Cardiovascular Manifestations in Argemone Mexicana Poisoning (Epidemic Dropsy)


The syndrome of epidemic dropsy is characterized by edema, its other chief manifestations being diarrhea, pyrexia, anemia, dyspnea, tachycardia, and elevated erythrocyte sedimentation rate. It was earlier believed to be a toxic syndrome caused by ingestion of poisonous bases formed in rice under certain conditions. That the disease is due to argemone poisoning has since been shown by the finding of argemone oil contamination in ingested mustard oil and experimental production of the disease in animals and human beings by administration of argemone oil. While outbreaks, some of them with a high incidence of mortality, have been reported mostly from India, the disease has also been reported from Fiji, Burma, and Africa. Mustard oil expressed from mustard seeds is used extensively in many states in India, particularly in Bengal and Assam, as a medium for cooking or frying vegetables and other food articles in place of butter, margarine, or other edible fats used in western countries.

Acton and Chopra first reported that pathologic changes in this disease are characterized by dilatation of the capillaries, and changes in the heart consist of increased vascularity, gross capillary dilatation, and edema separating the muscle fibers, which are otherwise normal. Shanks and De confirmed these changes and noted that both ventricles are equally involved. Though mention was made in the earlier literature of cardiac dilatation, murmurs, and failure, cardiovascular manifestations in epidemic dropsy were first stressed by Chopra and Basu and Chopra and Bose and have since been described in several reports.

There has, however, been no comprehensive study, and the findings have often not been substantiated by sufficient laboratory data. The purpose of this paper is to report a study of 11 cases of argemone poisoning to focus attention on this disease and to stress that several aspects of this disease are not clearly understood and require further investigation.

Material and Methods

Eleven patients who gave a history of ingestion of mustard or groundnut oil were selected for this study; in each instance the sample of consumed oil was proved on chemical examination to be contaminated with argemone oil. There were 8 male and 3 female patients, and their ages ranged from 11 to 40 years, with an average of 19 years. The duration of the symptoms at the time of admission varied between 6 days and 3 months, with an average of 39 days. Eight patients gave a history of edema in other members of the family. All patients were hospitalized until they were cured.

The following laboratory investigations were carried out: red blood cells, reticulocyte, and total differential leukocyte counts; hemoglobin; erythrocyte sedimentation rate (Westergren); osmotic fragility; blood sugar, urea, and free and esterified cholesterol; total proteins and albumin: globulin ratio; glucose tolerance test, serologic tests for syphilis, and liver function tests; examination of urine, stool, cerebrospinal fluid, and smear from sternal marrow; fluoroscopic examination and a posteroanterior roentgenogram of the chest; a 12-lead electrocardiogram; a ballistocardiogram with a 2-coil electromagnetic apparatus of the Doek type with a 20-mfd. condenser in circuit and attached in series with limb lead II of a direct-writing Burdick electrocardiograph; determination of the venous pressure by direct method, and of arm-to-lung and arm-to-tongue circulation times by ether and magnesium sulfate methods, respectively; liver biopsy with a Vim-Silverman needle; and cutaneous patch test with argemone oil.

Results

Edema was present in all the cases, being the initial symptom in 6 of them. It was confined to the lower extremities in 8 and was generalized in 3 cases. As a rule it either appeared or increased after exertion. It preceded dyspnea except in 2 patients who had fever and were confined to bed from the onset. Two patients were orthopneic and 4 had exertional dyspnea (table 1). Dyspnea was never an ini-
<table>
<thead>
<tr>
<th>Case no.</th>
<th>Age</th>
<th>Blood pressure (mm Hg)</th>
<th>Cardiac apex</th>
<th>Heart rate (beats per minute)</th>
<th>Vascular pattern</th>
<th>Arterial pressure (mm Hg)</th>
<th>Pulse pressure (mm Hg)</th>
<th>Jugular venous pattern</th>
<th>ECG</th>
<th>Roentgen</th>
<th>Blood test</th>
<th>Laboratory data on admission in eleven patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>11 M</td>
<td>120/70</td>
<td>8.2</td>
<td>10</td>
<td>4</td>
<td>8</td>
<td>4</td>
<td>5</td>
<td>8</td>
<td>+</td>
<td>++</td>
<td>+ LV, + RV, + S, + ST, + T</td>
</tr>
<tr>
<td>2</td>
<td>40 F</td>
<td>140/70</td>
<td>10.2</td>
<td>11</td>
<td>4</td>
<td>10</td>
<td>N</td>
<td>7</td>
<td>14</td>
<td>++</td>
<td>+ PA</td>
<td>+ LV, + RV, + S, + ST, + T</td>
</tr>
<tr>
<td>3</td>
<td>16 M</td>
<td>124/70</td>
<td>14.6</td>
<td>14</td>
<td>7</td>
<td>14</td>
<td>N</td>
<td>9</td>
<td>10</td>
<td>++</td>
<td>+ nil</td>
<td>+ LV, + RV, + S, + ST, + T</td>
</tr>
<tr>
<td>4</td>
<td>13 M</td>
<td>140/70</td>
<td>11</td>
<td>11</td>
<td>5</td>
<td>14</td>
<td>++</td>
<td>++</td>
<td>++</td>
<td>++</td>
<td>+ nil</td>
<td>+ LV, + RV, + S, + ST, + T</td>
</tr>
<tr>
<td>5</td>
<td>11 M</td>
<td>190/60</td>
<td>15.8</td>
<td>10</td>
<td>4</td>
<td>10</td>
<td>N</td>
<td>4</td>
<td>10</td>
<td>++</td>
<td>+ nil</td>
<td>+ LV, + RV, + S, + ST, + T</td>
</tr>
<tr>
<td>6</td>
<td>13 M</td>
<td>148/70</td>
<td>12</td>
<td>12</td>
<td>9</td>
<td>14</td>
<td>N</td>
<td>4</td>
<td>12</td>
<td>++</td>
<td>+ nil</td>
<td>+ LV, + RV, + S, + ST, + T</td>
</tr>
<tr>
<td>7</td>
<td>25 M</td>
<td>92</td>
<td>145/70</td>
<td>14.4</td>
<td>10</td>
<td>10</td>
<td>N</td>
<td>4</td>
<td>12</td>
<td>++</td>
<td>+ nil</td>
<td>+ LV, + RV, + S, + ST, + T</td>
</tr>
<tr>
<td>8</td>
<td>14 M</td>
<td>96</td>
<td>138/70</td>
<td>14.8</td>
<td>8</td>
<td>10</td>
<td>N</td>
<td>4</td>
<td>14</td>
<td>++</td>
<td>+ nil</td>
<td>+ LV, + RV, + S, + ST, + T</td>
</tr>
<tr>
<td>9</td>
<td>30 M</td>
<td>120/70</td>
<td>8.8</td>
<td>14</td>
<td>6</td>
<td>10</td>
<td>N</td>
<td>5</td>
<td>14</td>
<td>++</td>
<td>+ nil</td>
<td>+ LV, + RV, + S, + ST, + T</td>
</tr>
<tr>
<td>10</td>
<td>37 M</td>
<td>94</td>
<td>120/70</td>
<td>10.4</td>
<td>10</td>
<td>5</td>
<td>N</td>
<td>11</td>
<td>6</td>
<td>++</td>
<td>+ nil</td>
<td>+ LV, + RV, + S, + ST, + T</td>
</tr>
<tr>
<td>11</td>
<td>20 F</td>
<td>128/66</td>
<td>6.2</td>
<td>11</td>
<td>6</td>
<td>8</td>
<td>2</td>
<td>8</td>
<td>8</td>
<td>++</td>
<td>+ nil</td>
<td>+ LV, + RV, + S, + ST, + T</td>
</tr>
</tbody>
</table>

**Abbreviations:** Plus signs indicate presence and degree; LVH and RVH, left and right ventricular hypertension; A, normal.
tial symptom but appeared after an interval of from 10 days to 2 months after the initial symptom. It improved rapidly and always disappeared before edema. Eight patients had pyrexia, which was present at the onset in 5 of them. Other manifestations included cough in 4 cases, palpitation in 2, diarrhea in 2, headache, impotence, and amenorrhea in 1, anemia in 9, ascites in 3, and lymphadenopathy in 3 cases.

The cardiac rate was rapid, and the blood pressure was normal in all cases. The pulse pressure was more than 60 in 6 cases but in none was a water-hammer pulse noted. Heart sounds were altered in all the cases; there was an apical protodiastolic gallop in 5 cases with a systolic gallop in 1, accentuation of the mitral first sound in 7, and of the pulmonic second sound in 5 cases. Gallop rhythm disappeared within a few days, with slight improvement of the disease. Murmurs were heard in 7 cases, systolic pulmonary alone in 3, systolic mitral alone in 1, systolic mitral and pulmonary in 2, and systolic and presystolic mitral in 1 case.

Roentgen examination showed cardiac enlargement in 4 cases; the right ventricle was enlarged in all 4 and the left ventricle in 1. The enlargement was slight, the maximum cardiothoracic ratio being 50 per cent, and was confirmed when subsequent examination revealed reduction in size. There was prominence of vascular markings in all 11 cases, with pulmonary congestion in 5 of them. The liver was enlarged in 9 cases.

The venous pressure was elevated in 4 cases. The circulation time was considered prolonged in 1 case in which the hemoglobin level was 6.2 Gm. per 100 ml.

The electrocardiogram was abnormal in all cases, and abnormalities consisted of S-T changes in 9 cases, T changes in 6, pattern of left ventricular hypertrophy in 5, biventricular hypertrophy pattern in 2, and hypokalemic pattern in 1 case. The criteria of Sokolow and Lyon were used to determine patterns of ventricular hypertrophy. The ballistocardiogram was abnormal in 7 cases; the abnormalities consisted of tall R in 4 cases, short K in 3, slurred J-K in 5, and notched J in 1 case (figs. 1 and 2).

There was leukocytosis in 2 cases, but no eosinophilia was observed. Anemia was present in 9 cases; the bone marrow smear showed normoblastic reaction in all 9 and late normoblastic reactions in 3 of them. In the absence of reticulocytes or normoblasts in the peripheral blood the anemia appeared to be due to a maturation defect in the bone marrow. The sedimentation rate was raised in 10 cases. Cutaneous patch test to determine allergy to argemone oil was negative in all cases. The total blood proteins were diminished in 3 cases, and the albumin:globulin ratio was deranged in these 3 and in 4 other cases with increase in the globulin fraction. Blood cholesterol was elevated in 7 cases. Liver function tests were abnormal in 3 cases. The urine, the cerebrospinal fluid, the blood urea and sugar, and the glucose tolerance test were normal, and the serologic tests for syphilis were negative in all cases. The changes seen in liver biopsy included cloudy swelling, reticuloendothelial-cell hyperplasia, congestion of the central vein, distention of the sinusoids, and scanty hemorrhages.

The usual sequence of disappearance of the manifestations was dyspnea, gallop rhythm, pulmonary congestion, edema, tachycardia, enlarged liver, elevated sedimentation rate, anemia, and murmurs.

Discussion

Chopra and Bose noted that while cardiac involvement was the rule in epidemic dropsy, its severity was not uniform. They described 3 types of cardiovascular manifestations, the acute fulminating and fatal type resembling acute left ventricular failure, the subacute or the chronic type, and forms frustes with slight or no cardiac involvement. Congestive cardiac failure has been described by several writers, and acute cardiac failure and acute dilatation of the heart have been reported to be the cause of death in this disease.

In the present series, the onset was insidious and the progress slow in all the cases. The prominent manifestations were edema, dysp-
ARGEMONE MEXICANA POISONING

Figure 1
Electrocardiograms: Case 2 (top) shows S-T and T changes. Case 3 (middle) shows hypokalemic pattern. Case 9 (bottom) shows biventricular hypertrophy pattern 0.5 mm. voltage in $V_2$ to $V_6$.

neumonia, pulmonary congestion, and hepatic enlargement. Edema was not related to the serum protein level and the albumin:globulin ratio or to the hemoglobin level. It was not associated with elevated venous pressure in 7 cases, including 2 of the 3 cases with generalized anasarca. The venous pressure was normal in 6 of the 9 cases with hepatic enlarge-
appeared unlikely that left heart failure was responsible for pulmonary congestion. Capillary dilatation has been reported to be the characteristic pathologic change in epidemic dropsy, and it is conceivable that it plays a major role in the causation of edema, hepatomegaly, and pulmonary congestion.

Chopra and Bose noted that dyspnea may occur early in epidemic dropsy while there are no gross changes in the cardiovascular or respiratory systems. They suggested 4 possible causes of dyspnea: (1) toxins acting on the respiratory center; (2) turgid lung capillaries encroaching upon the alveolar spaces; (3) anemia of the respiratory center; and (4) cardiac failure. In the present series in cases 2 and 3 with orthopnea the roentgen examination showed bilateral pleural effusion in one and severe pulmonary congestion in the other. In the former the circulation time was considered prolonged, the venous pressure was elevated, and the dyspnea could be attributed to cardiac failure, whereas in the latter it could be attributed to pulmonary congestion. It is conceivable that in these 2 cases and in case 11 anemia may have been partly responsible for dyspnea because the hemoglobin was less than 9 Gm. per 100 ml. There was, however, no definite relation between dyspnea and pulmonary congestion because in none of the 4 patients with exertional dyspnea was there roentgen evidence of congestion, and in 4 of the 5 patients with congestion there was no dyspnea. As stated earlier, heart failure was considered unlikely in 5 of the 6 cases with dyspnea. The mechanism of dyspnea was thus not clearly understood.

In this series cardiac enlargement was minimal and was mostly right ventricular in type, while in other reports left ventricular enlargement has been described as common.15,18 Except in one case with hypokalemia, the electrocardiographic and the ballistocardiographic changes were also minor. The former, apart from the nonspecific S-T and T changes, were characterized by a hypertrophy pattern, predominantly of the left ventricular type. Shortening of the P-R interval was not found in any case on comparison with the

Figure 2

ment, including 2 of the 3 cases with considerable enlargement. The circulation time was normal in 10 cases. Edema and hepatic enlargement therefore could not be attributed to congestive failure in most of the cases. Moderate to severe pulmonary congestion was demonstrated roentgenologically in 5 cases. It did not appear to have definite relation to cardiac enlargement; in cases 2 and 5, with severe congestion, there was no demonstrable cardiac enlargement. It is realized that cardiac enlargement may not be demonstrable in some cases of left ventricular failure. Congestion was, however, not associated with a prolonged circulation time in any of the 5 cases, and in 4 of them there was no dyspnea. It therefore
tracings taken after cure. The short K wave in the ballistocardiogram was not related to lowered peripheral resistance; there was no lowering of the diastolic pressure in any case with this abnormality, and this abnormality was not present in the only case with lowered diastolic pressure. The abnormalities may be due to inefficient myocardial ejection.25

It was thus seen that while tachycardia, alterations in the heart sounds, cardiac enlargement, or the electrocardiographic and ballistocardiographic changes indicated myocardial involvement in every case in this series, the latter appeared to be of a minor nature. The findings may be attributed to a nonspecific type of myocarditis, a term which includes not only inflammatory and specific lesions but degenerative and other lesions encountered in a variety of other conditions.26

In some of our cases dyspnea or orthopnea and clinical and roentgenologic evidence of severe pulmonary congestion simulated left ventricular failure, and these findings associated with edema, and enlarged tender liver simulated congestive cardiac failure. It was therefore of interest that the presence of cardiac failure was considered unequivocal in one case only. In others it was considered unlikely, or at least it was not a dominant feature. Congestive cardiac failure occurs only occasionally as a result of myocarditis alone, except in rheumatic fever.27

It is thought that the clinical picture in argemone poisoning may be misleading and heart failure may be diagnosed incorrectly in the absence of laboratory data, and that congestive failure is unlikely to be the cause of death in argemone poisoning. The characteristic pathologic change in this disease is vascular dilatation, and it seems more likely that the fatal termination, which occurs with extreme rapidity, is due to acute circulatory failure or shock as a result of severe vasodilatation with consequent pooling of the blood in the small vessels. It must also be emphasized that the clinical picture in argemone poisoning may lead to an erroneous diagnosis of rheumatic heart disease (case 1) and even miliary tuberculosis (case 2).

The chemical nature of the toxic substance in argemone oil (derived from the seeds of the plant argemone mexicana) is uncertain. Mukherjee and co-workers28 isolated a crystalline free base C_{19}H_{15}O_{4}N from the oil and suggested that this represented the toxic fraction. Sarkar29 found that the oil contained at least 2 toxic alkaloids, dihydrosanguinarine and sanguinarine, forming 87 and 5 per cent, respectively, of the total alkaloids, the latter being more toxic. Mitra30 however observed that even that fraction of the oil from which the above 2 alkaloids were removed was toxic. It has also been observed that toxicity of different batches of argemone oil varies,31 and 2 different toxic alkaloids, argemone (protopine) and berberine, have been found in the African plant.11

Treatment of argemone poisoning has also remained a problem. Chopra and Basu15 found digitalis ineffective and sometimes even harmful, while Chaudhuri and Chakravarty19 suggested that it should be tried in all cases with heart failure. The latter authors noted dramatic response, even of heart failure, to Phenergan. Administration of vitamins C, D, and P, for protection and restoration of damaged capillaries, and of a diet rich in protein and poor in fat with glucose, and insulin and vitamin B complex, for protection of the liver, have been recommended.11 In one instance dramatic improvement was noted after cortisone therapy.32 In animal experiments, however, rutin, Phenergan, ephedrine, BAL, cortisol, and BAL with cortisol were found to possess no effect in preventing the toxic effects of argemone oil.33 In this study digitalis, administered in cases with orthopnea or severe pulmonary congestion, and antihistaminics were found to be of no value. Anti-anemic drugs including iron preparations, liver extract, folic acid, and cyanocobalamine were found ineffective for some time, probably because of depression of the bone marrow. For the present, early recognition of the condition and immediate stoppage of the incriminating oil, bed rest, and blood transfusion in cases with anemia, remain the main principles of treatment in chronic cases. In
cases with acute circulatory failure (not encountered by us) measures to combat shock should prove beneficial.

Case Reports

Case 1

An 11-year-old boy was admitted on September 19, 1957, complaining of cough for 2 months, intermittent fever and edema of the feet for 6 weeks, oliguria for 15 days, and edema of the face for 3 days. There was no history of diarrhea and dyspnea.

Examination revealed generalized anasarca, slight ascites, cardiac rate of 120 per minute, blood pressure 135/70, and liver enlarged 2½ inches below the costal margin and tender. There was a pre-systolic murmur at the mitral area, and the pulmonary second sound was accentuated.

Laboratory examination of the blood revealed hemoglobin, 8 Gm. per 100 ml.; erythrocyte sedimentation rate, 42 mm. per hr.; cholesterol, 350 mg. per cent; and total proteins 4.38 Gm. per cent, with an albumin:globulin ratio of 1.1:1. The venous pressure was 8.2 cm. of saline, and the arm-to-tongue and the arm-to-lung circulation times were 10 and 4 seconds, respectively. A roentgenogram of the chest (fig. 3) showed prominent vascular shadows, moderate pulmonary congestion, and slight enlargement of the cardiac shadow with straight left border. Fluoroscopy showed right ventricular enlargement. The electrocardiogram showed left ventricular hypertrophy and S-T and T changes, and the ballistocardiogram was normal.

On October 2 the cardiac rate had decreased to 97 per minute, the edema had diminished, and the presystolic murmur had disappeared. Roentgenogram on October 5 showed absence of pulmonary congestion, while the cardiac shadow was still enlarged. On October 20, edema had disappeared and the blood pressure had decreased to 110/60. On November 16, the pulse rate was 72 per minute, the systolic murmur had disappeared, the erythrocyte sedimentation rate had decreased to 17 mm. per hour, the hemoglobin was 12.5 Gm. per 100 ml., and the liver had receded below the costal margin. Roentgenogram on November 27 showed a normal-sized heart (fig. 3).

Comment

At the time of admission a diagnosis of rheumatic mitral stenosis with congestive heart failure was made in this case because of the elevated erythrocyte sedimentation rate, mitral presystolic murmur, pulmonary congestion, and cardiac configuration. The presystolic murmur was not considered to be due to anemia as it is not encountered when the hemoglobin level is 8 Gm. per 100 ml. Absence of dyspnea and normal venous pressure and circulation time were, however, evidences against congestive failure. History of ingestion of groundnut oil and of edema in other family members, and disappearance of the murmur after improvement established the diagnosis of argonene poisoning.

Case 2

A 40-year-old housewife was admitted on October 12, 1957, complaining of irregular pyrexia for 2 months, weakness, palpitation, and dyspnea on exertion for 1 month, edema of the feet for 15 days, orthopnea for 8 days, and dry cough for 4 days.

Examination revealed a moderately anemic woman in severe distress with orthopnea, edema of

Circulation, Volume XXI, June 1960
the feet and face, slight enlargement of cervical and axillary lymph nodes, temperature of 100° F., cardiac rate of 140, respiratory rate of 38 per minute, a blood pressure of 120/78, a tender liver enlarged 3 inches below the costal margin, and slight ascites. There was no cyanosis or engorgement of the neck veins. The cardiac borders appeared to be enlarged clinically, the mitral first sound and the pulmonic second sound were accentuated, and there was a protodiastolic apical gallop. Rales were heard over both lungs.

Laboratory examination revealed hemoglobin, 8.4 Gm. per 100 ml.; erythrocyte sedimentation rate, 43 mm. per hour; blood cholesterol, 290 mg. per cent; blood proteins, 5.4 Gm. per cent, with an albumin-globulin ratio of 1.8:1, venous pressure, 10.2 cm. of normal saline; and arm-to-tongue and arm-to-lung circulation times, 11 and 6 seconds, respectively. A roentgenogram of the chest (fig. 4) showed a normal-sized heart, prominent pulmonary artery and vascular shadows, and extensive mottling over both the lung fields. The electrocardiogram showed S-T and T changes, and the ballistocardiogram showed slurred J-K segment. The liver biopsy showed congestion of the central veins, cloudy swelling of the cells, and reticuloendothelial-cell hyperplasia. Biopsy of an axillary lymph node showed reactive hyperplasia.

The patient was given 1 mg. of strophanthin intravenously followed by Digoxin, 1 tablet every 8 hours orally, but with no significant influence on dyspnea and heart rate. The condition gradually improved and the temperature returned to normal on October 17; the heart rate was still 116 per minute. Digoxin was then discontinued. By October 21 there was no orthopnea, edema, or ascites, and the patient had lost 8 pounds of weight. The gallop rhythm was not audible, but a systolic murmur was heard at the mitral and the pulmonic areas. Roentgenogram of the chest showed some pulmonary congestion on November 7 and was normal on November 25 (fig. 4). The hemoglobin, the erythrocyte sedimentation rate, and the liver returned to normal only after 3½ months of treatment.

Comment

In this case, at the time of admission, orthopnea, severe pulmonary congestion, enlarged tender liver and the electrocardiographic changes suggested a diagnosis of myocarditis with severe cardiac failure. The normal-sized heart and normal venous pressure and circulation times, however, were evidences against failure. Miliary tuberculosis with secondary anemia and edema due to hypoalbuminemia was also considered because of irregular pyrexia, lymphadenopathy, and the chest roentgenogram. The blood examination, however, revealed normal proteins, the lymph node biopsy did not show tuberculous lesion, and the temperature settled to normal 5 days after admission without antibiotic therapy. History of use of mustard oil, which was proved to contain argemone oil, and of edema in other members of the family established the diagnosis.

Case 3

A 16-year-old girl was admitted on November 9, 1957, complaining of irregular pyrexia for 3 months, anorexia, cough, and diarrhea with 6 to 8 stools per day for 2 months, dyspnea on exertion for 1 month, edema of the feet for 15 days, and orthopnea for 8 days. She had amenorrhea for 3 months.

Examination revealed a well-built and poorly
nourished girl with orthopnea, edema of the feet, severe anemia, cardiac rate 124 and respirations 36 per minute, temperature 100.4 F., blood pressure 106/54, liver enlarged 3 inches below the costal margin, and slight ascites. The neck veins were slightly engorged. There were systolic and protodiastolic gallop sounds at the mitral area and signs of moderate pleural effusion on both sides.

Laboratory examination revealed hemoglobin, 6.2 Gm. per 100 ml.; erythrocyte sedimentation rate, 11 mm. per hour; total leucocyte count 16,200 per mm.\(^3\) with a normal differential count; proteins, 3.61 Gm. per cent, with an albumin : globulin ratio of 1.1:1; cholesterol, 235 mg. per cent; venous pressure, 14.6 cm.; and arm-to-tongue and arm-to-lung circulation times, 14 and 7 seconds, respectively. Roentgenogram of the chest (fig. 5) showed bilateral pleural effusion. An electrocardiogram showed changes consistent with a hypokalemic pattern, and a ballistocardiogram showed grade-I respiratory variation, notched J wave, slurred and notched J-K segment, and short K wave. The serum potassium on the same day was 3.2 mEq. per liter. The liver biopsy showed areas of parenchymal degeneration and few hemorrhages. On paracentesis, 870 ml. of clear fluid could be aspirated from the left pleural space and 900 ml. from the right, with relief of the orthopnea. The fluid was straw colored, with a protein content of 3.5 Gm. per cent and cell count of 2,200 per mm.\(^3\) with 95 per cent lymphocytes.

Potassium chloride was given orally in doses of 1 Gm. every 6 hours. There was considerable improvement of the electrocardiographic pattern on November 17, when potassium chloride was discontinued. The pyrexia and diarrhea subsided, and the gallop rhythm disappeared within a week. On November 17 there was no edema. On November 23 the chest roentgenogram was normal. She was discharged at request on December 5, 1957, although the hemoglobin had risen only to 10 Gm. per 100 ml.

Comment

Severe anemia with congestive heart failure due to chronic diarrhea was suspected in this case at the time of admission. Slightly elevated venous pressure, and arm-to-tongue circulation time of 14 seconds in the presence of severe anemia, were indicative of congestive failure. The high cell count in the pleural fluid was suggestive of increased capillary permeability and against a transudate due to congestive failure or hypoproteinemia. Argemone poisoning was suspected when a history of edema in other members of the family was obtained and was proved when the mustard oil consumed by the patient was found positive for argemone oil. The hypokalemia in this case was apparently due to diarrhea of 2 months' duration.

Case 4

A 13-year-old boy was admitted on January 16, 1958, complaining of edema of the feet on exertion for 6 days. There was no history of pyrexia, diarrhea, or dyspnea.

Examination revealed a well-built and well-nourished boy with + edema of the feet, cardiac rate 110, and blood pressure 140/70. The heart boundary was slightly enlarged, and the mitral first sound was accentuated.

Examination of blood revealed hemoglobin, 14 Gm. per 100 ml. and sedimentation rate, 44 mm. per hour. The venous pressure was 11 cm. and the arm-to-tongue and arm-to-lung circulation times were 11 and 5 seconds, respectively. A roentgen examination (fig. 6) showed prominent
vascular shadows, pulmonary congestion more marked on the left side, and enlargement of both the ventricles. The electrocardiogram revealed left ventricular hypertrophy pattern and S-T and T changes. The ballistocardiogram was normal.

Comment

While in case 2 there were severe pulmonary congestion, orthopnea, and normal-sized heart, in this case there were cardiac enlargement and moderate pulmonary congestion, but no dyspnea.

Case 5

An 11-year-old boy was admitted on February 13, 1958, complaining of headache for 25 days, and edema of the feet and face for 15 days. There was no history of diarrhea, fever, or dyspnea.

Examination revealed a well-built, well-nourished boy with 1+ edema of the feet, cardiac rate 130, blood pressure 110/60, and just palpable liver. Mitral first sound was accentuated and there was a soft systolic murmur at the pulmonary area. Rales were heard over both lungs.

Laboratory examination revealed hemoglobin, 10.4 Gm. per 100 ml.; erythrocyte sedimentation rate 70 mm. per hour; venous pressure, 15.8 cm.; and arm-to-tongue and arm-to-lung circulation times 10 and 4 seconds, respectively. Roentgen examination (fig. 7) showed normal-sized heart, prominent vascular shadows, and severe pulmonary congestion. The electrocardiogram showed left ventricular hypertrophy and S-T changes, and the ballistocardiogram showed tall H waves.

Comment

In this patient though the venous pressure was slightly raised and there was severe pulmonary congestion, the circulation time and the heart size were normal, and, despite severe pulmonary congestion, this patient denied a history of dyspnea. The presence of cardiac failure was therefore considered unlikely.

Summary

Cardiovascular manifestations have been studied in 11 patients with argemone oil poisoning.

Tachycardia, alterations in the heart sounds, cardiac enlargement, or electrocardiographic and ballistocardiographic changes indicated myocardial involvement in every case. The latter was suggestive of a nonspecific type of myocarditis and was considered to be of a minor nature. The prominent manifestations included dyspnea, edema, hepatic enlargement, and pulmonary congestion. The last 3 were probably due to capillary dilatation, whereas the cause of dyspnea was uncertain.

These manifestations suggested the presence of cardiac failure, which, however, was considered unlikely except in 1 case.

Acknowledgment

Dr. L. R. Sarin, Superintendent, Sawai Man Singh Hospital, kindly permitted the publication of this report.

Summario in Interlingua

Le manifestiones cardiovascular esseva studiata in 11 patieientes con invenienteamento per olo de Argemone Mexicana.

In omne le casos, alteraciones del sonos cardiaque, tachycardia, allargamento del corde, o signos electroe ballistocardiographic reflecteva un affezion mycardiale. Isto pareva sugerir un typo nonspecific de myocarditis e esseva considerate como de natura minor. Le plus prominente manifestaciones includeva dyspnea, edema, allargamento hepatic, e congestione pulmonar. Le ultime 3 in ista lista de manifestaciones esseva probabilmente le consequentia de dilatation capillar. Le causa del dyspnea remaneva obscur.

Le complexo de manifestaciones sugervava le presente de decompensation cardiaque, sed isto esseva considerate como pauclo probable, excepte in un caso.

References


Cardiovascular Manifestations in Argemone Mexicana Poisoning (Epidemic Dropsy)
L. M. SANGHVI, S. N. MISRA and T. K. BOSE

Circulation. 1960;21:1096-1106
doi: 10.1161/01.CIR.21.6.1096

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1960 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/21/6/1096

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org/subscriptions/