Serum Glutamic Oxaloacetic Transaminase in Dissecting Aneurysm of the Thoracic Aorta

By John B. Tredway, M.D., and Edward E. Kemble, M.D.

The behavior of serum glutamic oxaloacetic transaminase level in cases of dissecting aneurysm of the thoracic aorta is reported. Certain clinical and laboratory observations suggest that intrapericardial hemorrhage is the responsible factor when the titer of serum glutamic oxaloacetic transaminase is elevated.

For obvious reasons, separation of the entities of myocardial infarction and dissecting aneurysm of the thoracic aorta has often presented a difficult and perplexing problem in clinical medicine. With the advent of serum glutamic oxaloacetic transaminase (SGOT) determination as a diagnostic measure in the diagnosis of myocardial infarction, it has been suggested1 that this procedure may be of help in differentiating the 2 conditions.

Review of the medical literature to date yields mention of SGOT activity in only 1 case of dissecting aneurysm of the aorta2 with a single normal value of 18 units. It is the purpose of this report to present 3 cases of dissecting aneurysm of the thoracic aorta, 2 of which showed elevation of serum glutamic oxaloacetic transaminase activity during their clinical course and a third that did not, along with certain observations as to the possible mechanism of the elevated SGOT activity. Determination of SGOT activity was performed by the method of Cabaud, Leeper, and Wroblewski.3

Case Reports

Case 1. A 71-year-old white physician was admitted to the hospital on May 20, 1956. He had noted severe pain in the substernal region, in the throat, and over the sacral area on the day before admission after treating some people who were badly hurt in an automobile accident. He did some lifting at the time, but returned to his office and worked until 10:00 p.m. During the night the chest pain became sufficiently severe to require meperidine (Demerol) and was made worse by inspiration. There was no sweating or vomiting and the pain persisted until the next day, when he was admitted to the hospital.

He stated that he had experienced some exertional dyspnea for several years and had had systolic hypertension of approximately 200 mm. Hg for the past 15 years.

On physical examination the blood pressure was 170/90 and the pulse was 90 and regular. The heart tones were of fair quality and there were no murmurs. A few fine rales were heard at the right base and some tenderness was noted in the epigastrium on palpation. No abdominal masses were noted. The hemoglobin was 15.3 Gm. per cent, the leukocyte count 14,400, with 57 per cent polymorphonuclear neutrophils, the hematocrit value was 43 per cent and the sedimentation rate was 38 mm. per hour (Westergren). The blood sugar was 102 mg. per cent, the blood urea nitrogen was 18 mg. per cent, and the prothrombin time was 57 per cent of normal. Serum glutamic oxaloacetic transaminase determinations were 115 units on May 20 (approximately 18 hours after onset of pain), 82 units on May 22, 40 units on May 23, and 17 units on May 24. An x-ray of the chest on May 21 showed the heart to be moderately enlarged, with some prominence of the left ventricle. There was no evidence of a double contour of the aorta and no sign of fluid in the pleural cavity. An electrocardiogram on May 21 showed elevated S-T segments throughout the limb and precordial leads, suggestive of acute pericarditis. A second electrocardiogram 4 days later showed that the S-T segments had, for the most part, returned to the isoelectric level.

The patient was not given anticoagulants because the electrocardiographic changes suggested pericarditis. An inconstant murmur was heard over the precordium and at one time a scratchy pericardial friction rub was detected. The patient was comfortable, had no recurrence of precordial pain, and seemed to be convalescing well, when he suddenly died on May 28.

At autopsy the pericardial sac was greatly distended with blood. The pleural spaces were not remarkable and the remainder of the thoracic

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viscera were normal. Some dilatation of the aorta was noted, and a rent was found in its intimal surface approximately 2 cm. above the orifice of the aortic valve. This was associated with a split in the aortic wall, which extended up to the arch. There was also a small rent in the adventitia about 1 cm. above the intimal defect, and around it was a considerable amount of blue discoloration. The adventitial tear was located below the pericardial reflection, so that it was the source of the hemopericardium. The coronary ostia were patent and free from dissection and the coronary arteries showed severe and extensive arteriosclerotic thickening. The remainder of the aorta revealed widespread arteriosclerosis, which was most conspicuous in the abdominal portion. The liver weighed 1,300 Gm. and showed a smooth capsule and passive congestion. Microscopic examination of the myocardium failed to reveal any evidence of infarction; although the lumina of the coronary arteries were narrowed, no thrombus formation within the vessels was noted. Microscopic examination of the liver revealed only terminal passive congestion.

Case 2. A 67-year-old white woman was admitted to the hospital January 2, 1957, because of precordial pain. Crushing, nonradiating anterior chest pain began suddenly about 1 hour prior to admission to the hospital. There was no previous history of any significant illness or disability. Examination on admission showed a semistuporous, desperately ill, cyanotic woman with a blood pressure of 90/60, apical heart rate of 120, distant heart sounds, and no murmurs or friction rub. The blood sugar on admission was 304 mg. per cent and 4 hours later it was 388 mg. per cent. The blood urea nitrogen was 35 mg. per cent, creatinine was 2.2 mg. per cent, carbon dioxide combining power was 14.3 mEq. per L., and the prothrombin time was 64 per cent of normal. A single SGOT determination 8 hours after onset of pain was 135 units. An electrocardiogram taken several hours after admission showed normal sinus rhythm with a rate of 150, the pattern of left ventricular hypertrophy, and depressed S-T segments in leads I, II, aV_{6}, and in the lateral precordial leads. The electrocardiographic changes were interpreted as indicating left ventricular hypertrophy and "strain," with S-T depression suggestive of acute coronary insufficiency.

On admission the patient was placed in an oxygen tent and was given meperidine (Demerol) for pain. Despite relatively large amounts of norepinephrine by intravenous infusion the blood pressure remained at a relatively low level, and she steadily weakened, and died 32 hours after admission.

Autopsy disclosed a moderately distended ascending aorta with an extremely soft wall. A 3.0-cm., irregular, longitudinal tear was present on the external surface 2.0 cm. above the aortic ring. A 2.0-cm. horizontal tear was noted on the intimal surface 5 cm. above the aortic ring, with dissection of the wall from the arch to the aortic ring. The proximal ends of the innominate, left common carotid, and left subclavian arteries were involved in the dissection. Inferiorly, the dissection extended through the aortic ring into the pericardium, which was filled with blood and blood clots. The interatrial septum was moderately distended and on section was found to be dissected and filled with blood clots. The dissection of the interatrial septum extended for a distance of 3.0 cm. and microscopically the dissection appeared to be fresh. The erythrocytes within the muscle were intact and there was no evidence of myocardial necrosis in tissue stained by hematoxylin and eosin and no leukocytic infiltration had occurred.

Aside from moderate thickening (2.0 cm.) of the left ventricular wall, examination of the heart was otherwise not remarkable. The coronary arteries were patent throughout, and their walls were free from dissection. Microscopic examination of numerous sections of the left ventricle showed no evidence of myocardial infarction. Each pleural cavity contained about 200 ml. of hemorrhagic fluid and areas of hemorrhage were noted in both hilar regions. Microscopic examination of the liver showed acute passive congestion with actual necrosis at the lobular centers, which was interpreted as occurring within a few hours before death. No other significant abnormalities were noted on gross or microscopic examination.

Case 3. A 53-year-old white woman was admitted to the hospital on June 9, 1957, because of severe upper and middorsal pain, radiating to the costal margins anteriorly and into the left arm. The pain began suddenly 4 hours prior to admission and was accompanied by sweating and vomiting. There was a history of a vague type of anterior chest pain over the preceding 2 years, which was precipitated by exertion and relieved by rest. No history of hypertension or of diabetes mellitus was obtained, but a chest x-ray done 1 year previously showed dilatation of the arch of the aorta without calcification of the aortic wall.

Physical examination showed an agitated, acutely ill woman, complaining bitterly of pain in the interscapular area. Good pulsations were noted in both common carotid and femoral arteries, and the blood pressure was 140/90 in both arms. General physical examination was not remarkable, save for evidence of slight enlargement of the heart to the left and a grade II apical systolic murmur.
DISSECTING ANEURYSM OF THE THORACIC AORTA

During the patient's hospital course of 10 days she had intermittent bouts of severe mid dorsal pain, radiating to the costal margin anteriorly. Serial chest x-rays showed a rapid increase in size of the arch of the aorta, but peripheral arterial pulsations remained normal. The routine laboratory data were not remarkable. Serial electrocardiograms remained within normal limits. On June 19 the patient was transferred to the Georgetown University Hospital, where operation was performed on June 20 by Dr. Charles Huf nagle. A dissecting aneurysm of the thoracic aorta was found with its point of origin just distal to the origin of the left subclavian artery. Three inches of tortuous aorta, including the site of origin of the dissection, were resected, the clot was removed from the aortic wall inferiorly to the diaphragm, and the area of dissection was obliterated by sutures.

The SGOT activity was 21 units on June 9 and on June 10 and 8 units on June 19. Her post operative course was very satisfactory except for paralysis of the recurrent laryngeal nerve and thrombophlebitis of the right leg, which was treated successfully with Dicumarol without peripheral hemorrhage or increase in size of the thoracic aorta.

DISCUSSION

A summary of the SGOT determinations in the 3 cases is given in table 1.

Review of the medical literature indicates that abnormally high serum GOT activity has been noted in the following conditions: acute myocardial infarction;2,4-6,10 myocarditis, rheumatic;6 and granulomatous (Fiedler's);9 cardiotoxic arrhythmias with ventricular rate of 160 and over;6 acute hepatitis, hepatic cirrhosis, and obstructive jaundice;6 pancreatitis (50 per cent of cases), with jaundice (75 per cent);6 crush injuries involving muscle;9 embolus to extremity;6 surgical trauma;7,9 pulmonary embolism (usually late in the course of the disease and associated with icterus);5,9 hemolytic crisis;9 dermatomyositis;8,13 experimental ligation of renal, mesenteric, splenic, and pulmonary arteries;6 and disseminated lupus erythematosus.12

While no mention is made of SGOT determinations in the presence of pericardial tamponade, it is of interest in connection with cases 1 and 2 that pericarditis is generally associated with normal GOT activity in the

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<th>Case no.</th>
<th>Day after onset of dissection</th>
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<tr>
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Table 1.—SGOT Values following Onset of Dissecting Aneurysm of the Thoracic Aorta, in Units

serum.2,5,10 However, in certain series, "false positives" have been reported, the incidence being from 0 to 75 per cent.6 Experimental pericarditis, induced in dogs by insufflation of talc into the pericardial cavity, has been shown to produce elevation of serum GOT activity, only if associated with subepicardial myocardial necrosis.12

Cases 1 and 2 showed no evidence of any of the above states known to be associated with elevated GOT activity, but the possibility is suggested that GOT may have been liberated in sufficient quantity from the hemolyzed dissecting blood to give rise to an elevated level in the serum. Since whole blood hemolysates have been shown to have a GOT activity 10 times that of serum,11 it is apparent that 1 ml of whole blood could release 300 units of GOT. Hemolysis of 1,000 ml of blood would be required to produce the amount of GOT contained in 1 Gm. of heart muscle, and the infarction of this small amount of cardiac muscle could scarcely be expected to produce serum transaminase levels of the magnitude recorded in cases 1 and 2. In view of the relatively low concentration of GOT in the human erythrocyte, it is evident that if hemolysis were the only factor accounting for the observed rise in GOT titer in cases 1 and 2, gross hemolysis of the serum should have been noted, but none was present. Moreover, our case 3 and the one reported by Kattus et al.2 had normal values, a finding that would not be expected were hemolysis the explanation of the elevated GOT.

Another explanation is that the elevation of GOT in cases 1 and 2 is the result of presence of blood in the pericardial sac, which
was not involved in case 3. While no significant morphologic changes were noted in cardiac muscle, case 2 showed dissection into the interatrial septum and it is entirely possible that some muscle damage, without evident morphologic change, may have resulted from the intrapericardial hemorrhage with release of GOT. It may be concluded that the mechanism of the elevation of GOT in these 2 cases is currently not well understood, but it is quite clear that SGOT determination is not always a reliable method in the separation of the clinical entities of myocardial infarction and dissecting aortic aneurysm.

SUMMARY

Three cases of dissecting aneurysm of the thoracic aorta are presented, 2 showing distinct elevation in serum glutamic oxaloacetic transaminase (SGOT) levels. Elevation of SGOT level was associated with extension of the dissection into the pericardial sac. Possible explanations for the rise in SGOT level in dissecting aortic aneurysm are discussed.

SUMMARIO IN INTERLINGUA

Es presenteate 3 casos de aneurysmo dissecante del aorta thoracique. Duo exhibiva un elevation distincte del nivellos de transaminase glutamic-oxaloacetic del sero (TGOS). Le elevation del nivellos de TGOS eseva associate con le extension del dissection a in le sacco pericardial. Explicaciones possibile del augmento del nivellos de TGOS in dissecante aneurysmos aortic es discutite.

ACKNOWLEDGMENT

The authors wish to thank Drs. James D. Weaver and Thomas M. Gocke for permission to include their cases, John A. Fust for his advice and assistance in presenting the pathologic data and John J. Sampson of San Francisco, Calif., for his encouragement and help.

REFERENCES

4. Ladue, J. S., and Wroblewski, F.: Clinical significance of SGO-Transaminase in heart and liver disease, Sloan-Kettering Division of Cornell University Medical College and Memorial Center, New York City.
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Circulation. 1958;18:37-40
doi: 10.1161/01.CIR.18.1.37

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