Echinococcus Disease of the Left Ventricle
A Clinical, Radiologic and Electrocardiographic Study

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Although echinococcus disease has a high incidence in many parts of the world and is therefore not rare, localization of this parasitic disease within the heart walls has not been recognized very often. In the vast majority of recorded cases a correct diagnosis of the condition was only made at postmortem examination. Prior to the publication of this paper surgery has been attempted in very few cases and the results have been rather discouraging because the patients came to operation in the late, complicated stages of the disease in most instances.

Diagnosis of this rare disease of the heart is of more than academic interest. Our experience in the early recognition and subsequent successful treatment of three consecutive cases of echinococcosis of the left ventricle prior to the occurrence of any complication has emphasized to us the importance of this disease which is less uncommon than has been thought.

In this communication we wish to discuss the clinical, radiologic and electrocardiographic features of uncomplicated echinococcosis of the left ventricle.

Case Report

Case 1. P. S. was a 43-year-old farmer. In 1950 a round mass in the left lung field, which apparently fused with the cardiac shadow, was discovered by radiologic examination. One year later the abnormal mass was somewhat larger. In August 1951 he was admitted to Professor Dr. V. Armand-Ugon's surgical service (Colonia Gustavo Saint-Bois, Montevideo) for further study.

His past history and family history were noncontributory. He gave no history suggesting illness; he complained of no symptoms attributable to a cardiac disease or to hydatid anaphylaxis. On physical examination of his heart no murmurs or thrills were elicited. Cardiac size was normal by percussion. Blood pressure was 130/80. The pulse was regular, at a rate of 80 beats per minute. No signs of congestive heart failure or acute pericarditis were present. Routine laboratory examinations were essentially normal.

Roentgenograms of the chest revealed marked prominence in the outline of the left ventricle in the posterior-anterior projection (fig. 1). A round mass of uniform density, superimposed on the heart shadow, was visible in the left lateral projection. By fluoroscopy this prominence had neither systolic expansion nor paradoxical contraction. No calcification was seen within, or at the edges of the abnormal cardiac shadow. Lung fields were clear and the diaphragm moved freely. A tomographic examination showed that the deformity was located mainly at the posterior region of the left ventricle. Angiocardiography showed a normal outline of the cavity of this structure.

An electrocardiogram (fig. 2) showed a sinus mechanism with a rate of 75 beats per minute and left axis deviation in a horizontal heart. T waves were inverted in leads I and aV\textsubscript{1} and upright in aV\textsubscript{R}. In the precordial leads, T waves were inverted from position 5 to position 9, inclusive, with a maximal inversion in V\textsubscript{4} and V\textsubscript{5}. Isoelectric RS-T segments were present in all leads. There was slight slurring of R waves in the precordial leads from V\textsubscript{4} on. No abnormal Q waves were present.

On Sept. 15, 1951 the patient was operated upon by Prof. Armand-Ugon, after anesthesia with sodium pentothal, cyclopropane, ether and oxygen. A left anterolateral thoracotomy at the fourth left intercostal space with no rib resection was per-
formed. A multilocular hydatid cyst localized at the posterolateral region of the left ventricle and protruding into the pericardial cavity was found. There were no pericardial adhesions at all in the region where the cyst bulged from the surface of the heart. The left ventricular wall underlying the cyst was of normal thickness. Through a cystostomy the hydatid membrane, seven daughter cysts, and a good amount of normal looking hydatid fluid were removed. After resection of a portion of excessive adventitia and irrigation of the internal surface of the cyst with ether, the adventitia and the pericardium were closed with interrupted sutures. The chest wall was closed in layers.

After developing a moderate left pleural effusion, the patient made an uneventful recovery and was discharged from the hospital 18 days after operation. He has been followed at regular intervals since discharge. The heart shape returned to normal immediately after operation. There has remained a persistent elevation of the left diaphragm. An electrocardiogram taken one year after operation was not changed from the preoperative tracing.

Case 2. P. C., a 24-year-old farmer, was admitted to Professor Dr. P. Larghero-Ybarz's surgical service (Pasteur Hospital, Montevideo) because of a left paracardiac round mass discovered on fluoroscopic examination after an acute illness diagnosed as acute bronchitis. His past history and his family history were noncontributory. On physical examination the heart sounds were normal and neither murmurs nor thrills over the precordial region were present. Blood pressure was 100/60. The pulse was regular at a rate of 65 beats per minute. Heart size by percussion was within normal limits. The patient was in no distress and there were no signs of cardiac failure. The lungs were clear. Laboratory tests were normal, except for an early, as well as a late, positive intradermal Casoni test.

The electrocardiographic report (fig. 3) was as follows: sinus rhythm, at a rate of 60 beats per minute; heart in vertical position; symmetrically inverted T waves in leads I and aV\(_5\); and upright T waves in aV\(_6\); very deep and acute inversion of T waves in leads V\(_2\) through V\(_6\), inclusive; slightly inverted T waves in V\(_1\) and diphasic T waves in V\(_2\); very small R waves in leads V\(_1\) to V\(_4\), inclusive, becoming suddenly higher in V\(_4\) and V\(_5\); QRS complexes of the r-s-r' type and of very low voltage in

**Note:** T wave inversion in leads I, aV\(_L\) and V\(_1\) through V\(_6\). There also is slight slurring of R waves from V\(_1\) on.
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V₂; absence of abnormal Q waves, as well as of abnormal displacements of the RS-T segments in all leads. Precordial leads taken at the level of the third intercostal space recorded similar, but not so marked, changes. This electrocardiographic pattern, highly suggestive or diagnostic of an important degree of myocardial ischemia of the anterolateral wall of the left ventricle in a 24-year-old man with no past or present history of heart disease, aroused our interest immediately.

Roentgenograms of the chest showed a clear cut deformity of the cardiac shadow in the apical region, best seen in the right anterior oblique position. Some areas of calcification at the level of the left ventricle were also present (fig. 4). A tomographic study, performed by Drs. E. R. Zerboni and A. Gorlero-Armas, confirmed the presence of both the ventricular deformity and spotty calcification. Angiocardiograms were made, using a Fairchild camera and 40 cc. of a 75 per cent solution of Neo-Iopax. In three consecutive exposures of the levoangiocardio gram taken at one-second intervals, obliteration of the outline of the left ventricular cavity at its apical region was demonstrated (fig. 5).

In view of the history, the radiologic findings, the positive Casoni test and, last but not least, our previous experience with echinococcosis of the heart, we felt that the electrocardiographic changes could be caused by a hydatid cyst located at the anterolateral region of the left ventricle.

Because of the electrocardiographic findings (deep inversion of the T waves and very small R waves in some precordial leads) and the angiocardio grams, we were able to warn the surgeon before

Fig. 3. Case 2. Preoperative electrocardiogram. Note the "coronary" type inversion of T waves in positions 3, 4, 5, and 6 of the precordial leads, as well as the marked reduction of R waves in some of these leads.

Fig. 4. Case 2. Roentgenographic left lateral view of the chest. Note the round shadow superimposed to the cardiac silhouette and calcifications.

Fig. 5. Case 2. This levoangiocardio gram shows a clear amputation of the outline of the left ventricular cavity at its apical region.
FIG. 6. Case 2. Comparative study of precordial leads, positions V₄ and V₅, and an intraeystic lead recorded during operation. Note the low R waves and the elevated RS-T segments registered in the latter (see text).

operation of the great probability of extreme thinning of the wall of the left ventricle underlying the cyst.

Assistant Professor Dr. Luis M. Bosch-del-Marco operated upon this patient, performing a left anterior thoracotomy with resection of the fifth rib under general anesthesia. A 6 by 4 cm. oval mass located slightly above the apex of the left ventricle was found. There were firm adhesions of both pericardial layers at the region where the abnormal mass bulged from the heart surface. Having failed to obtain any substance from a tap through the adhesions, a rather ample incision was made on its surface. After cutting through a 2 cm. layer of fibrous tissue, a few fragments of a highly modified hydatid membrane and some quantity of caseous substance (“hydatid putty”) were obtained from the cavity of the cyst. A finger introduced in this cavity failed to feel contractions of muscle fibers at its very bottom, but did detect the thinness of the myocardial wall beneath the cyst. An electrocardiogram made with an electrode inside the cavity of the cyst (fig. 6) showed very low R waves and elevated RS-T segments. Both findings were thus confirmatory of our prediction and demonstrated that the muscular layer beneath the cyst was thin, yet excitable. The cavity was emptied of all hydatid material, washed with ether and injected with 400,000 units of penicillin. The myocardial incision, as well as the pericardial sac, was closed with interrupted sutures. The thoracic wall was sutured in layers and two drains were left in the left anterior and posterior pleural sinuses.

The patient was discharged one month after operation and made an uneventful recovery. Serial postoperative electrocardiograms have shown slight improvement, but a marked degree of T-wave inversion and changes in the QRS complex has persisted.

Case 3. J. S. B., a 40-year-old housewife, who had lived on a farm for a few years, was admitted to Professor Dr. P. Larghero-Ybarz’s service (Pasteur Hospital, Montevideo) on Oct. 21, 1951, because of a second attack of acute cholecystitis. As she had had late toxemia of pregnancy with very high blood

FIG. 7. Case 3. Comparative study of precordial leads taken at the standard positions and at the level of the third intercostal space, respectively. QRS and T wave changes are more obvious in the latter.
pressure, she was referred to our service for evaluation of her cardiovascular status.

On clinical examination, the heart sounds were found to be normal. Blood pressure was 130/90 and the pulse was regular at a rate of 65 beats per minute. On fluoroscopy we found a bulge at the upper part of the left ventricle. This deformity did not expand, but showed a line of marginal calcification. Because of these features a diagnosis of hydatid cyst of the left ventricle was made.

The principal electrocardiographic findings were: inverted T waves in leads I and aVL; progressive reduction in amplitude of the R waves from V1 through V5; taller R waves from V1 on; slight slurring of the R waves in some left precordial leads; isoelectric RS-T segments; diphasic T waves in V5, V6, V7, V9, and V9 and inverted T waves in V3 and V4. Precordial leads made at the level of the third intercostal space showed (fig. 7); inversion of T waves in all positions, but most marked in positions 4, 5, 6 and 7; isoelectric RS-T segments; progressive diminution in amplitude of the R waves from V1 through V5 with return to a normal amplitude from position 6 on. On the basis of our previous experience, these changes were thought to be caused by a cyst located at the high lateral region of the left ventricle and a thin myocardial wall underlying the cyst, as was the case in the previous observation.

Drs. Zerboni and Gorlero-Armas' roentgenologic and tomographic studies revealed "an irregularly calcified image, highly suggestive of hydatid cyst, located at the superior region of the left ventricle" (fig. 8). Angiocardiography revealed a filling defect in the outline of the upper region of the left ventricular cavity facing the zone where marginal calcification was present. Laboratory examinations were all within the range of normal. Casoni's intradermal test was negative.

On Nov. 14, 1951 Professor Larghero-Ybarz performed a left anterior thoracotomy with resection of the fifth rib under general anesthesia. A hard, partially calcified, helmet-like mass adherent to the pericardium and bulging out from the surface of the high lateral region of the left ventricle was found. This mass turned out to be a degenerated hydatid cyst with a very thick and calcified adventitia. After puncture of the cyst that did not yield any fluid, an incision of the adventitia was made and a certain amount of caseous material was obtained after curettage of the cyst cavity. The internal layer of the adventitia showed patchy necrosis. The cavity of the cyst was carefully washed with hypertonic saline solution. Palpation of its base showed remarkable thinning of the muscular wall underlying it. After partial resection the adventitia was closed with interrupted sutures. The pericardium was closed in the same way. The thoracic wall was closed in layers.

The patient was discharged from the hospital 15 days after operation in good condition. She has been followed postoperatively at regular intervals. She is now doing very well and is quite active. The cardiac deformity disappeared. Serial electrocardiograms taken after operation show the same changes that were present before operation, although the T waves are now not so deeply inverted.

**DISCUSSION**

According to Dévé and most of the authors who have dealt with this disease, hydatid cyst of the heart is always single. The hexacanth embryo, after traversing both hepatic and pulmonary capillary networks, reaches the left heart chambers and passes through the coronary circulation to reach the myocardium of any one of the four heart chambers or the cardiac septa. In the course of a few weeks the hexacanth embryo becomes vesicular and grows at a rather quick rate. The hydatid vesicle, giving rise to a unilocular cyst, exerts pressure on the surrounding myocardial fibers which become more or less ischemic, depending on the degree of pressure caused by the parasite and the resistance of the cardiac tissue. As the cyst grows, the tissue reaction in the host leads to the formation of a fibrous capsule called adventitia, which shows cellular infiltration and becomes gradually thicker.

Due to the particular density of the myo-

**FIG. 8. Case 3. Tomography. Calcification of the ventricular bulge shows concentric distribution.**
cardium, the development of the hydatid cyst is restrained. This phenomenon is more obvious when the cyst is located in either ventricle. This is the reason why in most cases there is a reactive daughter cyst formation and/or why the primary cyst undergoes other changes such as degeneration or suppuration. When one or more of these changes take place, the adventitia becomes thicker, denser and even partially calcified and causes comparatively more harm to the surrounding myocardial structures.

The primary hydatid cyst shows a marked tendency to rupture either into one of the four heart chambers or into the pericardial sac. This accident gives rise to well known complications that we will not comment upon here in detail. While the pericardium usually reacts to a hydatid cyst by developing adhesions, the endocardium develops no reaction and is easily penetrated by the cyst.

Until rather recently it was thought that the primary cyst was more frequently lodged in the walls of the right heart chambers than in those of the left. In a review of this subject made in 1928, Dévé pointed out that this concept was wrong and that there were comparatively more cases of primary hydatid cysts located in the wall of the left chambers, particularly the ventricle, than in the wall of the right chambers or in the cardiac septa. Dévé expressed the opinion that this was due to the fact that the left ventricle has a relatively richer coronary circulation.

The growth of a hydatid cyst within the myocardial wall of any one of the heart chambers usually produces a localized bulge in its silhouette, a feature which makes possible roentgenologic recognition of the disease. It should be pointed out that this localized deformity of the heart is readily detectable only when the hydatid cyst has reached a certain size. Medical literature abounds in cases where cardiac cysts ruptured before causing a deformity of the organ. In some of these instances the diagnosis of echinococcosis of the heart was made only after recognition of some of its complications. Therefore early detection of localized deformities of the cardiac silhouette in echinococcosis of the heart is the only way of making an early diagnosis when surgery can cure the disease and prevent accidents and complications. This is particularly true, when we realize that uncomplicated hydatid disease of the heart has no particular clinical picture.

Roentgenology must be given credit in our various cases and in some other cases for first calling attention to the disease. Fluoroscopy represents a very important diagnostic method, because the deformity and calcifications may be discovered and studied in all projections. It usually gives very valuable information about the size, shape, and location of the abnormal mass, about its contour and about its movements. However, we feel that kymography may be much more accurate in the study of the latter.

Roentgenograms taken in different projections permit a more detailed study of ventricular deformity and the importance, the location, and the extension of calcifications. As pulmonary hydatid cysts very seldom or never show calcification, every calcified shadow in the periphery of the heart, if not due to constrictive pericarditis, should suggest the possibility of a hydatid cyst. When spotty areas of calcification are seen in the cardiac shadow, one may also think of echinococcus disease, if calcification of the heart valves can be ruled out. Intramural fibromas may also calcify, but this disease is extremely rare. Tomography usually gives very good information about the size, shape and location of the intramural cyst, as well as about the outline and position of the calcific zones, if present.

Angiocardiography has proved to be most helpful in the study of hydatid cysts of the left ventricle. Up to the moment of writing our first two papers on this subject we did not find bibliographic data relative to angiocardiography applied to the study of this condition. Although only our last three cases were studied by this method, we feel that this procedure gives valuable information when the hydatid cyst bulges into the left ventricular cavity; it is also particularly helpful in estimating the thickness of the myocardial wall underlying the cyst. If the hydatid vesicle is primarily located in the superficial layers of the myocardium (case 1), the layer of uninvolved
myocardium prevents the cyst from producing deformities of the ventricular cavity. If, on the other hand, the cyst is closer to the endocardial surface, the ventricular cavity may show localized deformities which are easily detectable by angiocardiography (cases 2 and 3). In such cases the surgeon must be extremely cautious in handling the cyst because the layer of myocardium underneath it is thin. Angiocardiography is also helpful in establishing the differential diagnosis between a hydatid cyst and an aneurysm of the left ventricle. Both diseases may cause a bulge in the silhouette of this cardiac chamber, but their angiocardiographic pattern is quite different.22

Electrocardiography, according to our recent experience1, 2, 6, 14, represents a very reliable diagnostic method as far as echinococcal disease of the left ventricle is concerned.21, 24, 25

Looking at the preoperative tracings of our three cases (figs. 2, 3 and 7), we find several changes that are common to all of them:

1. Inversion of T waves in leads I and aV L.

2. Upright T waves in lead aV R in two cases.

3. Absence of abnormal Q waves in the standard, as well as in the precordial unipolar leads.

4. Small R waves (cases 2 and 3) in those precordial leads taken from sites overlying that part of the left ventricular wall in which the cyst is implanted.

5. Abrupt increase in amplitude of the R waves immediately beyond the above mentioned site.

6. R waves of normal or nearly normal amplitude in the precordial leads in cases in which the hydatid cyst occupies only the most superficial layers of the myocardium of the left ventricle (case 1).

7. Slight slurring of the QRS complexes in leads made from precordial positions overlying the cyst.

8. Inversion of the T waves (of “coronary” type in some cases) in precordial leads which show changes in the QRS complexes.

9. Absence of abnormal displacements of the RS-T segments in the standard, as well as in the unipolar limb and precordial leads, even in the positions where T-wave changes are most marked.

10. A lesser degree of change in the QRS complexes and T waves in the precordial leads recording the potential variations of the peripheral regions of the cyst.

When a hydatid cyst of the left ventricle has not ruptured into the cavity of this chamber (as in our three cases), the subendocardial layers of the myocardium beneath the cyst remain excitable and the Purkinje network does not show changes worthy of comment. Therefore the excitation process in these fibers gives rise to R waves, whose amplitude depend on the thickness of the myocardial wall separating the cyst from the ventricular cavity. If the cyst is superficially located and has grown mainly toward the pericardial cavity, the R waves of the precordial leads made from positions on the chest wall immediately over the cyst will show a normal amplitude or a slight reduction in voltage (case 1, fig. 2). If, on the contrary, the cyst is more deeply located in the myocardium and an altered adventitia has caused comparatively more harm to the surrounding muscle fibers, the R waves in the precordial leads made from positions over the cyst are considerably smaller than normal (cases 2 and 3, figs. 3 and 7). At the same time there is a relative reduction of the voltage of the R waves in such cases, when compared with the amplitude of the other components of the QRS complex. When the precordial electrode is located over the peripheral zones of the cyst, the QRS complexes exhibit a normal amplitude. Thus, in cases in which the cyst pushes deeply into the myocardial wall, the voltage of the QRS complexes in multiple precordial leads give accurate information concerning its size. We believe that the low voltage of the R waves and the QRS complexes is caused by a reduction in thickness of the myocardial wall beneath the cyst and not by the presence of the cyst itself. This interpretation of the significance of the size of the R waves received some confirmation by comparison and agreement of the findings at operation and the electrocardiogram, as in cases 2 and 3. (See fig. 6.)

Because of the integrity of the myocardium
in the region underlying the cyst, the absence of abnormal Q waves and of intraventricular conduction defects is readily explainable.

There still remain to be explained the isoelectric RS-T segments and the inversion of the T waves. Echinococcosis of the heart is much more frequent in young than in elderly people. Therefore there is little likelihood that degenerative heart disease will exist concomitantly with echinococcosis. It is probable that electrocardiographic changes will only be caused by the cyst and its adventitia in the myocardial wall.

Although the cause of persistent elevation of the RS-T segments in ventricular aneurysm is not agreed upon, it is agreed upon that the elevation is caused by tension during systole at the junctional zone where fibrous replacement joins living myocardium. It seems to us that the same circumstances are present in echinococcus disease of the left ventricle with marked thinning of the myocardial wall; yet, as far as we know, no case of hydatid disease of the left ventricle has shown abnormal displacement of the RS-T segments, even after surgical removal of the cyst. Therefore, a sounder explanation for the elevation of the RS-T segments in ventricular aneurysm other than transient "injury" must be looked for. We feel that isoelectric RS-T segments in multiple precordial leads in cases of abnormal bulging in the outline of the left ventricle is a rather reliable sign, in addition to the absence of abnormal Q waves, in differentiating between echinococcus disease and ventricular aneurysm, although it is known that not all the cases of the latter show persistent elevation of the above mentioned segments.

Pathologic specimens of echinococcus disease of the heart, by demonstrating a process of ischemia and compression atrophy of the myocardial fibers in close relationship with the cyst, explain the inversion of the T waves in multiple precordial, as well as in standard and unipolar limb leads. This process of ischemia, caused by the cyst in the myocardial wall near the precordial electrode, is revealed by inverted T waves. The inversion of T waves is most marked in the leads made from sites on the chest wall over the center of the cyst and decreases gradually as the electrode registers the potential variations of the more peripheral zones (figs. 2, 3 and 7).

As we have found that changes in the T waves (chiefly in precordial leads) have been most helpful in the correct localization of the cyst, multiple precordial leads at different levels of the chest must be recorded in cases where echinococcus disease of the left ventricle is suspected. In case 3 (fig. 7) there were some changes in the QRS complexes and T waves in the standard precordial leads, but they were not so marked as those encountered at the level of the third intercostal space. This indicated that the cyst was located at the high lateral region of the left ventricle, but was sufficiently large to cause some changes even at the level of the fifth intercostal space.

It seems quite probable that in case 1 the precordial electrode only recorded the potential variations of an ischemic wall near it. In cases 2 and 3, where only a very thin muscular wall was interposed between the cyst and the ventricular cavity, the precordial electrode may have well recorded not only the "ischemia" pattern of the wall bearing the cyst, but also the potential variations of the opposite wall. This would explain the deep inversion of the T waves, since we know that T waves produced by activation of the posterior wall are recorded as inverted waves in anterior chest leads. This deep inversion cannot be explained on the basis of disturbances in the repolarization process of the ventricular wall located near the recording electrode, because, if this were so, T waves should show a low amplitude just as the R waves in the same leads show low voltage. (See fig. 3, leads V3, V4, and V5 and fig. 7, position 4, 5, and 6 of the precordial lead taken at the level of the third intercostal space.)

If this explanation seems suitable, one must then accept that the more marked the degree of T wave inversion in some precordial leads the thinner the muscular wall beneath the cyst in the left ventricular wall. This interpretation has been confirmed in all three of our cases by angiocardiology and by surgery. A case previously reported by two of us
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(J. D. and E. J. C.) and the case of Pérez Fontana and Sapirza add support to this explanation. The latter authors have demonstrated that, if the cyst is quite superficially implanted in the myocardial wall, T-wave inversion may disappear some time (weeks or months) after operation.

Inversion of T waves in lead aVL may be interpreted as the result of transmission to the left arm of an altered repolarization process occurring in the left ventricle. If the hydatid cyst is located in the apical region of a vertical heart, T-wave inversion may be present in lead aVF instead of aVL.

Positive T waves in aVR may either be the consequence of forward rotation of the apex (case 1) or of right ventricular cavity potentials (case 2) in a patient with inverted T waves in the precordial leads. T-wave changes in aVL and aVR, in their turn, explain why these same waves are inverted in lead I.

Emphasis must be put on the definite superiority of multiple precordial leads in the study of cases suspicious of hydatidosis of the left ventricle. While in these leads the QRS complexes, RS-T segments and T waves may exhibit changes of diagnostic value, the standard, as well as the unipolar limb leads show changes which are present in a number of other cardiac conditions.

Summary

1. The clinical, electrocardiographic and radiologic features of three cases of echinococcus disease of the left ventricle, which were diagnosed correctly, localized accurately and operated upon thereafter successfully, are presented and discussed.

2. Hydatid disease of the heart may remain latent for some time and give symptoms only when the myocardial cyst ruptures into a heart chamber or into the pericardial sac. Because of its latency, early recognition of this disease is only possible by radiologic examination of the chest and/or by electrocardiography.

3. Fluoroscopic and roentgenologic examination may show a localized deformity, with or without marginal calcification, of the outline of the left ventricle. In some cases spotted areas of calcific density may be seen at the zone of implantation of the cyst in the myocardial wall. Angiocardiography may demonstrate localized deformities and filling defects within the ventricular cavity.

4. A typical electrocardiographic pattern, yielding information concerning the position of the cyst and the thickness of the myocardial wall underlying it, is described.

5. Early surgical intervention is strongly recommended as the only reasonable way of dealing with echinococcus disease of the left ventricle.

Summario in Interlingua

1. Es presentate e discutite le caracteristicas clinic, electrocardiographic, e radiologic de tres casos de morbo echinococal del ventriculo sinistre le quales eseva correctemente diagnosticate, accuratemente localisate, e subsequente mente operate con bon successo.

2. Morbo hydatidic del corde pote remaner latente pro alucin tempore e exhibir symptomas solmente quando le cyste myocardial se rumpe e se discarga a in le camera cardiac o a in le sacco pericardial. A causa de su prolongate stato latente, le precoce recognition de iste morbo es possibile solmente per medio de examines radiologic del thorace e/o per medios electrocardiographic.

3. Le examine fluoroscopic e roentgenologic pote exhibir un deformitate local—con o sin calcification marginal—in le contorno del ventriculo sinistre. In alucin casos areas maculate de densitate calcifie pote esser visible in le zona de implantation del cyste e in le pariete myocardial. Angiocardiographia pote demonstrar deformitates local e defectos del replenanimento intra le cavitate ventricular.

4. Nos describe un typic configuration electrocardiographic que provide information in re le position del cyste e le spissitate del subjacente pariete myocardial.

5. Nos sublinea le importantia de precoce identification del morbo. Isto es le sol methodo rationabile de attaccar casos de morbo echinococal del ventriculo sinistre.

Addendum

Since this paper was submitted for publication, we have studied six additional cases of uncomplicated hydatidosis of the left ventricle.
All of them showed findings similar to those shown by the three cases presented in this paper.

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