Thrombosis of the Superior Vena Cava Following Osteomyelitis and Septicemia[1]

REPORT OF CASE WITH AUTOPSY FINDINGS

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SUPERIOR vena caval thrombosis is the rarest of all venous thromboses. Obstruction of the superior vena cava, whether partial or complete, is in itself a rare clinical entity. In 1933 Graham[3] and his associates were able to collect from the world literature only 309 cases of this condition. Obstructions due to thrombosis occur much less frequently than to other causes.

In 1936 Ochsner and Dixon[4] collected in a very comprehensive review only 120 cases of superior vena caval thrombosis and added two more cases of their own to the series. Since then, four additional cases have appeared in the literature.[5] From their analysis Ochsner and Dixon concluded that forty-four cases (36.6 percent) were caused by phlebitis, thirty-five (29.1 percent) by external compression, and twenty-eight (23.3 percent) by mediastinitis, while in thirteen (10.8 percent), the cause was unknown. The etiology of the phlebitis in the cases reported was: 16.6 percent idiopathic, 10 percent luetic, 5.8 percent pyogenic, 3.3 percent tuberculous, and 0.9 percent traumatic. The case which is reported herein falls in the group of pyogenic infections.

The clinical findings of obstruction of the superior vena cava depend upon the stasis of blood in the tributaries draining into this vessel and are, therefore, mostly limited to the upper half of the body. The venous pressure in any of the vessels draining into the

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3. W. Erhlich, H. C. Ballon, and E. A. Graham, "Superior Vena Caval Obstruction with a Consideration of the Possible Relief of Symptoms by Mediastinal Decompression," *J. Thoracic Surg.*, III (1933), 352.

4. A. Ochsner and J. L. Dixon, "Superior Vena Caval Thrombosis," *J. Thoracic Surg.*, V (1936), 641.

5. M. Szour and R. Berman, "Contribution a l'étude des thromboses dans la phthsis pulmonaire; un cas de thrombose de la veine cave superieure," *Rev. de la Tuberc.*, II (1936), 1068.

F. J. L. Blasingame, "Thrombotic Occlusion of Superior Vena Cava, Associated with Established Collateral Circulation." *Arch. Path.*, XXV (1938), 361.

L. A. Soloff, "The Syndrome of Superior Vena Caval Obstruction," *Am. Heart J.*, XVIII (1933), 318.

superior vena cava is increased in contrast to a normal venous pressure found in the lower extremities. An increased venous pressure in the upper half of the body with a normal circulation time is said, by Lian and Abaza,[6] to be diagnostic of superior vena caval obstruction. However, Soloff[7] claims that this cannot be so if the vena cava is completely occluded; that it may be true only if occlusion is partial. This latter conclusion seems the most reasonable.

In experimental obstruction of the superior vena cava of dogs, Carlson[8] determined that venous pressure increases immediately after obstruction to a level at least 100 percent higher than normal, and that it takes fourteen to twenty-nine days to return to normal, as collateral circulation develops. Because of venous stasis, there occurs transudation of fluids into the tissue spaces with resultant edema and swelling of the affected area, which comprises the face, neck, and upper extremities. Both edema and cyanosis are said to be aggravated when the patient assumes a horizontal position and relieved, when erect. If the condition lasts long enough, there is evidence of development of collateral circulation, with dilatation of the veins and, ultimately, varicosities in the anterior chest wall. When the occlusion has occurred above the opening of the azygos vein, this vein and its tributaries form the chief channel for the return flow of blood from the upper part of the body to the heart. When the occlusion is below that level, the superficial and deep abdominal vessels and the vertebral plexus are the main channels forming the collateral circulation. Thus all blood from the upper extremities must return to the heart through the inferior vena cava.[9] Dyspnea is a prominent symptom, particularly marked while the patient is in a horizontal position and relieved, when he is erect. Pleural effusion, particularly on the right side, and coughing are frequent developments. Rarer findings are cerebral symptoms due to venous stasis, such as headache, vertigo, somnolence, prominence of the eyes, increase of the cerebrospinal fluid pressure, auditory disturbances, etc. These symptoms, particularly headache, are aggravated by coughing, sneezing, and shouting.[10]

6. C. Lian and A. Abaza, "Dissociation de la pression veineuse et de la vitesse circulatoire signe characteristique de l'obstruction de la veine cave superieure," *Bull. et mém. Soc. méd. d. Hop. de Paris*, LI (1935), 730.
7. Soloff, *op. cit.*
8. H. A. Carlson, "Obstruction of the Superior Vena Cava," *Arch. Surg.*, XXIX (1934), 669.
9. *Ibid.*
10. A. Castellanos and R. Pereiras, "La Cavografía Superior," *Arch. latino-am. de cardiol. y hemat.*, VIII (1938), 149.

Prognosis in these cases is indeed very poor. It depends upon the rapidity of development and the severity of the obstruction. Of all patients with thrombosis of the superior vena cava, 75.9 percent die in the acute phase.[11] If the acute stage is survived, the symptomatology will depend on the amount of collateral circulation that has developed. Should this be adequate, there may be no symptoms, and the condition may be compatible with life, as shown in the case reported by Blasingame,[12] whose patient lived to the ripe old age of ninety-three.

REPORT OF CASE

R.L., a sixteen-year old boy, was admitted on June 7, 1940, as an emergency to the wards of the Presbyterian Hospital, with chief complaints of draining sinuses in the left hip, fever, dyspnea, and palpitations.

The history, obtained mostly from the patient's father because the patient was too ill to coöperate, was very indefinite as to dates and events. However, as far as could be gathered, the patient had been entirely well up to one month before admission, when he developed pain in the region of the left hip, without tumor or swelling but with high fever. He was admitted to a clinic in his home town, where two incisions, which drained profusely during his stay in that hospital, were made in the region of the left hip. The history is rather indefinite at this point, but shows that the patient was extremely ill with a very high temperature and a marked toxemia. He was given three transfusions and a third incision had to be made. His temperature is said to have gone down, and all incisions became continuously draining sinuses. Eight days before admission to the Presbyterian Hospital, after the patient had returned home, he rather suddenly developed marked dyspnea, pain in the chest, cyanosis, and palpitations. Fever again appeared and he became extremely ill. He brought with him X-ray plates of the pelvis, which are said to have been taken before the operation and which showed no obvious pathology; also a film of the chest in which the heart shadow was peculiar in appearance, globular in shape, and extending considerably toward the right.

A brother of the patient, a twelve-year old boy, had been in the wards of the Presbyterian Hospital about four months previously with congenital heart disease, type undetermined, and heart failure. He died, apparently of acute heart failure, about three weeks after leaving the hospital. Our patient, however, gave no history of previous cardiac decompensation.

Examination on admission revealed an extremely ill young boy whose temperature was 102° F., pulse, 160, respiration, 55; the blood pressure could not be recorded. The patient was obviously in shock and apparently

11. Ochsner and Dixon, *op. cit.*
12. Blasingame, *op. cit.*

in cardiac failure. He was cyanotic and dyspneic, with cold, sweaty, clammy skin. He was undernourished and very irritable. The head, neck, ears, nose, and throat were not remarkable. Neurologic examination was negative.

Examination of the chest revealed hyperresonance of the entire left side and flatness of the entire right hemithorax. The breath sounds were distantly audible over the left chest, accompanied by an occasional *râle*. Over the right base the sounds were also distant, but they were roughened and followed by coarse *râles* and crackles. The voice sounds were distant over the entire left chest while over the right and, particularly, in the parasternal region, there was marked egophony. The heart sounds were not audible over the left side of the chest; they were very distant over the right parasternal region. The left border could not be made out on percussion. The rate was rapid and irregular. The pulse was very feeble; on occasions a deficit was noticed. The abdominal wall was held moderately tense. There was slight tenderness over the right upper quadrant, where the liver edge was palpable. The extremities were atrophied. There was no subcutaneous edema. Moderate cyanosis was observed. The patient was able to move his legs without pain. Over the left thigh and just below the iliac crest there were three draining sinuses, each admitting a probe five inches long. A nonfetid, yellowish, watery fluid drained profusely from all three openings, and this drainage increased with deep respiratory movements and with pressure over the iliac fossa and lower abdomen. No deformities were found and no tenderness elicited over the spine and lower back.

Impressions on admission were that the thoracic pathology consisted of a spontaneous pneumothorax on the left side, with pleurisy and marked effusion on the right, masking a consolidation of the right lung from either a pneumonic or atelectatic process. There was obvious heart failure and arrhythmia, the nature of which could not be recognized; a probable pericarditis with pericardial effusion; and an infectious process of the left hip.

Admission radiographs (Figure 1) showed no pathology in the lumbar and thoracic vertebrae, but there was extensive destruction of the left ilium, interpreted as due to osteomyelitis. Films of the chest (Figure 2) showed, on admission, a pneumothorax on the left side with about 10 percent collapse of the lung and a fluid level reaching the left tenth rib. The left border of the heart was straightened out. There was a dense homogeneous shadow, obliterating practically the entire right side of the chest and completely obscuring the right border of the heart and the right leaf of the diaphragm. Only a small portion of the right lung was visualized at the level of the fifth, sixth, and seventh ribs, posteriorly. The conclusion was: "Pneumothorax on the left side with slight pleural effusion. Massive pleural effusion on the right side. The straight border of the heart is suggestive of pericardial effusion."

Laboratory studies on admission revealed: Hgb., 72 percent; R.B.C., 4,270,000; W.B.C., 10,650; neutrophils, 73 percent; lymphocytes, 27 percent. Urinalysis: albumen +, a few epithelial cells in the sediment. The feces contained hookworm and whipworm ova. Kahn test of the blood was negative.

It was believed that the intrathoracic pathology was in some way related, or secondary, to the process in the left hip. There were two main possibilities: first, Pott's disease with bilateral pleural effusion, pericardial effusion, and a cold abscess draining down the psoas space into the gluteal region; second, osteomyelitis of the hip with septicemia and metastatic foci in the pericardium, pleura, and lung.

The patient was placed on a Karell diet and digitalized fairly rapidly. However, despite thorough digitalization throughout the illness, the heart continued weak and hyperactive; the arrhythmia persisted. On June 9, his second day in the hospital, a thoracentesis of the right chest was performed and 500 cc. of cloudy, blood-tinged fluid was removed, with some relief of the dyspnea. The following day, June 10, 1,000 cc. of fluid were again removed. The patient, however, continued to have rapid and difficult breathing so that oxygen was administered by means of a B.L.B. mask. The fluid from the chest revealed 20,000 leukocytes, with 65 percent neutrophils. On culture, slightly hemolytic staphylococci were grown from both specimens.

The following day, June 11, the patient continued to be dyspneic and moderately cyanotic, even though oxygen was given continuously. Cyanosis was particularly marked in the upper extremities. He was extremely restless, and complained frequently of pain in the abdomen and over the precordium. On the following day his condition was unimproved. Thoracentesis was again performed and 1,100 cc. of fluid obtained. A pericardial tap yielded 100 cc. of blood-tinged fluid. Through some mistake in the laboratory, culture was made only of the pleural fluid, and again a hemolytic *Staphylococcus aureus* was grown. An electrocardiogram taken this same day revealed a moderate right axis deviation; a fairly low voltage of all leads and inversion of the T-waves in leads 2 and 3 with a "digitalis type" of T, typical of overdigitalization. In addition, there was a complete dissociation between auricles and ventricles, a fairly rare finding. On the night of June 12, slight swelling of the patient's face was first noticed and he complained of pain in the arms, which were very cyanotic.

On the thirteenth, thoracentesis was again performed and 1,000 cc. of fluid removed. Radiograph (Figure 3) of the chest, obtained with a portable machine immediately after the thoracentesis, showed a pneumothorax on the right side with 30 percent collapse of the right lung, a dense shadow obscuring the right base, and another dense shadow obscuring the left base and left diaphragm, interpreted as bilateral pleural effusion. The left border of the heart still appeared as a straight line, and the cardiac image was enlarged. That night the arms and hands became extremely cyanotic, and marked dependent edema appeared in the elbows and forearms. Examina-

tion revealed palpable tender cords in the arms and axillary region and a hardened palpable and slightly tender jugular vein on the left side. The conclusion was reached that an obstruction of the superior vena cava existed, probably due to thrombosis of that vein and its tributaries. By June 14, the cords were more prominent, the swelling and cyanosis more marked. On this same day the following tests were tried in an attempt to localize the extent of the thrombosis: circulation time with decholin from left arm to tongue was 33 seconds (normal, 14-16 seconds); from the right arm to tongue, 20 seconds. Venous pressure taken at the left radial vein was 300 mm. of water (normal venous pressure ranges from 40-120 mm.). A tourniquet placed around the left forearm raised the venous pressure at the wrist to 350 mm. but, when it was applied at the elbow, upper arm, and axilla, the venous pressure at the wrist failed to rise, remaining at 300 mm. The pressure on the right wrist was 290 mm., but rose when the tourniquet was applied at the right elbow, forearm, and even at the axilla. This was interpreted to mean that the obstruction extended well down into the left brachial vein, reaching the elbow. On the right side, however, the findings proved that the obstruction (thrombus) probably did not extend below the right axillary vein. The thrombosis was thought to commence above the point where the azygos vein enters the vena cava, with the thrombus extending along both the left and right innominate veins into both axillary veins, thence into the brachials, more so on the left, and also up the left external jugular. No evidence was found of extension into the right external jugular or into the deeper vessels of the neck.

Blood cultures taken on June 14 and June 24 were positive for hemolytic *Staphylococcus aureus*. The pus obtained from the draining sinuses of the hip was sterile on one occasion but, on another, also yielded *Staphylococcus aureus*.

On June 13, chemotherapy with sulfamethylthiazol was started. Six gm. were given daily for the first three days; then 4 gm. daily for five more days, with no sign of improvement in the patient's condition. A change was made to sulfathiazol, with a dose of 4 gm. daily for the following six days. Salyrgan was also administered intravenously, 1 cc. every other day. The general condition of the patient remained unimproved but, by the eighteenth, the swelling and cyanosis of the arms had decreased. On this day the chest was tapped, following which the patient complained of pain on the right side of the chest, especially when breathing.

Films of the chest taken on June 20 showed re-expansion of both lungs, marked thickening of the pleura on the right side with possibly some encapsulated fluid there. Both lungs showed increased peribronchial markings. A dense shadow obscured the left base. The cardiac shadow was somewhat smaller than in the films taken on admission. There was widening of the superior mediastinum to the right, which the radiologist thought might be due to a dilatation of the superior vena cava. On June 21 the

following note was made: "There are evidences that collateral circulation is beginning to compensate; veins in the upper anterior chest wall are becoming more and more prominent, while the arms are less cyanotic and less edematous. The heart is working under great strain, and digitalis in usual doses has failed to slow it down. Fluid continues to accumulate in the right pleural space but has not become frankly purulent. Chemotherapy has not altogether controlled the fever. Purpuric spots have appeared. The patient is holding his ground and there is hope that he may pull through yet." Venous pressure on this day:

Left arm:	Needle at wrist	280 mm. of water
	Tourniquet applied 6″ above needle	500 mm.
	Tourniquet applied in upper arm	500 mm.
	Digital pressure on axillary vein raised the pressure by only 10 mm. to 290 mm.	
Right arm:	Needle at wrist	190 mm. of water
	Tourniquet applied 6″ above needle	500 mm.
	Tourniquet applied in upper arm	440 mm.
	Digital pressure on axillary vein raised the pressure by only 10 mm. to 200 mm.	

Electrocardiogram taken June 24 continued to show complete dissociation between the auricles and ventricles, a tendency toward right axis deviation, and a digitalis type of T-wave. The white blood cells remained elevated throughout the illness, ranging from 10,000 to 21,000. The percentage of neutrophils was always elevated, at times even up to 94.

Death took place suddenly on June 26, nineteen days after admission and approximately two months after onset of the osteomyelitis.

AUTOPSY REPORT[13]

A well-developed, markedly emaciated white boy. On the left side of the neck, a cordlike structure is palpable in the region of the external jugular vein. The superficial veins throughout the anterior portions of the upper part of the chest, over the shoulders, and over the proximal third of each upper arm, particularly on the left side, are prominent. The thorax is symmetrical. There is some swelling laterally over the left hip, where there are three longitudinally placed wounds suggesting old surgical incisions and averaging 3 cm. in length. Two of them are situated immediately below the crest of the ilium, and the third, farther down, above the head of the femur. The borders of these openings are violaceous and a thin, yellowish fluid flows from all of them. The subcutaneous fat is reduced to 0.2 cm. in the anterior abdominal wall.

13. The autopsy was performed by Dr. Enrique Koppisch, of the Department of Pathology, School of Tropical Medicine, to whom the writer is indebted for this abstract of the autopsy protocol.

The peritoneal cavity contains one liter of thin, clear, amber-colored fluid. The inferior margin of the liver reaches 4 cm. below the costal border in the right midclavicular line. The spleen reaches the costal border but does not extend below it.

Not less than a liter of clear, thin, amber-colored fluid fills the left pleural cavity. A few adhesions bind portions of the left lung to the parietal wall. The right pleural cavity is opened under water but no gas escapes from it. Laterally and inferiorly, there is a pocket formed by adhesions, lined by granulation tissue, and filled with yellowish purulent material. This pocket extends down to the diaphragm. The remainder of the right pleural cavity contains somewhat turbid amber-colored fluid to an amount of about 500 cc. Dense fibrous adhesions bind the upper and middle lobes to the mediastinum and the lower lobe to the mediastinum and diaphragm. Both lungs are attached to the pericardial sac by easily separable, organizing fibrous adhesions. The pericardial sac has undergone obliteration by recently developed, congested fibrous adhesions throughout the left side and adjacent portions of the anterior and posterior surfaces corresponding, in general, to the left ventricle, while the remainder of the surface is free; this portion of the pericardial sac is filled with rather thick, yellow pus under tension. The free pericardial and epicardial surfaces are covered by a shaggy coat of fibrinopurulent exudate. The *thymus* is of normal size for the age of the individual and is somewhat congested and slightly edematous. The tissues of the upper mediastinum about the thymus are edematous and undergoing fibrosis.

The *heart* is normal. The *aorta* is delicate and elastic, but the intimal aspect presents numerous granular, slightly elevated, very small, pale yellow, atheromatous deposits. The *left lung* shows hypostatic congestion. The *right lung* shows a large area in the lower lobe, some 10 cm. in maximum diameter, throughout which the pleura is covered by a layer of pink granulation tissue, over which there is purulent exudate. At the diaphragmatic border anteriorly there is a firm, quadrangular, intensely hemorrhagic area surrounded by a pale yellow zone. This area, on section, penetrates into the parenchyma for 1.5 cm. and is intensely hemorrhagic and moderately soft. A second similar, but smaller, area is found in the lower lobe near the hilum. Thrombosed vessels are encountered in relation to both lesions, but the larger pulmonary branches are free of thrombi or emboli. The *spleen* is enlarged to 310 gm.; the pulp is soft, scrapes off easily, and is pale red. The *liver* seems slightly enlarged in relation to the other organs and weighs 1,650 gm. It shows slight central congestion of the lobules. The *kidneys* are normal, except for the presence in a medullary pyramid of a pale streak 1 cm. long and 0.2 cm. broad. The *gastrointestinal* tract is not remarkable, exception made of rather numerous hookworms attached to the mucosa of the small intestine and four or five whipworms in the appendix.

The superior vena cava is completely occupied by a soft, mixed thrombus

which, in its central portions, is semifluid. It extends along both innominate veins, the subclavian and axillary veins, both internal jugulars (as far, approximately, as the level of the larynx, beyond which point there is a propagated blood clot), and left external jugular. On the right side of the neck there is no one principal external jugular trunk; in place of this several small veins are found, none of which contains thrombotic material. In the left arm the thrombus has extended as far as the middle portion of the humerus and, beyond this point, there is a short propagated blood clot. On the right side, the dissection is not carried beyond the axillary vein but, since this trunk is completely filled with a thrombus, it is obvious that the process on this side must extend at least as far as the beginning portion of the brachial vein. The terminal portion of the azygos vein is likewise thrombosed. All the veins mentioned, as affected by the thrombotic process, show thickening of the walls which, in the superior vena cava, measures not less than 2 mm. in thickness. There is likewise broadening and edema of the adventitia of these veins and of the surrounding connective tissue.

The left ilium is extensively involved in an osteomyelitic process, which has caused widespread caries, with roughening of the surfaces, separation of the periosteum, and extension of the suppurative process across the musculature laterally to discharge through the three incisions in the left hip. The muscles in this area are very pale and edematous, having been partly replaced by fibrous tissue. The suppurative process has also extended inwardly, leading to the formation of a large abscess some 8 by 5 cm. in main diameters, on the inner aspect of the ilium behind the psoas muscle. This abscess contains an abundance of thick yellow pus. The lumbar vertebrae are entirely normal.

The pancreas, gall bladder, suprarenal glands, urinary bladder, prostate, seminal vesicles, testes, esophagus, stomach, large intestine, larynx, trachea, thyroid, and parathyroid glands are normal. The brain was not examined.

Microscopically, the *superior vena cava* showed replacement of the intima and subintima by a broad zone of granulation tissue, infiltrated by plasma cells and monocytes and covered by a broad layer of fibrinopurulent exudate. The muscle coat was thinner than normal. The nutrient arteries were surrounded by plasma cells. In some portions part of the wall had been replaced by granulation tissue. The adventitia was broadened by the formation of loose, edematous, fibrous tissue, in which there were numerous perivascular foci of infiltration with round cells. The *left innominate, external jugular,* and *brachial* veins showed the lumen to be variously occupied by thrombotic material and blood clot, which were being replaced by granulation tissue growing from the intima. There was edema of the wall of these veins and some perivascular round-celled infiltration, as well as congestion and edema in the adventitia.

In some sections of the *heart,* there was fibrous tissue obliterating the pericardial cavity, accompanied by the presence of small numbers of plasma

cells and monocytes surrounding blood vessels in the subepicardial tissue. The *lungs* showed numerous monocytes in many of the acini; also a hemorrhagic infarct that was surrounded by a broad zone of dense infiltration with polys and by a narrow area of bronchopneumonia. The *spleen* showed congestion of the pulp and a slight excess of polymorphonuclear leukocytes. The central portions of the *liver* lobules were congested, and hemosiderin-laden phagocytes were present in these areas. The cortical lipoids of the *suprarenal glands* had undergone depletion; there was serous atrophy of the periadrenal fat. In the *kidneys* the convoluted tubules were slightly dilated and contained coagulated protein, but this may have been partly due to postmortem autolysis. A small thrombus was found in a capsular vein. Sections of the testes revealed that spermatogenesis had not yet been established.

Anatomical diagnosis: Osteomyelitis of ilium, left; operation: incision and drainage of abscesses of left hip; bacteriemia with metastasis due to *Staphylococcus aureus hemolyticus;* infected infarcts of lung, L.L.L.; empyema, right; hydrothorax, left; thrombosis of superior vena cava, innominate, subclavian, and internal jugular veins, bilateral, and of vena azygos and external jugular and brachial veins, left; acute mediastinitis; acute pericarditis; acute splenic tumor; chronic passive congestion of liver; atheromatosis of aorta, early; fibrous pleural and pericardial adhesions; uncinariasis; trichuriasis.

DISCUSSION

The postmortem studies confirmed most of the writer's clinical deductions. It is clear, from the results of the autopsy, that the disease started as an osteomyelitis of the left ilium, and that this lesion constituted the primary focus of infection from which the process extended to the intrathoracic veins.

SUMMARY

Thrombosis of the superior vena cava is a rather rare condition, a total of 126 cases having been reported in the world literature. Its pathognomonic signs and symptoms are:

1. Edema and cyanosis of the face, neck, and upper extremities
   a) Aggravated when the patient assumes a horizontal position
   b) Relieved when erect
2. Venous hypertension in arms
3. Normal venous pressure of lower extremities
4. Development of deep collateral vessels
5. Development of subsequent varicosities on anterior thorax

The mortality rate in the reported cases is high, with death occurring in 75 percent of them in the acute phase.

Herein is reported an additional fatal case of thrombosis of the superior vena cava following osteomyelitis and septicemia, confirmed by postmortem examination, with a review of the main diagnostic criteria.

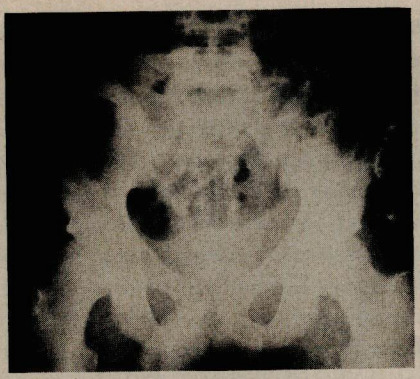

FIGURE 1

EXTENSIVE DESTRUCTION OF THE LEFT ILIUM BY OSTEOMYELITIC PROCESS

GRABADO 1

PROCESO OSTEOMIELÍTICO DEL HUESO ILÍACO IZQUIERDO

FIGURE 2

PNEUMOTHORAX OF THE LEFT SIDE OF CHEST WITH SLIGHT PLEURAL EFFUSION. MASSIVE PLEURAL EFFUSION OF RIGHT SIDE. THE STRAIGHT BORDER OF THE HEART IS SUGGESTIVE OF PERICARDIAL EFFUSION

GRABADO 2

NEUMOTÓRAX DEL LADO IZQUIERDO CON MODERADO DERRAME PLEURÍTICO. ENORME DERRAME PLEURÍTICO DEL LADO DERECHO. EL BORDE RECTO DEL CORAZÓN INDICA EL DERRAME PERICÁRDICO

FIGURE 3

PNEUMOTHORAX OF RIGHT SIDE WITH 30 PERCENT COLLAPSE OF RIGHT LUNG. A DENSE SHADOW OBSCURES THE RIGHT BASE AND ANOTHER DENSE SHADOW OBSCURES THE LEFT BASE. BILATERAL PLEURAL EFFUSION

GRABADO 3

NEUMOTÓRAX DEL LADO DERECHO CON EL PULMÓN COLAPSADO EN GRAN PARTE (30%). UNA SOMBRA DENSA OSCURECE SU BASE Y OTRA LA DEL IZQUIERDO. DERRAME PLEURÍTICO BILATERAL