TISSUE REACTION TO SPHERICAL AND LOBULAR HYDATID CYSTS OF ECHINOCOCCUS GRANULOSUS (BATSCH, 1786)

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Abstract. We have studied the tissue reaction to spherical and lobular cysts of *Echinococcus granulosus* (Batsch, 1786) on a set of 108 observations of hydatidosis in the liver and lung of pigs. Typical of an early response of the host to a growing hydatid cyst is a local allergic inflammation characterized by a heavy infiltration of eosinophiles. If the infection is massive, sensitization increases the tissue reaction, and there is a marked exudation of eosinophiles into the pseudocyst which results in the ultimate death and liquidation of the parasite. In an organ infested with lobular and multilobular hydatid cysts, this early type of a tissue reaction persists around pouches in the hydatid wall. Typical of this reaction is an epitheloid rim, a new production of connective tissue, and an inflammatory infiltration. In fertile, large-sized cysts, the acute inflammatory reaction disappears from the inner side of the connective tissue sheath, and the wall of the hydatid remains in a direct contact with a thick layer of hyalinly degenerated, fibrous, connective tissue. A moderate, chronical, infiltrate along the periphery of the encapsulation provides evidence for a balanced relationship between the parasite and its host.

In an earlier study on a set of 108 observations of hydatidosis in the pig (Vaněk 1980), attention has been given to the macroscopic appearance of hydatid cysts of *Echinococcus granulosus* (Batsch, 1786), mainly to irregularly shaped cysts with pouching walls. Pending on the degree of development of the pouching, the author distinguished between lobular and multilobular cysts. By using a specially designed method for making casts of hydatid cavities, it was possible to demonstrate their complicated structure (Vaněk 1975). We have tried to obtain an understanding of the causal genesis of pouching in the hydatid wall during various stages of the larval development, and of several other questions concerned with a tissue reaction in pigs with hydatidosis, using histological methods for this purpose.

MATERIALS AND METHODS

In 1972, we obtained more than 900 unilocular hydatids from pigs, brought from various places of the Warsaw district to the Warsaw abattoir for slaughter. The shape of the cysts has been discussed in an earlier paper (Vaněk 1980). We selected several hydatids of a different age and size in order to study their morphogenesis with histological methods.

Selected parts of hydatids together with the adjoining liver tissue were fixed with 4% neutralized formaldehyde, and washed in water for 24 hr. Having regard to difficulties in cutting the material, it had to be dehydrated in a series of alcohols for considerably prolonged periods, i.e., 2—3 hr in 50% alcohol, 4 hr in 80 and 90% alcohol, 3 hr in 96% alcohol and three times 20 min in 99% alcohol. Ultimately, the material was placed for three times 10 min in anhydrous acetone, transferred to a mixture of acetone and xylene, and finally placed in pure xylene. Having completed the clearance of the material in xylene, it was placed in at least 5 changes of xylene-paraffin (40 °C), then in three changes of pure paraffin where it was kept for 12 hr and, finally, selected parts of the hydatid were embedded in pure paraffin for 24 hr. Suitably arranged tissue pieces were placed in liquid paraffin in paper boxes and left to harden.

Short series of histological sections cut from the paraffin blocks with an MSE microtome were stained with a number of both standard and specific histological methods.
RESULTS

1. Tissue reaction to hydatid cysts of a small and medium size

The number of hydatids in the organ was sometimes remarkably high, once 81 cysts, once 57 cysts. The cysts were minute, many were regularly spherical in shape and at a stage of differentiation. The tissue reaction was about identical to that observed in a three-to five-month-old infection as found in a comparison with experimental materials (Dévé 1916). The development of an inflammatory encystment of a hydatid measuring 5 mm in diameter was uniform, its thickness was 0.4 mm (Plate I, Fig.1). The parasitary, hyalinoid membrane of a laminated structure was adjacent to a layer of elongate, epitheloid, cells (height 100—120 μm) sometimes resembling giant cells. Intermediary between this layer and the fibrous layer of connective tissue encapsulating the parasite was a layer heavily infiltrated with round cells, plasma cells and eosinophiles of which many penetrated to the membrane of the parasite. The encysting sheath of connective tissue (thickness 0.4—0.5 mm), of a parallel arrangement, was mildly infiltrated and, in its general character, a mature collagenous connective tissue. Signs of a more marked proliferation of fibroblasts were not observed in the middle zone. The situation was similar for most minute cysts (up to 10 mm in diameter) except for a few cysts, where we found in their middle zone an increased concentration of a microcellular infiltrate which produced a secondary lymphoid follicles without a germinal centre. The general picture of the tissue reaction was typical of the active phase of development of a connective tissue encystment around a young, growing, hydatid.

Once, the encystment of a minute hydatid cyst measuring 2 mm in diameter, was made up of maturing, newly formed, connective tissue. At the site of contact with the membrane of the parasite, there was only a layer of typical giant cells, but no epitheloid elements. An increased cellular infiltration was found in the part of the encystment which bulged in direction to the surface of the organ.

Several hydatid cysts attained a bigger size and they were lobular in shape. In these cases, an active type of tissue reaction persisted along the entire periphery of the hydatid cyst.

Sometimes, the inflammation weakened and disappeared from the surface of the growing cyst (10—20 mm in diameter). Characteristic of this process was the loss of the epitheloid border, the establishment of a direct contact between the connective tissue and the surface of the cyst, a hyaline degeneration of the connective tissue followed by its necrosis. This state suggested a balanced parasite-host relationship.

Both in the case of a heavier infestation of the liver (55 cysts) and a medium-high infestation (12 cysts), we observed an increased invasion of certain sites of the space between larger cysts and their epitheloid border by eosinophiles. Owing to this increase in the number of eosinophiles, the border was destroyed and the infiltration increased in the middle layer of the encapsulating sheath. Simultaneously, it affected the outer layer of connective tissue and consequently, the sheath lost its differentiated character. Sometimes, the epitheloid border became necrotic without a previous cell destruction (Plate I, Fig.2). In case of a heavy invasion by eosinophiles, the parasitary membrane was separated from remnants of the epitheloid border, and a focal calcification started in the destroyed structures. Generally, the inflammatory infiltration was remarkably heavy in sites which had not previously been affected by such advanced changes. Evidently, this development of the tissue reaction occurred at the time of commencement of regressive changes in the hydatid cyst.

The appearance of a shallow pouching was observed in several hydatid cysts, the connective tissue sheath of which had been affected by similar changes. An inspection of
connective tissue septa between individual pouches disclosed a focal proliferation of immature connective tissue (Plate II, Fig. 1). Bundles of young fibroblasts with distinct nuclei and a cytoplasm made visible by staining, traversed in a criss-cross pattern a faintly intracellular substance (Plate II, Fig. 2). Capillaries branching from arterioles which emerged from the outer layer of the fibrous, sclerotic, connective tissue adjoining the liver tissue, were seen to enter nodules made up of connective tissue. Also the nodules were moderately infiltrated mainly by eosinophiles. At sites containing nodules, the middle layer of the infiltrated connective tissue lying between the epitheloid border and the outer layer of connective tissue, disappeared. Although septa made up of this type of connective tissue were present between the individual pouches, they did not contain remnants of atrophic liver lobules. Actually, these septa originated from a local thickening of the enclosing connective tissue pressing into the encystment of the hydatid and deforming the cyst. This provided evidence that the process involved was not an active pouching of the hydatid cyst, but the beginning of regressive changes as evidenced also by the state of the wall proper of the parasite.

2. Septa between neighbouring cysts and the development of their histological structure

Using histological methods, we studied septa between two neighbouring hydatids of a different size. Smaller neighbouring cysts (8—10 mm in diameter) were still separated from the liver tissue by a sheath of connective tissue enclosing them. The tissue reaction, at that time an active inflammation, showed a tendency of developing in a massive eosinophile exudation if the vitality of the parasite became lessened. A pressure atrophy of the liver tissue wedged between the two cysts started at sites of production of the connective tissue sheath. In cross section, atrophic liver lobules present in the top- and bottom part of the originating septum, were of a typical, wedge-like shape, i.e., attenuated in direction to the centre of the septum. A complete atrophy of the liver tissue in the most narrow part of the septum separating the two cysts was caused by the distension of the growing cyst wall. A more loosely arranged collagenous connective tissue was found in the space between the encystments of the two cysts. If contained vessels, bile ducts and a remnant of atrophic liver cells (Plate III, Fig. 2). The size of the liver lobules was reduced partly by the growing parasite causing a pressure atrophy of the adjacent liver tissue, partly by an atrophy due to a venous stasis originating from a compression of local branches of the portal system (Plate IV, Fig. 1). Changes in the connective tissue encapsulating larger, neighbouring cysts (30—40 mm in diameter) were more advanced. The acute inflammatory reaction on the inner surface was replaced by a layer composed solely of parallelly arranged hyaline connective tissue in which the number of cells was reduced. The periphery of this layer was passing into the thickened and blended connective tissue of the portobiliary spaces remaining after an atrophy of the liver parenchyma. The connective tissue layer of these cysts measured 0.5 mm in diameter at the site where a septum was separating the two cysts. Atrophic changes in the connective tissue of the septum were caused by the growing cyst. Using histological methods, we examined a septum between two fertile cysts (cyst diameter 35 and 40 mm respectively) (Plate IV, Fig. 2A). The encystments of the hydatids were composed of a layer of hyaline, fibrous, connective tissue (thickness 0.4 mm) overlaid by an outer layer of irregularly arranged connective tissue. The total thickness of the sheath was 1.5 mm. The connective tissue was moderately invaded by inflammatory cells. Heavily basophilic foci and minute basophilic lumps representing a calcified and necrotic exudate, were present in the space between the hyalinoid membrane and the encysting layer. The thickness of
the laminated membrane of the parasite was 0.2 mm. Brood capsules containing vital protoscoleces as demonstrated by staining adjoined the germinal layer. The encystments of the two hydatids fused and attenuated until they formed a thin septum (0.2 mm) consisting of a necrotic, hyalinely degenerated connective tissue.

This description of the development of a septum was in agreement with the course of regressive changes in the liver tissue between two neighbouring hydatid cysts which originally had been at some distance from one another. However, in a multiple hydatidosis, we found cysts with two cavities indicating that two cysts might have fused at the very beginning of their development (Plate IV, Fig. 2B; Plate VI, Fig. 2). Sofar, we have been unable to confirm this assumption, because even in materials containing initial stages of hydatidosis, all small cysts (1.5 mm in diameter) were surrounded by their own encysting sheath, and no evidence was obtained of a disappearance of sheaths at the site of contact between two cysts.

3. Comparison of larger spherical and lobular cysts

Regressive changes were frequently found in a mixed type of infestation characterized by a simultaneous incidence of spherical cysts, those with an occasional pouching, and multilobular cysts. Also this factor impedes an estimate of the character of the tissue reaction as does, e.g., an insufficient fixation. In spite of these difficulties, we observed that the histiocytic inflammatory rim was either greatly reduced or fully absent in spherical hydatids. The deeper the pouching in the cyst wall, the more remarkable the differences between the inner surface of the pouching and the inner surface of the hydatid cyst. The pouch was still lined with a high, active rim, and sometimes, owing to a heavy infiltration, lymphoid follicles appeared in the middle layer. Most typical of the tissue reaction of the host was the state of the periphery of the inflammatory encystment. Several rows of atrophic liver lobules facing the peak of the pouch were flattened to thin discs. A comparison of liver lobules from the top and the bottom of the pouch indicated that the highest rate of growth of a hydatid cyst occurred in the pouch (Plate V, Fig. 1).

Sometimes, multilobular cysts of similar measurements were seen to be pouching from the central cavity in all directions. A high, active rim was found everywhere except for the top edges of connective tissue septa separating the pouches. The septa were made up of an excessively proliferating connective tissue, and atrophic liver lobules were present at their axes (Plate V, Fig. 2) In support of a multifocal growth of this type of hydatid cyst was the fact that the liver tissue was compressed by the peaks of pouches and caused a pressure atrophy of liver lobules.

In a multilobular cyst of this structure, the picture of the tissue reaction was disturbed by regressive changes in the parasite. If the exudate entered between the wall of the hydatid cysts and its encystment, and particularly, if this exudate was predominantly serosal in its nature, the epithelioid rim disappeared completely and there occurred a considerable focal proliferation of loose, fibrillar, connective, tissue. Owing to a progress in necrotic changes and a shrivelling of the parasitary membrane, many pouches were filled with a cellular exudate which succumbed to necrosis and became focally calcified. A new, histiocytic, demarcating reaction developed at the site facing the necrotic zone. Typical of irregularly lobular cysts in the lung were a marked necrosis of the encapsulating connective tissue layer and a new demarcating border (Plate VI, Fig. 1), although regressive changes in the parasite were less conspicuous. In addition to a necrosis, the chronic, inflammatory, reaction accompanied by the production of lymphatic follicles with germinal centres was intensified. This might have been an indication of the fact that a more severe local allergic reaction was developing in the lung. In support of this assumption was our observation of another, fertile, hydatid cyst of a bi-cystic appearance which measured 14×31 mm. The layer of connective
tissue enveloping the parasite was affected by a necrosis without exudation (Plate VI, Fig. 2). The bronchus was perforated by a pouch in the wall of the hydatid, and the proliferating epithelium of the bronchus produced a layer circumscribing the parasite’s membrane.

In several multilobular hydatids, pouches were bulging so widely from their narrow bases that incomplete septa of connective tissue originated. Their cavities appeared to be separated on histological sections. In a multiple hydatidosis in which hydatids displayed such a marked multilobular character, we observed the disappearance of the active, histioytic border around the hydatids and its replacement by an organized layer of fibrous connective tissue.

4. Pouching of old, larger, cysts

Pouches in the wall of a fertile hydatid cyst measuring 55 mm in diameter, were considerably elevated above the liver surface. The cyst wall (thickness 0.8 mm) consisted of fibrous, hyalinelY changed, connective tissue arranged in traversing layers. The connective tissue was not infiltrated at sites of contact with the hydatid cyst. The enclosing sheath around the parasite was greatly attenuated at the site of pouching; it was bulging prominently and invaded by numerous eosinophiles which were seen to concentrate even in the slit between the cuticular membrane and the encysting sheath. The inner surface of the sheath was formed by a layer of loose connective tissue which was heavily invaded by an inflammatory infiltrate. The tissue reaction at the site of contact with the pouch suggested its progressive nature, although the hydatid itself was of a considerable age.

The situation was similar in large multilobular cysts from another observation. The wall of the encapsulating layer was attenuated at the site facing the pouches, it was infiltrated, and its inner side was formed by epitheloid and giant cells. Eosinophiles concentrated in slits in the inner surface. A gradually developing tissue reaction resorbed lamellae split off the considerably thick cuticular membrane (optimum 0.7 mm). Extensive regressive changes in the membrane occurred mainly in the area of the pouching. Pouches contained considerably less brood capsules with protoscolecies than did the central part of the cyst. Macroscopically visible blisters appearing on the inner side of the wall were produced by an accumulating, proteinaceous, eosinophile, substance which uplifted split-off inner lamellae together with the germinal layer. In several places, there was a most advanced resorptive inflammatory reaction.

DISCUSSION

The pathology of hydatidosis has been studied on a number of animals. Hydatidosis in the goat was studied on a large number of animals (1014), of which 6.5% were found to be positive (Pandey 1971). According to the author’s description the shape of a heavily infected liver appeared to be almost “racemose”. Unfortunately, the study lacked a description of the shape of cysts. Histological changes in these hosts were similar to those in other hosts. Ohshima et al. (1967) maintained in their study on hydatidosis in cows that hydatid cysts were different in size and mostly spherical in shape. They observed septa on the inner surface of several hydatids, and their figures indicated the start of regressive changes in the cysts. A rare incidence of pulmonary hydatidosis in primates was described by Allen (1967) for Macacus mulatus. The encystment of hydatids in the lung was typical of a hydatid of E. granulosus. The animal died of anaphylactic shock. Histopathological changes were identical to those in man with pulmonary hydatidosis of which a detailed description was given by Hourël et al. (1961). In histopathological studies on hydatidosis, the pericystum, i.e., the sheath of connective tissue enveloping the larval bladder, received considerable attention. Studies on hydatidosis in man dealt mainly with the encapsulation of larger cysts in which the sheath of fibrous, hyaline, connective tissue was directly adjoining the superficial, already thick cuticular membrane. The character of the encapsulation was described by Helfer (1962) for cysts of a less specific location such as muscle hydatids. The main concern of the authors was either the presence
or the absence of inflammatory processes on the periphery of the sheath. The character of the inflammation was estimated mainly on the basis of the nature of the infiltrate, whereby importance was ascribed mainly to the incidence of eosinophiles, lymphocytes and plasma cells. According to Lorenzetti (1962), the incidence of eosinophiles depended on the developmental stage of the hydatid cyst and, more or less directly, on the chemical composition of the fluid in its cavity. Changes in the wall of a fertile hydatid prevented a dialysis of the hydatid's antigen, and this accounted for the disappearance of a hyperergic inflammation in the pericystium, a complete check of an eosinophile invasion and a considerable reduction in the number of plasma cells. His conclusions were in agreement with a severe eosinophile exudation during the initial development of the hydatid and conform to the situation occurring at the time of its sterility. A similar description of the development of hydatid cysts in an experimental infection was given by Dévé (1916), Dew (1925), Cameron and Webster (1969) and others. This description did not differ from our results. Recently, Gopalakrishna and Mohiyuddin (1974) describing hydatidosis in bovines and the buffalo, found distinct differences in the tissue reaction to a sterile cyst filled with fluid and to a degenerated and fertile cyst.

Lichtenheld (1904) gave a completely different explanation of differences in the structure of the sheath encapsulating sterile cysts. In his opinion, the effect of a sterile cyst on the surrounding tissue was weak and confined to the inner layer of the pseudocyst. It initiated the production of epitheloid cells and giant cells, and that of a thick, newly formed layer of connective tissue. By contrast, the irritation of a fertile cyst was so heavy that it prevented cellular reproduction in the vicinity of the hydatid, and caused a proliferation of connective tissue on the periphery of the pericystium. The author assumed that the presence of some young hydatids caused a necrosis of fine cells in their close vicinity, and initiated the development of a thick sheath of connective tissue around the parasite. Some of the cysts became fertile while others, although growing continuously, remained sterile. The inflammation retained its active character at the site of contact of the cuticular membrane and the inflammatory capsule. Gasse (1910) described histological differences in the appearance of the pseudocysts of sterile and fertile hydatid cysts. He drew attention to a particularly marked reaction to a hydatid with pouching walls and maintained that a massive exudation produced by destoyed eosinophiles occurred in the space between the hydatid wall and the inner layer of the pericystium composed of a high rim of epitheloid and elongate giant cells. Underlying this layer was a layer of maturing, newly formed, connective tissue. The peripheral layer contained a round-cell infiltrate with eosinophiles, and this bordered with the atrophic liver parenchyma.

In our material, an active epitheloid rim was found in younger spherical and also multilobular cysts. In bigger and mostly fertile cysts, this type of reaction persisted at sites of contact of the tissue with pouches in the hydatid wall. A higher rate of growth of the multilobular cyst was indicated by increased signs of a pressure atrophy at these sites; on the other hand, an increasing quantity of newly formed connective tissue was pressing as a septum in the cyst cavity. Owing to our present knowledge, it was difficult to decide, whether the proliferation of the germinal layer of the multilobular cyst was responsible for the intensive growth of certain parts of the hydatid, or whether the irregularly developing newly formed connective tissue produced a septum over which the cyst wall was bending. It is known that the internal pressure is relatively high in small hydatids. The outer pressure of the cyst on the surrounding tissue is brought forth by an increase in the inner pressure pending on an imbition of the fluid, and by the pressure of the gradually growing cyst. The inner pressure decreases as the cyst grows and becomes fertile (De Rycke 1966). Therefore, the production of irregular pouches must have started at an early developmental stage of the multilobular cyst, but it will need a detailed study of this material in order to obtain an understanding of the origin of a multilobular hydatid cyst. In close association with pouching is the hypothesis on an exogenous vesiculation in hydatids leading to the origin of outer, separated, daughter cysts inside a common encystment made up of connective tissue, reconsidered by Aguilar and Herrera Caballero (1964) in their study on pulmonary hydatidosis.

Generally, a marked eosinophile exudation in the space of the pseudocyst occurs at the time of dystrophic changes in the hydatid cyst and its starting collapse. Regressive changes might either be responsible for a lobular shape of the cyst or underline its sacculcation. Therefore, it is difficult to determine the reasons bringing forth such
irregular shapes in the hydatid cyst at this phase of development. This applies mainly
to a multiple hydatidosis in which the tissue reacts differently to the individual cysts
and does not always result in their resorption. Therefore, detailed studies on an expe-
imental material might make it possible to distinguish between a progressive and a
regressive sacculcation in the hydatid wall.

Benex (1968) figured sacculcation in hydatids from the liver and lung of horses similar in type
to that observed in our multilobular cysts. In his opinion, sacculcation in the wall is caused by a pro-
lifeation of the germinal layer. In order to confirm his hypothesis, he cultivated pieces of a multi-
lobular cyst wall. According to his results, the cuticular layer of the cysts wall was relatively thick.
In a growing culture he found structures similar to those generally described as a chitinome or cuti-
elome, and maintained that this type of growth was semblant of the mode of evolution of E. multi-
locularis. An occasional chitinome or cuticleome might be found on large hydatid cysts frequently
present in human hydatidosis. More recently, Cabanié and Lamy (1960) described the hyalinoid
membrane of a large liver cyst removed by surgery. Sacculcation was of a cabbage-like appearance
with an optimum diameter of 5 mm. Because the wall of the hydatid was very brittle, the authors
were unable to determine whether the cabbage-like processes were formed on the outer or inner
side of the germinal layer. It might have been helpful to compare these structures with several of
the so-called transitory forms between a hydatid cyst of E. granulosus and an alveococcus. A survey
of dubious forms of hydatidosis was given by Dévé (1934), who observed that alveolar structures
occurred in a topographical dependence on large and generally degenerate univesicular hydatid
cysts. A detailed study on the forms of growth in the wall of the parasite recurred from the bone
(osseous hydatidosis), which is typical of the production of complicated and even multiseptic com-
exes, might contribute to an elucidation of the ununiform proliferation of the wall of E. granulosus.

CONCLUSIONS

Using histological methods, we have tried to elucidate the genesis of pouching in the
wall of a hydatid cyst in relation to the tissue reaction of the host, and to find an ex-
planation of the problem that some regularly spherical cysts retain their regular shape
when enlarging in size, while others produce complicated pouches in their wall and attain
a large size and fertility in this multilobular shape. The tissue reaction to young, deve-
loping, hydatids is very marked and active. It is characterized by the production of
a three-layered, inflammatory coat surrounding the parasite. Typical of the site of
contact of this layer with the cuticular membrane is the presence of tall, slender, epi-
theloid cells which change sometimes into multinucleate giant cells. The layer abuts
a layer of loosely arranged connective tissue containing activated fibroblasts, reticular
fibres, which, generally, is invaded by round cells. The contemporary relationship between
the parasite and its host is best indicated by this layer. The intensity of immunological
processes brought forth by the defense reaction of the host, is disclosed by a marked
increase in the quantity of the infiltrate. A protracted disagreement between the anti-
genic effect of the parasite and defense reactions of the host evidences itself in an in-
crease in the infiltrate, mainly in the number of plasma cells which indicates an unbal-
anced relationship. Of importance in the reaction of the host to hydatidosis is the allergic
factor. A local allergic reaction occurs mainly in the first months of the diseases and
evidences itself mainly in an invasion by eosinophiles. If the host possesses strong de-
fence mechanisms, the parasite succumbs to regressive changes. The allergic inflamma-
tion is increased by decomposition products of the tissues, the parasite shrivels and
becomes liquidated in the resorptive granuloma. Sometimes, the process of cicat-
ization is checked by an early calcification of the focus. However, a locally restricted
calcification of the necrotic exudate and the sheath of connective tissue around the
parasite occurs even while the parasite is still alive, and the local allergic inflammation
is greatly accelerated by either an acquired or innate sensitization of the organism of
the host.
A gradual establishment of a balance of immune processes in the hydatid cyst and the host is accompanied by a decrease in the inflammation of the encapsulating tissue, a disappearance of the epitheloid border and the infiltrate, and the appearance of a layer made up of a hyalinely changed connective tissue at the site of contact with the cuticular membrane of the hydatid cyst. A fibroproliferation and a thickening of the connective tissue encystment occurs in the periphery. There, a pressure atrophy of the liver parenchyma is caused by the distension of the growing cyst wall, and the interlobular and portobiliary connective tissue fuses with the connective tissue of the encystment. In old and big hydatid cysts, this encystment has been shown to be at an advanced state of a hyaline dystrophy and necrosis. Decomposition products of the dying parasite reactivate the inflammation, and there is a resorptive inflammation inside the encystment. Frequently, the focus calcifies at a different stage and changes in a permanent calcificate.

It is of interest that the reaction of the host to the hydatidosis varies from tolerance to intolerance. Sometimes, the host reacts with a severe inflammation which soon causes the ultimate death of the parasite, sometimes, mainly in the case of a multiple hydatidosis, one or two cysts become fertile and attain a considerable size and age. This variation in tissue reactions accounts for considerable difficulties mainly in an evaluation of irregularly shaped hydatids, because it is difficult to determine whether their irregular shape has developed under the influence of the tissue reaction or should be ascribed to an uneven local proliferation and an ununiform growth of the hydatid cyst.

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ТКАНЕВАЯ РЕАКЦИЯ НА СФЕРИЧЕСКИЕ И ДОЛЬЧАТЫЕ ЦИСТЫ ECHINOCoccus GRANULOSUS (BATSCH, 1786)
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Резюме. Изучали тканевую реакцию на сферические и дольчатые цисты Echinococcus granulosus (Batsch, 1786) на основе 108 случаев гидатидоза печени и легких свиней. Ранняя тканевая реакция вокруг цист проявляется собой как аллергическое воспаление, с типичной высокой инфильтрацией эозинофильных лейкоцитов. При массивном заражении сенсибилизация ведет к увеличению тканевой реакции и к резкой экссудации эозинофильных лейкоцитов в псевдокисту, что приводит к отмиранию и ликвидации паразита.

В органах, зараженных дольчатыми и многодольчатыми цистами, ранняя тканевая реакция сохраняется вокруг мешков в стене цисты даже и в последующие стадии развития. Эта реакция характеризована эпителиальной каймой, новым образованием соединительной ткани и воспалительной инфильтрацией.

В зрелых, больших цистах воспалительная реакция исчезает на внутренней стороне и стенка цисты остается в прямом контакте с толстым слоем гиализированной волокнистой соединительной ткани. Слабый хронический инфильтрат на периферии капсулы свидетельствует о равновесном взаимоотношении между паразитом и хозяином.

REFERENCES


The 8th Conference of the Parasitological Society of the German Democratic Republic 19.—22. 9. 1979 in Cottbus

The 8th national conference of the Parasitological Society of the German Democratic Republic was organized in Cottbus jointly by local centres of medical and veterinary hygiene services. The papers and discussion at the conference dealt with ecological problems of parasitology. Within the framework of the 30th anniversary of the German Democratic Republic the introductory paper of Prof. Hiepe reviewed the development of parasitology during that period. Other introductory papers were concerned with the basic concept of mathematical expression of the ecological system (A. Knijnenburg) and terminological problems in ecology and parasitology (L. Britz). The remaining papers were divided into three large groups.

In the first group of papers K. Odening at first defined the natural and cultivated landscape, including in these two categories everything except towns and villages, i.e. different types of natural landscape on one hand and agricultural lands on the other. In these types of terrain he determined the concept of ecological system and animal populations living in it. The clarification of the parasite population concept is important in the target control of mass parasitoses causing losses in animal production and troubles in public health. The main paper was devoted to parasite populations in aquatic environment (L. W. Reimer), with particular emphasis on the role of intermediate hosts. The paper by B. Rosický elucidated the role of the existence of natural focus and elementary foci of tick-borne meningo-encephalitis in Central Europe. J. Slais reported on the focus of alveococciosis in the Sumava region, with the possible occurrence of this disease in the Thuringian Forest in the German Democratic Republic. A few papers were concerned with the incidence of parasites in some game fishes living in cold oceans and with their importance in the know-
Fig. 1. Encystment of a hydatid cyst measuring 5 mm in diameter in a multiple hydatidosis. From the left: 1 — Hydatid wall, 2 — layer of epitheloid cells, 3 — layer of a microcellular infiltration, 4 — connective tissue encystment, 6 — liver tissue (HE — × 200). Fig. 2. Encystment (0.8 mm in thickness) of a hydatid cyst measuring 13 x 17 mm. 1 — Thick cyst wall, 2 — necrotic epitheloid rim, 4 — hyalinely changed connective tissue arranged in layers, the main component of the encystment, 5 — layer of sclerotic connective tissue after a pressure atrophy of the liver tissue, 6 — disturbed structure of the parenchyma with dystrophic changes (HE — × 65).
Fig. 1. Nodular thickening of immature connective tissue (a) in hydatid encystment differentiated distinctly from fibrous connective tissue (b) adjacent to the liver parenchyma which contains a larger artery persisting after an atrophy of the functional tissue. (Weigert van Gieson — × 45.) Fig. 2. (detail of Fig. 1) — Proliferation of young fibroblasts differentiated from the loose basic substance by staining (Goldner — × 170.)
Fig. 1. Asymmetric atrophy of liver lobules caused by the pressure of the growing hydatid. Parenchymal remnant of wedge-like shape in separating septum (HE — × 50). Fig. 2. Septum between hydatids measuring 33 and 33.5 mm respectively (Inset). Disappearance of liver parenchyma in attenuated part of septum (left) and fusion of connective tissue encystment around the two cysts. (HE — × 35.)
Fig. 1. Atrophy of liver lobules in sclerotic connective tissue of septum. Marked increase of connective tissue around central vein and in liver lobules. Disappearance of liver parenchyma owing to an atrophy of a local venous stasis. Packed sinusoids of a dark colour. (HE — × 65.) Fig. 2. A — Greatly attenuated septum between two fertile hydatid cysts (cyst diameter 35 and 40 mm respectively). B — Hydatid cyst in longitudinal section suggesting fusion of two hydatids (7 and 8 mm in diameter).
Fig. 1. Approximately spherical hydatid cyst (20 × 25 mm) producing individual pouches (Inset). Note active epithelial rim inside the pouch and a marked pressure atrophy and basophilia of atrophic liver lobules in direction of growth of the pouch. (HE — × 7.) Fig. 2. A — Total view of multilobular cyst situated at the margin of the liver lobe (longitudinal axis 24 mm). B — High epithelial rim in individual pouches; compressed lobules in parenchyma in direction of pouch growth (HE — × 4.5).
Fig. 1. Necrotic exudate and encystment made up of connective tissue in a regressively changing multilobular hydatid cyst from the lung. a — Hydatid wall, b — necrotic exudate, c — necrotic connective tissue, d — calcified focus, e — new, demarking, epitheloid, rim, f — chronic inflammatory infiltration of connective tissue encystment, g — compressed liver tissue (HE — × 50). Inset: Longitudinal section through a regressively changing focus measuring 12.5 × 14 mm. Fig. 2. Necrosis of connective tissue encystment in fertile hydatid cyst from the lung, with chronic infiltration in the periphery. Brood capsules with protoscoleces on cyst wall (HE — × 60). Inset: Longitudinal section through hydatid cyst measuring 14 × 31 mm, on the margin of the liver.