Section of Obstetrics (Head obstetrician: B. SZENDI) and Section of Pathology (Head pathologist: V. HEIM) County Hospital, Gyula, Hungary

GENITAL TUBERCULOSIS IN THE FEMALE: THE MECHANISM OF ITS GENERALIZATION

B. SZENDI and V. HEIM

(Received April 29, 1959)

The rare occurrence of female genital tuberculosis in association with pregnancy, the interaction of the two processes and their final issue have always claimed great attention. H. KRÄUBIG [15], KREIBICH [16], PATAT [17] and other authors have recorded several cases of genital tuberculosis either cured by tuberculostatic drugs or reduced to a state permitting conception. According to JAMESON, there has been an increase in the incidence of tubal pregnancy since the introduction of tuberculostatic drugs. KIRCHOFF [12] reports 49 out of 130 patients (almost 40 per cent) to have become pregnant after recovery from the bacteriologically proved disease. The good results and positive pregnancies attained with the therapeutic methods employed by SCHÄFER [20], KIRCHOFF [12], KREIBICH [16], etc., encouraged JEDBERG [9], SZENDI [25] and others to abstain from the removal of the tuberculous oviducts in women anxious to conceive, provided these were healed or the oviduct was for them to be made passable by surgical intervention. Opinions to the contrary maintain that the coincidence of genital tuberculosis and pregnancy is rare but all the more dangerous (KOVÁCS, 14; SCHÄFER, 20; SZENDI and LAKATOS, 24; PATAT, 18; FEKETE et al., 5; etc.).

During the past 7 years the present writers have been treating successfully 90 out of 160 cases of genital tuberculosis conservatively with tuberculostatics, in 10 instances not removing the tuberculous oviduct but making it passable by surgical intervention. None of the patients conceived. Reviewing the literature, STÜPER [22] concluded that pregnancy should be avoided while the tuberculous affection of the genitals is lasting and for one year after it. The question, however, arises whether or not in the present state of our knowledge it is possible dependably to establish recovery from genital tuberculosis. Departing from DIEHL's experimental results, KOVÁCS [14] believes genital tuberculosis to be a matter of organic disposition independently of whether the patient has merely passed through the disease or recovered completely. A subsequent exogenous infection or endogenous dissemination is liable to bring about a relapse. Nor is there any evidence for the new therapy to render gestation less dangerous once the genital tuberculosis

has healed. The present report is to call attention to the incalculable risks involved in pregnancy of the tuberculous genitals, mainly due to the difficulty of discovering the disease. As pregnancy seldom goes hand in hand with genital tuberculosis, there is no reason to suspect the latter, not even in the presence of adnexitis, sepsis, etc. The case under review merits attention not only as one of unilateral chronic tuberculosis combined with pregnancy or as one of lethal generalization consequent upon its activation due to interruption of pregnancy or upon insufficient therapy but also as one throwing light upon the mechanism of the process of generalization.

Case Report 171-I/1958

A woman aged 32 years with two previous deliveries and two abortions. Her last pregnancy two years ago had terminated in spontaneous abortion without complications. Tuberculosis in the left Fallopian tube must be assumed to have developed subsequent to her pregnancies. On January 8, 1958, she had been curetted in another hospital for a 2-month pregnancy. Three days later she had developed a high temperature and slight jaundice with enlargement of the liver. After 2 weeks of unsuccessful medical treatment she was referred to our department with a diagnosis of postabortive sepsis. The record at discharge mentioned postabortive febrile state, cholangitis, jaundice, slight anaemia and brucellosis. Treatment included penicillin, streptomycin, chloramphenicol, blood transfusion, etc. At admission no resistance was felt in the hollow abdomen. The liver reached two fingersbreadth below costal arch. Chest X-ray was negative. Gynaecological finding: moderate perineal rupture, erosion, uterus of normal size, somewhat enlarged and sensitive adnexa on both sides. Temperature 37.8 to 38.6 C°, pulse 90—100/min; RBC 3.800.000, WBC 6.800; differential count neutrophils: young 3, unsegmented 8, segmented 59, eosinophils 2, monocytes 4, lymphocytes 20. ESR 25/h. Serum bilirubin, indirect, 0.18 mg per 100 ml. Thymol turbidity 9 units. The non-typical clinical symptoms following interrupted pregnancy were suggestive of puerperal fever. After blood transfusion, streptomycin and aureomycin treatment were prescribed and in view of the jaundice and the pathological liver function test, the patient was transferred to the Internal Department with a tentative diagnosis of epidemic hepatitis. Here she was treated for six weeks with penicillin, streptomycin, chloramphenicol, erithromycin, cortisone, delta-cortisone. Repeated attempts to recover the pathogenic agent from the blood were unsuccessful except on two occasions when *Staphylococcus albus* and *Streptococcus viridans* were identified. Six weeks later the patient was transferred to the Surgical Department with the diagnosis of hepatic abscess.

At operation no liver abscess was found, but a thick inflamed left tuba with pus in its lumen. Behind the tuba and extending behind the ovary there was a chronic abscess. Tuba, ovary and abscess wall were removed.

Their histology deserves to be discussed in some detail in order to gain some insight into the mechanism of relapse and miliarization of chronic tuberculous processes in the course of pregnancy. The findings were as follows: the wider lumen of the thin-walled oviduct is replete with a homogeneous eosinophilic substance. In place of the mucous membrane there is a thin tissue rich in cells, continuing in fibres in the homogenous substance, with heterocellular tubercles amidst the thinner or wider canals which are lined with cuboidal epithelium (Fig. 1). Among the muscle fibres there are lymphocytic infiltrations with some granulocytes and plasma cells. This tuberculous lesion must have been of long standing. The structure of the ovary is mostly normal, the surface showing a few, the hilum numerous single and confluent tubercles with caseated centres. Mesosalpinx and mesovarium are rich in cells, with many tubercles below the superficial layer around dilated blood-filled vessels. The inflamed granulation tissue penetrates into the walls of the small veins and extends as far as the uneven granulation tissue replacing the endothelium (Fig. 2). Some blood-filled sinuses and spaces are lined with confluent tubercles.

The peritoneum which has been removed together with the adnexa is overgrown by granulation tissue rich in cells, with numerous tubercles. Serial sections revealed a growth of inflamed funguslike granulation tissue penetrating from the neighbouring tubercles into a lymph space lined with epithelium (Figs. 3—4). The histological diagnosis was caseous

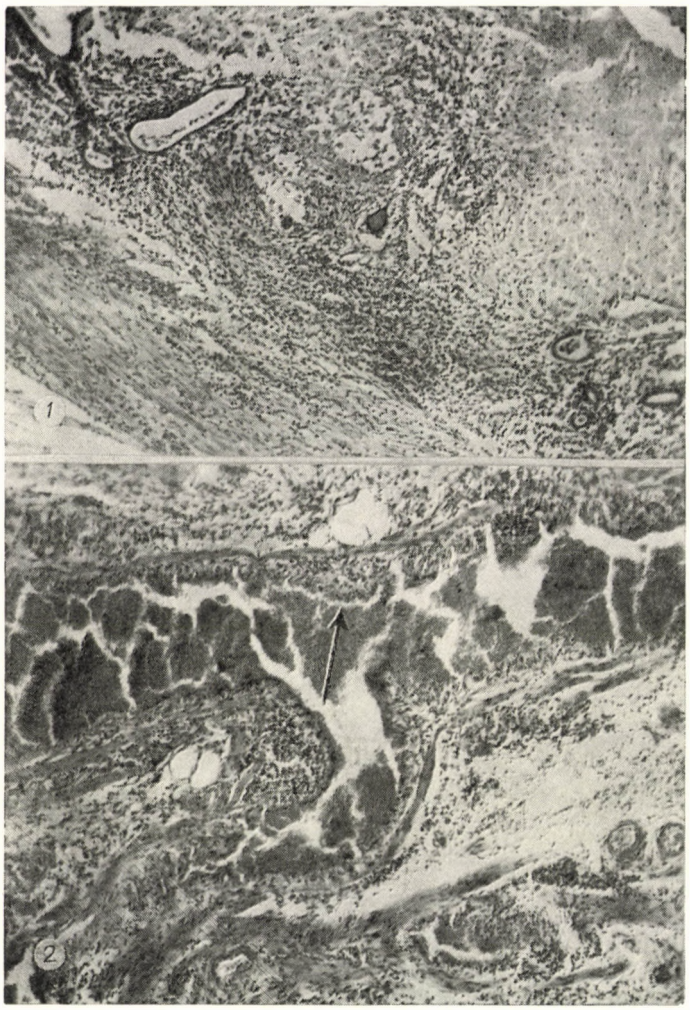


Fig. 1. Left tuba filled with caseous substance. Tubercles covering most of the mucosa
Fig. 2. The tuberculous granulation tissue of the left mesosalpinx had invaded the wall of the small veins

chronic tuberculosis and peritonitis, perisalpingo-oophoritis, chronic tuberculous phlebitis and lymphangitis.

The patient died 6 days after the operation, with rapidly spreading paralysis.

Post-mortem gross examination revealed the following: in the thick grey basilar arachnoid there are numerous grey solid granules from boiled semolina to poppy seed in size, and thick congested ventricular plexuses with many grey and dull yellow granules from poppy seed to millet or even pea size. A smear made from the basilar arachnoid contains a multitude of *Micobact. tuberculosis.* The lungs are free both these and the thin smooth pleura are covered with numerous solid granules from poppy seed to millet size. The hilar lymph nodes of the size of a bean are uniformly black in colour except for one single compact lymph node situated at the right main bronchus near the tracheal branching. This lymph node demarcated from its environment shows a solid white cut surface with blackish spots on its periphery, the liver is nor-

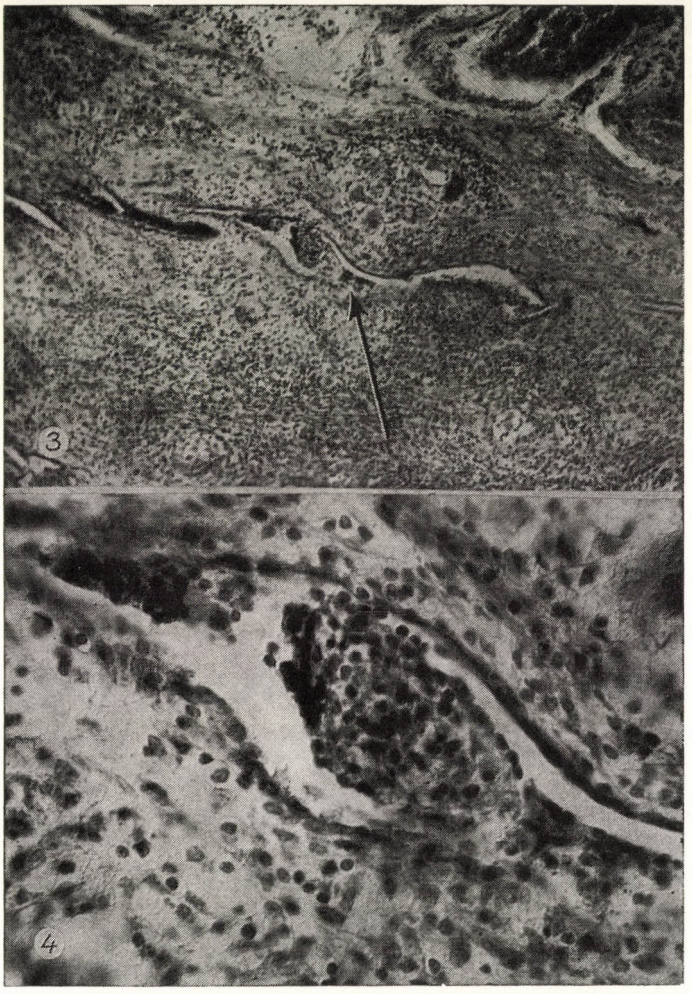
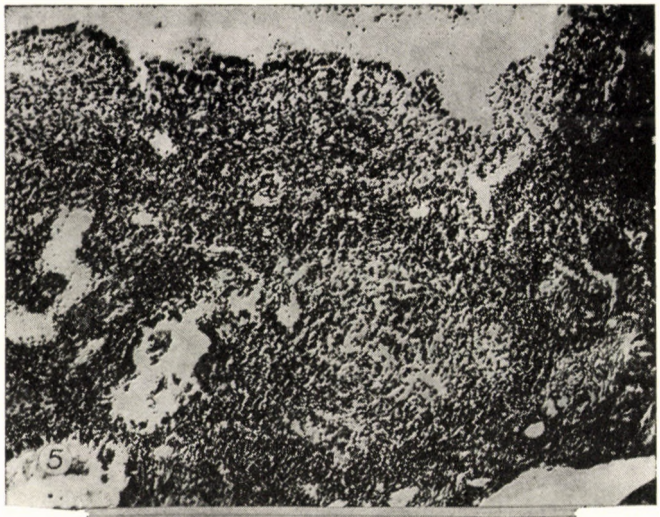


Fig. 3. Tuberculous granulation tissue penetrating a lymph space of the peritoneum
Fig. 4. Lymph space as on Fig. 3 showing granulation tissue with fungus-like inflammation. High-power

mal with a thin smooth capsule, and with soft substance, its cut surface is dull brown and fragile and contains numerous scattered solid dull yellow granules from semolina to poppy seed in size. The left oviduct and ovarium are missing. Spleen and kidneys are devoid of tubercles. The uterus is medium sized with a smooth surface and a solid wall of normal thickness, flattened lumen, closed cervical canal, normal cervix. Its mucous membrane is dull light-brown in colour with small yellowish spots. The right tuba is of pencil thickness, has a smooth surface and its lumen is empty. The right ovary displays superficial protrusions, is half a plum in size, with scarred follicles on the cut surface.

Microscopic examination revealed in the thin endometrium no structure characteristic of some functional stage. The gland lumina vary in width, the lining epithelium is detached in many places, with the nuclei situated in the cell centres (Fig. 5). Scattered epitheloid tubercles are present in the interstitium. The capillaries of the basal layer are wide and filled with blood. In the cervical mucosa there are some heterocellular tubercles amidst the normal glands (Fig. 6). The thin epithelium of the portion is of normal structure.



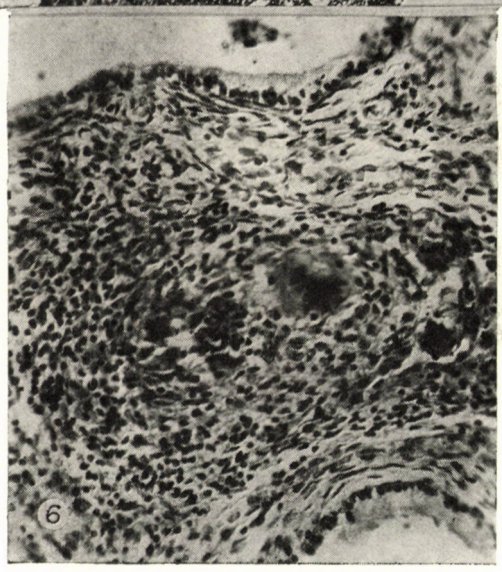


Fig. 5. Tuberculous uterine mucosa
Fig. 6. Tuberculous cervical mucosa

The lumen of the right tuba is slightly widened, its mucosa is rich in cells (Fig. 7), forms villous elongated processes and is covered in some areas by single-layered columnar or cuboid epithelium (Fig. 8). In the connective tissue there are numerous lymphocytes; neutrophiles and plasma cells are rare. The tissue does not present any manifest sign of tuberculosis (Figs. 7—8). The right ovarium is normal.

The ventricular plexus and the basilar arachnoid present numerous single or confluent heterocellular tubercles; those in the plexus are caseated.

In the lungs and the liver there are a few necrosed tubercles. Nearly the hole of the lymph node lying next to the right principal bronchus consists of roughly bundled homogeneous hyaline connective tissue, with a few small spots of black granules. The periphery of the lymph

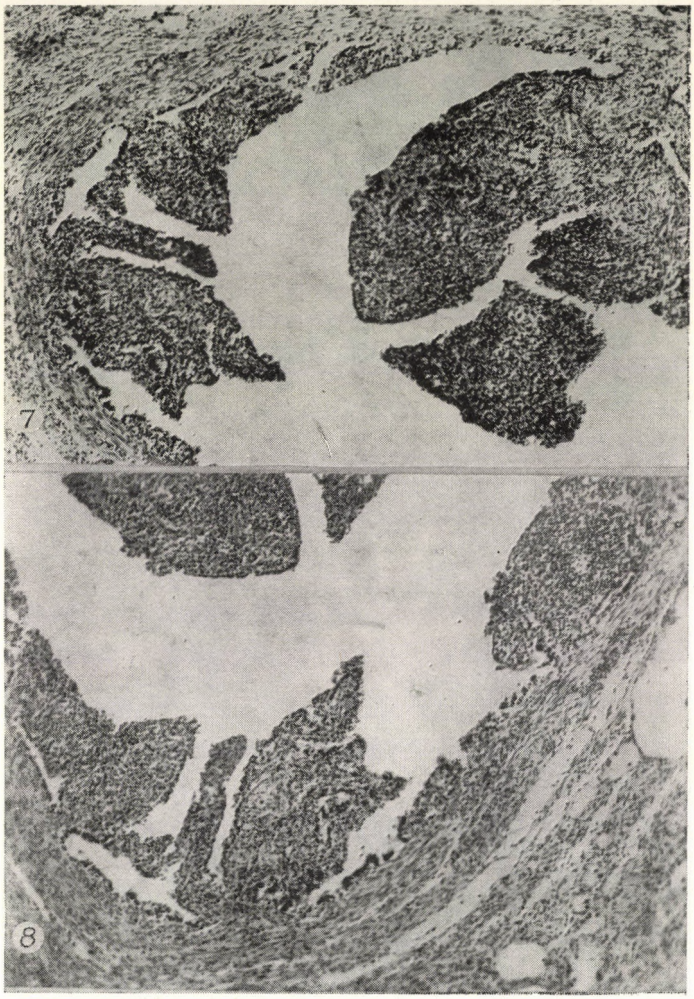


Fig. 7. Right tuba; inflamed granulation tissue forming abnormal rough folds in the place of the mucosa. Magnification, as in Fig. 1

Fig. 8. Right tuba. Low power

node consists of fibrous connective tissue more abundant in cells, with groups of lymphocytes and black granules. In the kidneys there are no tubercles only a few small lymphocytic infiltrations in the cortex. The spleen is devoid of tubercles.

According to the microscopic examination, death was due to generalized miliary tuberculosis and tuberculous meningitis. As to the mechanism of miliarization the following questions arise.

A) Which local tuberculous affection was responsible for the dissemination and

B) through what channels did that occur?

C) What had mobilized the tuberculous focus and started its generalization?

A) The probable sources were the removed left uterine adnexum and the scarred lymph node close to the right principial bronchus. In the latter which consisted of scarred connective tissue there was no sign of activity. Admitting the lymph node ever to have been infected, it must certainly be regarded as the oldest among all identifiable lesions. The active tuberculosis in the removed left tuba and ovarium and in the adherent broad ligament and peritoneum certainly was of longer standing than any in the rest of the genitals or in any other part of the organism. Consequently, the adnexum was the only probable focus from which tuberculosis could have disseminated. There is abundant evidence both from before the use of tuberculostatic drugs (STÜPER, 22; KOVÁCS, 14, etc.) and from after it (ALEX; STUDDIFORD; KOVÁCS; 14; etc.) to prove the possibility of a postpartal or postabortive miliarization of genital tuberculosis. STÜPER [22] refers to 364 recorded cases (apart from 5 observed by the present writers) of miliary and meningeal tuberculosis combined with pregnancy. In 53 out of 100 cases the fatal complication was due to genital tuberculosis plus pregnancy, while in combination with extragenital tuberculosis it was less dangerous and less frequent.

B) The venules in the mesosalpinx of the removed left adnexum were intruded by tuberculous granulation tissue. Similar tuberculous intrusions were found behind the adnexum in the wall of the peritoneal abscess and in the lymph spaces of the peritoneum. Consequently, the tuberculous process had disseminated from that point, by either the blood or the lymph. The portacaval anastomoses in the venous system of the parametrium and the paraproctium suggest haematogenous dissemination either from the vena rectalis cranialis via the vena mesenterica caudalis towards the vena portae or from the vena rectalis caudalis via the vena iliaca interna towards the vena cava. From the recent findings of KISS and TARJÁN [13] the venous system of the genitals is known to possess no valves so that there is no obstacle for the tuberculosis bacilli or for other substances to enter the major cardiac veins. On the other hand there are good reasons to allow for a dissemination along the lymph vessels. BRAUDE [4] asserts that the peritoneum covering the broad uterine ligament and the coecum is an elective resorbent area. According to HIGGINS and GRAHAM [8], the lymph vessels leading off the peritoneum empty not into the thoracic duct but into the mediastinal and pulmonary lymph nodes, *i. e.* for the greater part into the right lymphatic trunk and the thoracic vessels. BARTALS [2] furthermore points out that the plexus subovaricus formed from the ovarian lymph vessels, together with the lymph ducts of the corpus uteri and the tuba is leading into the preaortal and peri-aortal lymph nodes. The gross examination of the mediastinal, pulmonary and aortal lymph nodes failed to reveal tuberculous lesions.

Our findings seem to permit the statement that fatal miliarization was due to haematogenous rather than lymphogenous dissemination. Dissemination through the portacaval system offers a ready explanation for the miliary tuberculosis of the lungs and the liver. The meningeal process seems to indicate that the bacilli have entered the arterial circulation without forming any tubercles in the spleen and the kidneys. They probably penetrated the lungs, though the disease may have spread to the basilar arachnoid *via* direct routes recently assumed but not finally proved to exist between the peritoneal lymph ducts and the subarachnoidal spaces (RUSZNYÁK et al.; 12).

C) Four ways suggest themselves to explain mobilization, with ultimate miliary tuberculosis, of the possibly several years old tuberculosis of the uterine adnexa and its environment as the only active focus of the organism.

1. Progression of salpingeal tuberculosis without the influence of external factors.

2. Haemodynamic conditions, involving abundance of blood and vessels peculiar to the pregnant uterus, activating the until then latent tuberculosis.

3. Bacillaemia in consequence of interrupted pregnancy.

4. Bacillaemia in consequence of the surgical removal of the tuberculous adnexum.

Assumptions 1 and 2 are weakened by the absence of complaints indicative of generalized tuberculosis in the period before pregnancy and even prior to its interruption. Only on the 3rd day after the interruption did fever appear, accompanied by general symptoms of illness. Presumably, puerperal infection and inflammation of the genitals consequent upon the interruption of pregnancy caused the disease of the left tuba to flare up; so tuberculosis seems to have been activated not by pregnancy but its interruption. Literature has recorded a great number of similar cases. Surgical intervention may have been another mobilizing factor. STERN [21] and other authors emphasize, and all gynaecologists are well aware of, the risk of mobilization due to injury and on account of it in febrile states they not only abstain from operation on the tuberculous genitals but disapprove even of a simple uterine examination or curettage, whether or not under the protection of tuberculostatic drugs.

It appears of course legitimate to inquire after the time and nature of the dissemination. According to STERN [21], dissemination may be due to some injury whenever this has not been preceded by pathological symptoms, provided that the time elapsed since the injury is equal to what the granules needed to attain the developmental stage ascertained at the *post-mortem* examination. In our case the patient died 10 weeks after the interruption of her pregnancy, 6 days after the operation. The miliary tubercles were from poppy seed to millet in size, with caseation in the latter. According to BAUMGARTEN [3] and others, tubercles become conspicious on the 10th

to 14th day of their development, attaining millet size and beginning to caseate after 6 weeks. There is long-standing experience from before the use of tuberculostatic drugs that miliary tuberculosis in conjunction with tuberculous meningitis usually killed the patient within 2 to 3 months (WEPLER, 26). In the case under review the structure of the tubercles allowed to presume a period of development equal to that from the interruption of pregnancy to the patient's death.

The surgical intervention had an effect decidedly to the worse considering that immediately after it the general state of the patient began to deteriorate and meningeal symptoms occurred. Since six days later the patient died, and since she was being treated with various antibiotics, including streptomycin, the tubercles as the morphological signs of a recent bacterial dissemination, due to the surgical trauma could not fully develop. A conspicuous feature was the postoperative appearance of meningitis and its exacerbation. In morphologic interpretation of this case the facts must duly be considered that from the beginning the patient had been given antibiotics and that these weakened the bacteria and inhibited formation, growth, etc. of the tubercles. It is known that on the effect of tuberculostatics the previously fatal clinical course of tuberculous leptomeningitis has greatly changed, even complete recovery has been attained in 25 to 30 out of 100 cases, with a corresponding change in the histological picture. In our particular case the applied therapy, accidental and insufficient as it was, seems to have been unable to suppress development of the tubercles and their development can safely be regarded to have proceeded for at least 2 months. The left tuba and ovary and their environments were suggestive of long-standing chronic tuberculosis while the absence of tubercles from the mucous membrane of the right tuba and its normal size and form pointed to an affection subsequent upon the dissemination. The tubercles of the endometrium appeared not older than a few weeks. The fact that conception would have been possible had the right tuba and the uterine mucous membrane been intact and properly functioning, is in itself evidence in favour of a primary and localized left chronic adnexal tuberculosis. In tuberculosis of the genitals, especially of the tubae, the endometrium may for some time remain intact; KAUFMANN [11] observed that the infection spreads more readily from the tuba to the peritoneum than to the uterine mucous membrane and is more frequently the cause of secondary tuberculosis in the first-mentioned organ; he also found that sometimes the tuberculous tuba may freely communicate with a circumscribed abscess-like small focus, confined to a part of Douglas' cavity. This may have been the case in our patient. FROMME and HEYNEMANN [6] found an open abdominal end of the tuba in 50 per cent of their cases, allowing the pathogenic agent together with the caseous substance to reach the peritoneum. Unilateral localized genital tuberculosis seems

to be more frequent than it is actually noticed. One of us (SZENDI [23]) has lately described a case of isolated left ovarian tuberculosis and is of the view that the circulatory conditions of the genital blood and lymph system are responsible for the fact that by the time the latent disease can be identified on the strength of its vague symptoms, it has usually attacked both adnexa as well as the uterina cavity. Presumably, for similar reasons salpingeal and genital tuberculosis are usually described as bilateral processes (GÖGL—LANG [7]). As according to the current view, genital tuberculosis is mostly due to haematogenous bacillaemia spreading along the respiratory ducts, the node in the vicinity of the bronchus, as the only spot suggestive of a long-standing tuberculous lesion, seems to indicate the hilum to have been the point of intrusion and the primary focus whence the disease had penetrated possibly many years ago the left tuba. Finally, it merits attention and has been confirmed by other authors that an insignificant and apparently healed pulmonary infiltration is capable of giving rise to serious tuberculous lesions in the predisposed genitals. The organism though it was able to cope with the primary lesion of the hilum, succumbed to the resulting genital tuberculosis that had remained latent as an infectious focus until its final flare-up in the form of fatal miliarization.

Summary

Departing from the histological findings in a case of female genital tuberculosis, the reasons, the mechanism and the routes of generalization consequent upon gestation have been discussed. It has been established that unilateral genital tuberculosis does not exclude uterine pregnancy but interruption of the latter combined with puerperal infection and inflammation of the genital organs is apt to involve generalization. The process has been observed to start with the intrusion of the tuberculous granulation tissue into the blood and lymph vessels and to spread by haematogenous rather than lymphogenous dissemination of the bacilli to the genitals and the peritoneum. Even up-to-date therapy does not exclude fatal termination of generalization.

REFERENCES

1. BATIZFALVY, J. (1957): Orv. Hetil. *98*, 197. A női genitális tuberculosis (Female Genital Tuberculosis). — 2. BARTELS, P.: cited by Rusznyák et al. (19). — 3. BAUMGARTEN, I.: cited by Stern (14). — 4. BRAUDE, A. I.: cited by Rusznyák et al (19). — 5. FEKETE, L.—FORGÁCS, M.—MISKOVITS, G. (1953): Placenta gümökór magzati gümőkór nélkül. Orv. Hetil. *94*, 1224. (Placental Tuberculosis without Foetal Tuberculosis). — 6. FROMME and HEYNEMANN: cited by Gögl—Lang (7). — 7. GÖGL, H.—LANG, F. J. in Kaufmann and Staemmler: Lehrbuch der speziellen pathologischen Anatomie. Vol. II. de Gruyter. Berlin (1957). — 8. HIGGINS, G. M. and GRAHAM, A. S.: cited by Rusznyák et al. (19). — 9. JEDBERG, I.: cited by Schäfer (13). — 10. KARDOS, F. (1957): Feltöltéses röntgenvizsgálat alkalmazása és jelentősége a női ivarszervi tuberculosisban. (Application and Significance of Contrast X-ray Examination in Female Genital Tuberculosis) Tuberkulózis. *10*, 36. — 11. KAUFMANN: cited by Gögl—Lang (7). — 12. KIRCHOFF, H. (1956): Die Genitaltuberkulose der Frau. Ber Ges.. Gyn. Obst. *57*, 274. — 13. KISS, F.—TARJÁN, GY. (1956): Az arteriovenosus anas-

tomosisok szerepe az arteria uterina rendszerében. (Arteriovenous Anastomoses in the Uterine Arterial System). Magy. Nőorv. Lapja. *19*, 129. — 14. Kovács, F.—Gavallér, I. (1958): Klinische Erfahrungen in 550 Fällen weiblicher Genitaltuberkulose. Acta Med. Hung. *9*, 249. — 15. Kräubig, H. (1954): Genitaltuberkulose und Schwangerschaft. Geburtsh. Frauenheilk. *14*, 220. — 16. Kreibich, H. (1958): Über die tuberkulose Erkrankungen der weiblichen Genitale. Fischer. Jena, p. 126. — 17. Patat, P. (1957): Genitális tuberkulózis. Tuberkulózis *10*, 131. — 18. Patat, P. (1959): Izolált endometritis tuberculosa utáni terhesség és szülés. (Pregnancy and Delivery after Isolated Tuberculous Endometritis). Orv. Hetil. *100*, 268. — 19. Rusznyák, I.—Földi, M.—Szabó, Gy. (1955): A nyirokkeringés élet- és kórtana. Akadémia könyvkiadó. Budapest. (Biology and Pathology of the Lymph System). — 20. Schaefer, G. (1953): Female Genital Tuberculosis. Obst. Gyn. Survey. *8*, 461. — 21. Stern, R. (1930): Über traumatische Entstehung innerer Krankheiten. Fischer, Jena. — 22. Stüper, P. (1954): Statistische Untersuchungen über das Zusammentreffen von Schwangerschaft und Miliartuberkulose. Zbl. Gynäk. *76*, 953. — 23. Szendi, B. (1958): Isolierte, disseminierte, akute halbseitige Ovartuberkulose. Beitr. Klin. Tuberk. *119*, 163. — 24. Szendi, B.—Lakatos, I. (1958): A női ivarszervi tbc. előfordulása Békés megyében az utolsó 5 évben kiszűrt 143 eset kapcsán. (143 cases of Female Genital Tuberculosis Observed in the Last 5 years in the County of Békés). Tuberkulózis. *3—5*, 108. — (1958) Endometrium vizsgálatok activ tüdő tbc-s asszonyokon. (The Endometrium of Women Suffering from Active Pulmonary Tuberculosis). Orv. Hetil. *99*, 1066. — (1959) Histologische und bakteriologische Untersuchungen (durch Züchtung) der Uterusmukosa von 100 in aktiver (akuter oder chronischer) Lungentuberkulose leidenden Kranken. Beitr. Klin. Tuberk. *119*, 434. — 25. Szendi, B. (1958): A női ivarszervi tuberkulózis műtétes terápiája konzervatív lehet-e a tuberkulostatikumok birtokában? Magy. Nőorv. Lapja. *21*, 328. (Possibilities of Conservative Surgical Therapy of Female Genital Tuberculosis under Application of Tuberculostatics). — (1959). Ermöglichen die Tuberkulostatika eine Anderung etc? Beiträge zur Klinik der Tuberkulose. *120*, 225. — 26. Wepler, W.: in Kaufmann and Staemmler: (1956) Lehrbuch der speziellen pathologischen Anatomie. Vol. III. de Gruyter, Berlin.

ДАННЫЕ К МЕХАНИЗМУ ГЕНЕРАЛИЗАЦИИ ТУБЕРКУЛЕЗА ЖЕНСКИХ ПОЛОВЫХ ОРГАНОВ

Б. СЕНДИ и В. ХЕЙМ

Проводилось патогистологическое исследование для выяснения причин, механизма и путей генерализации туберкулеза женских половых органов, наступающей в ходе беременности. Было установлено, что при одностороннем туберкулезе половых органов может наступить внутриутробная беременность. Прекращение беременности (роды, выкидыш) и родовой сепсис или же воспаление половых органов и т. д. могут вызвать генерализацию туберкулеза. Согласно исследованному случаю данный процесс начинается врастанием туберкулезной грануляционной ткани и туберкулезных палочек в кровеносные и лимфатические сосуды, и к его распространению и генерализации заболевания предоставляют соответствующечю возможность кровеносные и лимфатические сосуды половых органов и брюшной стенки. В исследованном случае туберкулезная инфекция рассеивалась скорее по кровеносным сосудам. Генерализация даже при современных терапевтических возможностях может привести к летальному исходу.

BEITRÄGE ZUM GENERALISATIONSMECHANISMUS DER WEIBLICHEN GENITALTUBERKULOSE

B. SZENDI und V. HEIM

Ursachen, Mechanismus und Weg der im Laufe der Schwangerschaft entstehenden Generalisation der weiblichen Genitaltuberkulose wurde untersucht und festgestellt, dass bei einseitiger Genitaltuberkulose eine intrauterine Schwangerschaft zustandekommen kann. Die Unterbrechung der Schwangerschaft (Geburt, Fehlgeburt) und puerperale Infektion,

bzw. Entzündung der Genitalien usw. können eine Generalisation der Tuberkulose hervorrufen. In dem untersuchten Falle begann die Generalisation mit dem Eindringen des tuberkulösen Granulationsgewebes und der Bazillen in die Blut- und Lymphgefässe; zur Weiterverbreitung bieten sowohl die Blut- wie die Lymphsysteme der Geschlechtsorgane und des Bauchfells eine Möglichkeit. Im untersuchten Falle wurde die tuberkulöse Infektion eher hämatogen verschleppt. Die Generalisation kann selbst bei moderner Therapie tödlich sein.

Dr. Balázs Szendi
Dr. Vilmos Heim
Gyula, Megyei kórház, Hungary