). Malignant hyypertension: Because the acclerated course glomendor vessels is invasiably involved to induce renal ischemia—> increased reain release——> AtII ↑ ——>aldosterone ↑ ——>increased potassium loss in urine ——>hypokalemia.

Except hypertension and hypokalemia, blood pressure is usually very higher proteinuria, retinopathy may rapidly develop, and Rbc or granular cast may be found in urinary microscopic examination. Blood pressure persists elevated without response to common antihypertensive drugs.

(3). salt-loss nephritis: Hypertension is very common sign. When renal tubales have been involved severely hypokalemia may occur because of the increased Na-K exchange in distal convoluted tubule. Proteinuria Rbc Wbc and various casts may be found in urine. Hypokalemia is secondary to increased aldosterone secsetion.

(4). Renal tubular acidosis: In late stage of renal tubular acidosis hypertension may ensue. Hypokalemia is due to increased potassium loss from urine because of the ipairment of renal tubular function. There are four types. Only type I is the most common one associated with hypertensive and hypokalemia syndrome. In early stage blood pressure is not elevated, but polyuria, polydyposia muscutar weakness and paralysis. Some patients may develope to osteoporosis or multiple pathogogical fractures. Ammonia chloride

loading test shows positive result. In type II glucosuria amine-acidouria can usually be found.

(5). Stenosis of renal artery: Thrombosis and compression by extrarenal lessions in one-side renal artery can induce this syndrome.

Stenosis of renal artery ——>blood flow decreased ——>renin release ↑
>AtII ↑ ——>aidostorone ↑
↓ ↓
hypertension hypokalemia

If the affected renal artery is not occlusion completely, bruit may be heard over abdomen surounding the umbilicas or along with the left side of spine. This sign is very usefui for diagnosis. renal angiography may be used to comfirm the disease.

(6). Reninoma: This is a rare disease, Up to now about twenty cases have been reported, over the world. Renin is normally synthesized by jaxtaglomerular apparatus in kidney. It is easy to understand How this disease can induce the syndrome of hypertension and hypokalemia.

Wilms tumor and extrarenal tumor such as some orary tumor can also induce the similar manifestations just like reninoma, but enlarged kidney may be found renal angiography.

The clinical features of reninoma include.

(1). > 40 years old

(2). Plasma renin activity significantly increased

(3). Blood presure can be controlled by diazoxide but not by regitine

(4). Serum NE.E.Urinary VMA, metacpinephrine metanorepinephine are normal

(5). Can be cured by surgery

(6). Renal arteriography may be helpful for diagnosis

(7). Hyperaldosteroniom: Hyperaldostioniom,irrispeitive of causes, is usually present with both of hypertension and hypokalemia. It is easily explained that the hyperaldosterone can induce hypokalemia. The pathogenesis of hypertension is related to sodium retention which can enhance the sensitivity of vessels to endogenous catcholamine,and the expansion of blood volume. In these case renin-angiotensin sytem is suppressed. serum and urinary aidosterone is significantly increased.

(8). Cushings syndrome: This syndrome is due to excass of hydrocortisone. Glucocorticoid have some action of mineralocorticoios, but its potency only equal to about 1/1000 aldostorone some pationts may manifestate hypertension and hypokalemia which is less severe than hyperaledosteronism. The clinical features are very impressive including moon-fave, buffalo nock, skin purple stria,centric obesity serum cortisol,urinary 17 hydroxy corticosteroid and 17 ketocortic—osteroid are increased, large-dose dexamathasone suppressive test can differentiate the hyperplasia from adenoma. Adrenocortical

carcinoma usually have both excess of glucocorticoled and minerdoc—orticoid, sometime a drencortical androgen steroid may also be in excess, Thesefore virilism may occur except the symptoms and signs of hypercortidolism and hyperaldostesonism. These patients often appear cachaxia becuse of the acclerated course.

(9). Congenital adrenocortical hyperplesia: These syndrome is caused by the deficiency of adrenscortical enzymes related to the biosnythesis of hydrocortisone, Only 11 and 17B hydroxylase deficency can induce hypertension and hypokatemia. The pathogenesis of hypertension and hypokalemia in these diseases is as follows.

11 or 17B hydnxylase deficiency

↓

Hydrocortisone ↓

↓

feedback to pituitary weakened

↓

ACTH ↑

stimulating adrenocortex to biosynthsige aldosterone and androstenedione and dehydroepiandrolone

Aldosterone ↑ — adrenocortical androgen Hormone ↑

↓ — ↓

hypertension and hypokalemia — pseudosexality premature in boy virilism in girl

If a patient with hypentension and hypokalemia is associated with abnormal external genitalia and Secondary sexal sign. Congenital adrenocertical hyperplasia should be considered at first.

(10). Ectopic ACTH syndrome: Some malignant tumor such as Oat cell pulmonary carcinoma. Panceatic carcinoma. Thyroidal medulary carcinoma and so on. can synthisize and release ACTH or ACTH-like substance to induce cushing's syndrome. with hypertension and hypokalemia. In these cases, clinical features of cushing's syndrome is not usually impressive because of its rapidly progressive course.

(11). Liddles's syndrome: The cause of liddle's syndrome have been attributed to intrinsic renal defect, It is characteristized by bypertension and hypokalemia alkalosis with negligibly increased aldosterone secretion Clinical manifestations presumably result from increased sodium reabsorption and potassium excretion in distal tubules independent of the effect of any known mineralocorticoids. Administraton of triamterone. which interfers with distal tubular ion transport by means of an action that does not involve mineralo—corticoid inhibition, resulting in natriuresis and decreased

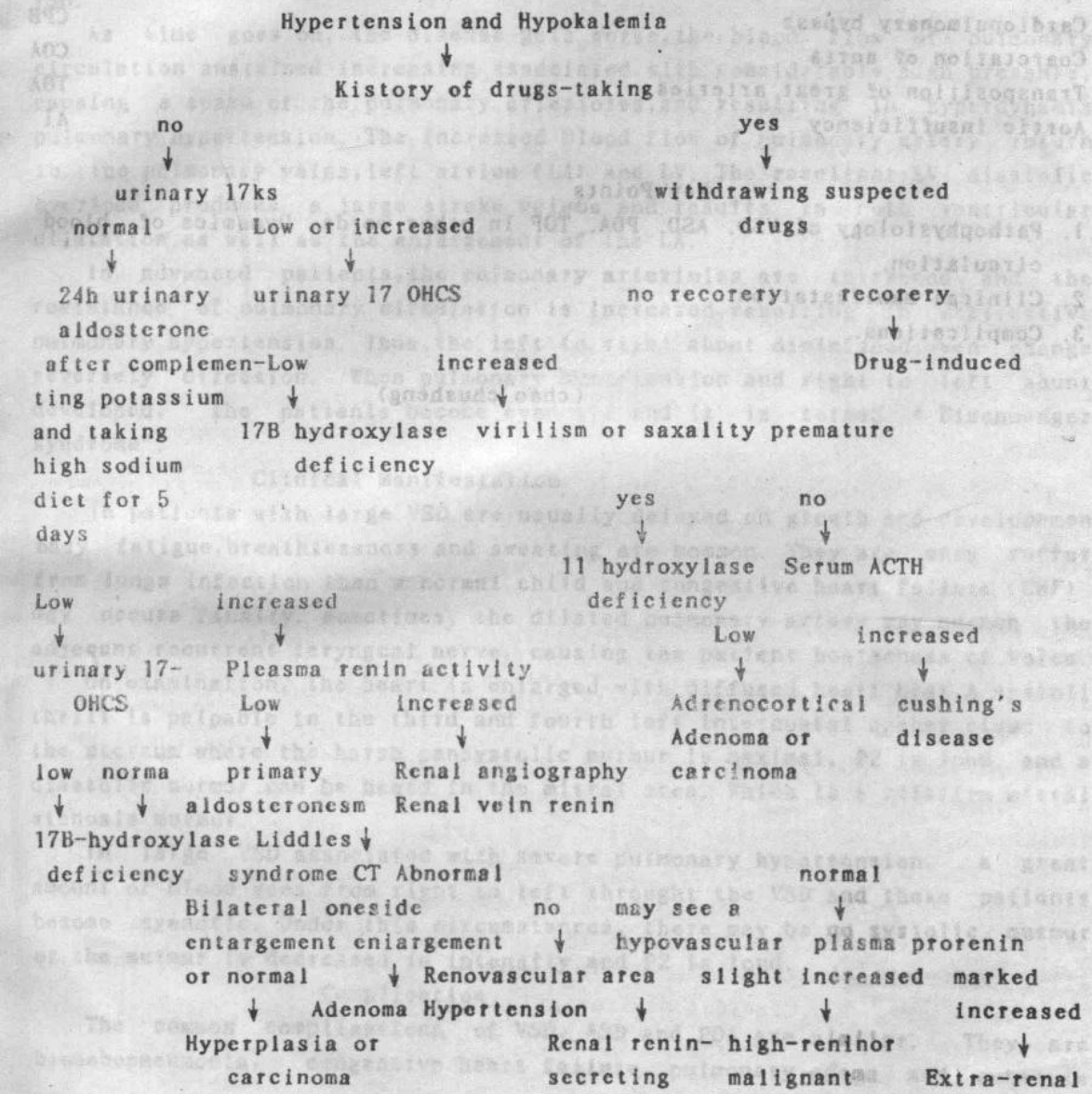
potassium excretion accornpanying with the correction of blood presure and hypokalemia while spirelactone have no efficiengy because it action is by mean of antagonism to aldostcrone.

02. Bruge-induced: Some drugs such as glycyrrhige and biogasterone, if long-term administration,of these druge can induce hypertension and hypokalemia. Both drugs have the mineralocorticold-like action. withdrawn these drugs,hypertension and hypokalemia can be corrected.

三、Appoarch to the diagnosis of this syndrome

The following diagram shows the diagnostic processes:

The diagram for work up of hypertension and hypokalemia



tumor hypertension renin-secreting tumor

Congenital Heart Diseases	CHS
Ventricular Sptal Defect	VSD
Atrial Sptal Defect	ASD
Patent Ductus Arteriosus	PDA
Tetralogy of Follot	TOF
Electrocardiogram	ECG
Ultrasound Cardiogram	UCG
Open Heart Surgery	OHS
Cardiopulmonary bypass	CPB
Coarctation of aorta	COA
Transposition of great arteries	TGA
Aortic insufficiency	AI

Key Points

1. Pathophysiology of VSD. ASD. PDA. TOF In other words: Dynamics of blood circulation.
2. Clinical manifestation.
3. Complications.

(chao chusheng)

Classification of CHD

Three groups of CHD are based on whether there is blood shunt between left and right heart or great vessels or not.

1. Left-to -Right shunt (potential cyanosis groups).
2. Right-to-Left shunt (cyanosis groups).
3. No shunt (no cyanosis groups).

VSD

Pathophysiology

Since the pressure of (LV) is ususally higher than the right ventricle (RV), there is always a left to right shunt from the LV to the RV across the VSD.

As time goes on, the disease gets worse, the blood flow of pulmonary circulation sustained increasing associated with considerable high pressure, causing a spasm of the puimonary arterioles, and resulting in hyperdynamia pulmonary hypertension. The increased blood flow of pulmonary artery return to the pulmonary veins, left atrium (LA) and LV, The resultant LV diastolic overload produces a large stroke volume and results in both ventricular dilatation as well as the enlargement of the LA.

In advanced patients, the pulmonary arterioles are thickened and the resistance of pulmonary circulation is increased, resulting in obstructive pulmonary hypertension. Thus, the left to right shunt diminfshed, even change reversely direction. When pulmonary hypertension and right to left shunt developed, the patients become cyanotic and it is termed " Eisenmenger syndrome".

Clinical Manifestation

In patients with large VSD are usually delayed on growth and developement Easy fatigue, breathtessness and sweating are common. They are easy suffer from lungs infection than a normal child and congestive heart failure (CHF) may occurs finally. sometimes, the dilated pulmonary artery may quench the adjacent recurrent laryngeal nerve, causing the patient hoarseness of voice.

On examinaiton, the heart is enlarged with diffused heart beat, A systolic thrill is palpable in the third and fourth left intercostal spaces close to the sternum where the harsh pansystolic murmur is maximal, P2 is loud, and a diastolic murmur can be heard in the mitral area. Which is a relative mitral stenosis murmur.

In large VSD associated with severe pulmonary hypertension, a great amount of blood goes from right to left throught the VSD and these patients become cyanotic. Under this circumstances, there may be no systolic murmur or the murmur is decreased in intensity and P2 is loud.

Complication

The common complications of VSD, ASD and PDA are similar. They are bronchopneumonia, congestive heart failure, pulmonary edema and subacute

bacterial endocarditis.

ASD

Ostium secundum defect

Pathophysiology

After birth, the pressure of right atrium may be higher than that of the left atrium, venous blood can be shunted across the ASD into the left atrium and results in temporary cyanosis. Once the pressure of left atrium rises above the right atrium, a left to right shunt occurs(cyanosis disappears), Persistant large amount of blood flow to the right side of the heart results in enlargement of the right atrium and ventricle,and dilatation of the pulmonary artery. The LV and aorta are smaller than usual. As time goes on, the "Eisenmenger Syndrome" may occur finally.

Clinical Manifestation

Symptoms: they are similar to those of VSD.

Signs: patients with large ASD usually are small and thin, A grade Ⅱ -Ⅲ /Ⅳ systolic ejection murmur is present at the second and third intercostal space close to the left border of the sternum. If is produced by the large amount of blood flow in the pulmonary artery which results in a relative pulmonary stenosis. Usually the murmur is soft and seldom accompanied by a thrill. In most patients, P2 is widely split and fixed in all phases of respiration. When there is a great anount of blood from left to right shunt, a middiastolic murmur may be audible at the lower left sternum border.

PDA

Pathophysiology

1. Since the aortic pressure is highter than the pulmonary artery pressure, there is a continuous run-off of blood flow from the aorta to the pulmonary artery through the patient ductus arteriousus.

2. In PDA, the pulmonary artery receives the excessive blood flow from both RV and aorta, thus the pulmonary arterial blood flow is inceased, as is the return of pulmonary venous blood to the LA and LV, the resultant left ventricular diastolic overload results in LV hypertrophy and dilatation and the LA also enarged.

3. The diastolic pressure of aorta is lower than normal due to a part of aortic blood flow run-off into the pulmonary artery, thus the pulse pressure widened and a pistol shot may be heard when picking up the blood pressure.

4. When a bidirection shunt or a right to left shunt occurs, venous blood from the pulmonary artery is shunted into the descending aorta a blue lower extremities and pink upper extremities occurs, and this phenomenan is called "differential cyanosis", namely cyanosis in the lower half of the body.

Clinical Manifestation

Symptoms: they are similar to those of VSD and ASD.

Signs: A loud continuous machinery, rolling thumder murmur is heard

maximal at the second intercostal space close to the left border of the sternum. In a small infant or if PDA is accompanied with severe pulmonary hypertension or congestive heart failure, it is quite possible while only a minimal systolic murmur will be present. At the site of maximal murmur, a thrill is palpable. Abloud P2 may be heard at the area of pulmonary valve. In patients with a large left to right shunt, a low pitched mitral murmur may be audible. A wide pulse pressure, " differentilal cyanisis" and clubbing of toes are usually present.

TOF

Tetralogy of fallot consists of four structural abnormalities. These are

1. Pulmonary stenosis
2. Ventricular septal defect
3. Dextroposition of the aorta
4. Right ventricular hypertrophy

Pathophysiology

Pulmonary stenosis is the key defect of the tetralogy abnormalities. The stenosis is most frequently occurred in the infundibula of the RV, obstruction to RV outflow results in elevation of the RV pressure and the hypertrophy of RV. Moreover, the aorta straddles over the VSD. A large volume of RV blood flow shunts into the aorta and leads to cyanosis, clubbing of fingers and toes and compensatory poiycythemia.

Clinical Manifestation

1. Cyanosis, once the ductus arteriosus closed, it becomes conspicuous.
2. Clubbing of fingers and toes.
3. Patients eager to take a squatting position in order to decrease the load of heart.
4. Hypoxic spells may suddenly occur, the attacks are thought to be due to contractility of the infundibular musculature.

Growth and developement are usually delayed. A systolic murmur of ejection type is usually heard in the second to the fourth left intercostal spaces close to the sternal border, maximal at the third. The murmur is caused by pulmonary stenosis and it may disappear when the infundibular contracts. P2 is disminished.

X-ray

Normal heart size. The outline of heart looks like a wooden slipper. Because of the diminished pulmonary arterial blood flow, the hilar aresas of the lungs fields are remarkably clear.

Complications

The complications are:

1. Cerebral thrombosis is most common in the presence of extreme polycythemia and may be precipitated by dehydration.
2. Cerebral abscess.
3. Bacterial endocarditis.

(Yu Xiaoliang)

GONORRHEA

Gonorrhea is the most common venereal disease and is particularly prevalent among, though not confined to young patients. Because it is invariably transmitted by sexual contact in the adult, its occurrence is far more likely under conditions of sexual promiscuity. There is little doubt that a major resurgence of gonorrhea is taking place at the present time. In 1969, 494,227 cases of infectious gonorrhea were reported to public health authorities in the United States. In 1974, it was estimated that more than 1 million new cases are developing annually in this country, and 60 million new cases annunally in the world. And these represent only a fraction of the cases, since not all are diagnosed and not all are reported.

ETIOLOGY AND DIAGNOSIS

Primary infection in the adult female occurs in the lower genital tract in the course of sexual contact during which infected material from the penis is deposited in the vagina. Under these circumstances, infection, or reinfection of a previously treated patient, occurs with a frequency which suggests that little or no natural or acquired immunity exists in this disease. The offending organism, Neisseria gonorrhoese, is a biscuit-shaped, gram-negative diplococcus, which is demonstrable in phagocytes within a few days of exosure.

Because of the large number and kinds of bacteria that occar naturally in the female reproductive tract, diagnosis on the basis of gram staining is now considered unreliable in women. The most accurate and generally available means of identifying the gonorrheal organisms is by use of the Thayer-Martin culture medium. This is chocolate agar that contains inhibitory antibiotics (vancomycin hydrochloride, colistimethate sodium, and nystatin) which allow growth of pathogenic Neisseria while almost completely suppressing growth of contaminants. Cultures should be taken, in order of importance, from the vervix, from the anal crypts from the urethra, and from the vagina. Fluorescent antibody techniques, while heghly accurate, are more cumbersome and can be used when needed for confirmation.

ACUTE GONORRHEAL INFECTION

The area most susceptible to infection appears to be the cervix, not only because of its proximity to the location of seminal discharge but because the endocervical glands afford ideat conditions for flourishing growth of the gonorrheat organism, within 3-5 days of initial infection, the patient suffers malaise, low-grade fever, and a profuse purulent discharge emanating from the cervical canal. The labia become swollen and edematous and a somilar purulent discharge may be seen in the urethral meatus. Urinary frequency and dysuria are common symptoms, and acute cystitis occurs.

It is noteworthy that of all the tissues of the lower genital tract, the adult vagina seems to be most resistant to gonorrheal as well as other forms

of bacterial infection, This is likely related to the thick stratified squamous epithelium which forms its lining. Thus, gonorrheal vaginitis occurring in children as a result of contact with contaminated sources can be effectively treated with estrogens, which bring about rapid thickening and cornification of this epithelial protecitve layer, Further, gonotrhea is seldom seen in the vagina of a woman previously subjected to surgical removal of cervix and uterus.

SPREAD

The most serious consequences of gonorrhea are related to its spread to othor genital organs and the development of subacute or chronic infections which impair fertility and lead to irreversible tissue damage and intractable symptoms.

In the lower genital tract ,this is evident within a few days in the development of acute inflammation of the Bartholin glands, which become indurated and exquisitely tender. This condition progresses rapidly to abscess formation, probably because the single duct draining these glandular structures is easily occluded by edema and local tissue swelling. While abscesses of th Bartholin gland can and do result from infections other than gonorrhea, their occurrence merits investigation for gonorrheal infection by smear and culture of cervical exudate, Bartholin abscesses require, in addition to antibiotics, incision and drainage at the proper time or, if well localized excision or marsupialization.

In the cervix, acute gonorrheal inflammation may subside over a peroid of days or weeks following inadequate or ineffective treatment, so that profuse cervical discharge and erythema are no longer present. Nevertheless, the cervical glands may harbor the organism for a long time and serve as a source of exacerbation of infection. Similarly, Skene's ducts in the urethra may become the seat of chronic infection leading to abscess formation requiring incisio and drainage.

The next step in spread of the disease is the most serious: progression to involve the internal genitalia, Gonorrhen spreads upward, in contrast to other forms of pelvic infection, which may start in the uterus or falopian tubes and spread downward to the lower tract. This spread may, infrequently, occur directly following the primary infection, so that the patinet shows evidence of salpingitis and pelvic peritonitis immediately after the first symptoms of cervical infection. However,direct and indirect evidence indicates that upward spread usually occurs at the time of a menstrual period. Abdominal signs and symtoms of serious illness usually begin toward the end of or following a menstrual period. Pathologic studies of the endometrium at this time show rapidly spreading superficial endometritis. This is reflected in some patients by profuse menstrual bleeding, but more often fever, abdominal pain, and malaise begin following menses. These cbinical and patologic observations suggest that upward spread of gonorrhea is favored by menstrual

discharge, but the occurrence of gonorrheat infections in the body of the uterus is rare, and myometrial involovement is seldom seen, even in the presence of advanced adnexal disease.

Gonorrheal infection may not spread to the fallopian tubes in the first menstrual period following primary infection, but may occur there several months after the initial episode. Whether this results from the presence of residual orgnisms in the cervix in untreated or inadequately treated casses or from reinfection is a matter for speculation. Once salpingitis occurs, however, serious consequences ensue.

ACUTE GONORRHEAL SALPINGITIS

Gonorrheal infection of the fallopian tube produces, as its initial consequence, a profuse exudate of infected material. The volume of this exudate is large in relation of the size of the tube because of the large mucosal surface presented by the arborescent mucosal architecture of this organ. It is clear from pathologic studies that this exudate is fibrinous and rapidly produces both agglutination of mucosal folds and occlusion of the tube some where along its length. Initially, however, the spill of this exudate into the pelvic peritoneal cavity results in acute pelvic peritonitis.

The picture thus produced is classic: severe lower abdominal pain, bilateral in character, with tenderness, spasm and guarding over the lower quadrants; leukocytosis, and fever. In some cases, the pain may be gastrointestinal symptoms are far less pronounced. On pelvic examination, no masses are evident but motion of the uterus produces severe pain, and palpation of thednexa is even more excruciating.

Prompt antibiotic treatment of acute gonorrheal salpingitis is usually rewarded by return of temperature to normal, rapid resolution of abdominal signs, and disappearance of malaise. This may be carried out with aqueous penicillin G or ampicillin adm-inistered intravenously. However, the damage may have already been done if closure of the tubes has occurred. The young patient with this disease may not attempt pregnancy until years later, and ivestigation of her failure to conceive will then reveal the ectent of tubal damage or occulsion. Recently, attempts have been made to retain tubal patency in patients with acute gonorrheal salpingitis by administration of prednisolone together with penicillin. The result, in 200 patients, was tuba lpatency in about 80%. The preservation of tubal patency, however, does not necessrily mean that the tubalmucosa, so necessary fer successful fertiliz—ation and ovum traqnsport, was normal.

Clinical observations indicate that occasionally gonorrheal sapingitis is unilateral or at least one tube is damaged more than the other, so that pregnancy is still possible. This clinical impression is reinforced by the fact that a unilateral hydrosalpinx and adnexal abscess are sometimes discovered in a patient with an intrauterine pregnancy. However, the vast majority of develop acute gonorrheal salpingitis have bilateral involvement