This trend of thought, coupled with the obvious resemblance of the lesions to the bulbous, pungent root of the monocotyledonous garlic plant, suggested the solution. For JAMA readers, the MEDLARS computers at the National Library of Medicine, and posteriorly, the name “garlic-clove” fibroma is proposed.

Summary
Previously unreported, a lesion was observed in three patients. A benign painless excrescence of the nail bed, fibromatous, and resembling a peeled garlic clove, the lesion has been described, illustrated, and named the garlic-clove fibroma.

Death Due to Migration of the Ball From an Aortic-Valve Prosthesis

Arthur Krosnick, MD

IN 1961, Starr and Edwards reported their first clinical experience with a ball-valve prosthesis as a replacement for the mitral valve.1 In 1963, these authors and their colleagues described their initial experience with a similar device for complete replacement of the aortic valve.2 The literature on the subject suggests that hundreds of diseased aortic valves have been replaced by the Starr-Edwards ball-valve aortic prosthesis in the past 20 months. In a number of instances multiple valves have been replaced.3 4 The authors have uniformly been interested in the late follow-up results, but none to date has reported migration of the ball from its metal cage. This report will describe such a complication.

Report of a Case

A 56-year-old male truck driver was admitted to the Lower Bucks County Hospital, Bristol, Pa, with acute failure of the left ventricle. He had the harsh systolic aortic murmur and thrill and absent aortic second sound characteristic of aortic stenosis. His response to medical therapy was good and he left the hospital 12 days later to convalesce at home.

The patient was later admitted to the Deborah Hospital, Browns Mills, NJ, for evaluation for cardiac surgery. Open-heart surgery was performed on Dec 5, 1963, at which time the intensely calcified aortic valve was resected and a No 9 ball-valve prosthesis (Starr-Edwards) inserted. On the third postoperative day, the patient developed paroxysmal atrial fibrillation associated with an acute anterior myocardial infarction. The patient was treated with anticoagulants and the remainder of the hospital course was uncomplicated until his discharge on Jan 18, 1964. Convalescence went very well except for a transient episode of painless hematuria, which necessitated temporary discontinuation of anticoagulant therapy.

The patient was reevaluated at the Deborah Hospital on April 21, 1964. The heart sounds were characteristic, i.e., opening and closing clicks of the prosthesis followed by a systolic ejection murmur. The electrocardiogram and chest x-ray were unchanged. Anticoagulation was reinstituted and maintained at therapeutic levels.

The patient made an excellent clinical response, complaining only of mild incisal discomfort and rare episodes of angina and paresthesias in the hands and feet. In July 1964, he obtained a full-time job as a guard, which he was able to maintain without difficulty. He felt smooth until he was readmitted to Deborah Hospital on Oct 12, 1964, for postoperative evaluation. His blood pressure was 160/70 mm Hg; a grade II systolic ejection murmur and the ejection click were audible. The ECG showed hypertrophy of the left ventricle and old myocardial damage. Catheterization of the right side of the heart, puncture of the left ventricle was performed. Studies of the right side of the heart “failed to show any abnormalities,” and “the data were indicative of normal hemodynamics and minimal obstruction to blood flow across the prosthetic valve (left ventricular-brachial artery gradient 15 mm Hg),” as reported by the cardiologist.

The patient was discharged on Oct 29, 1963, with advice to return to full activity and to continue to take sodium warfarin (Coumadin Sodium). He remained quite well until Nov 5, 1964, when he was rushed to the accident ward of the Lower Bucks County Hospital by ambulance at 12:05 PM. He was planning of chest pain and numbness, and weakness and pain of both legs; the symptoms had started 20 minutes earlier. The patient was extremely apprehensive with severe respiratory distress and cold, moist skin. His blood pressure was 80/0 mm Hg; his pulse was weak and grossly irregular. His chest was filled with bubbling rales; heart sounds were not heard. Both lower extremities, which were weak, felt cold and rapidly became a mottled purple, as did the abdomen below the umbilicus. Neither the aorta, the femoral, nor the peripheral pulses could be felt. The blood pressure and pulses in the upper extremities disappeared and the patient became comatose. An ECG disclosed ventricular fibrillation. Cardiac resuscitation, with attempted electrical defibrillation, external cardiac massage, and vasopressor drugs failed. The patient was pronounced dead at 12:55 PM.

An autopsy was performed three hours later. The significant findings were limited to the heart, lungs, and aorta. The pericardial cavity was almost completely obliterated by fibrous adhesions, but there was a small amount of recent hemorrhage in the visceral pericardium due to needle puncture. Both lungs were severely edematous. On opening the arch of the aorta and dissecting downwards toward the heart, the aortic valve prosthesis was encountered, but there was no ball found within the metal cage. Further examination of the aorta revealed the plastic ball lodged at the bifurcation of the abdominal aorta into the common iliac arteries. The prosthesis was otherwise in satisfactory position and condition. There was no antemortem clot in or near the prosthesis.

There was marked myocardial hypertrophy and extensive scarring of the endocardial surface of the left ventricle. The anterior descending branch of the left coronary artery was completely occluded by healed calcified atheromatous; the remaining heart valves were not remarkable.

Death was attributed to acute aortic insufficiency with failure of the left ventricle and pulmonary edema, due to ejection of the plastic ball from the aortic prosthesis with migration and occlusion of the terminal aorta. When replaced into the cage of the prosthesis, the ball easily fell through each and every one of the distal orifices, indicating that the ball had worn smaller in size. The ball itself was smooth and symmetrical, and the metal portions of the prosthesis were undisturbed.

Comment

When Starr and Edwards, in 1961, originally described their clinical experience with a ball-valve prosthesis for mitral replacement, they discussed in detail the manufacturing of the device. They also issued a warning:
While the laboratory demonstration of firm and lasting fixation, satisfactory hydraulic function and long-term survival is important in the evaluation of a proposed valve substitute, there will remain uncertainty regarding the long-term wearing ability of a prosthesis.

Extracorporeal tests by these authors in which the ball “received the mechanical equivalent of approximately 43 years in vivo” “revealed no change in ball dimension, shape or weight.” The construction materials for the aortic prostheses are the same as those used in the mitral ball-valve consisting of a silicone rubber ball enclosed in a highly polished cage of a material similar to a cobalt-chromium alloy (“Stellite 21, a Vitalium-like alloy”). Starr and his associates tested the prosthesis extensively in dogs and commented that long-term-survival animals were “still alive more than eight months after implantation.”

The Starr-Edwards prostheses have become very popular devices for replacement of damaged aortic or mitral valves, singly or in combination. Starr has indicated that “20 to 25 aortic valve replacement operations are performed each day” throughout the world.” Until the present case report, late deaths have largely been the result of intractable infection (bacterial endocarditis), while thrombosis and emboli have not been a problem with aortic replacement and the use of anticoagulants.

It is not clear why the ball wore down in this case; other patients have had the aortic prosthesis in situ for longer than the 11 months that this patient had the prosthesis in situ. One may only refer this problem back to the developers of the prosthesis. It is clear that this patient died of the most dramatic form of acute aortic insufficiency as the result of a mechanical failure of his Starr-Edwards ball-valve aortic prosthesis, namely, a reduction in size of the ball with its subsequent ejection from its metal cage and migration to the terminal aorta.

Studies on the heart were done by P. Chongvatana, MD,Cardiology Department, Deborah Hospital, Browns Mills, N.J. David E. Bassert, MD, Lower Bucks County Hospital, Bristol, Pa, performed the autopsy.

**Generic and Trade Names of Drug**

Sodium warfarin—Coumadin Sodium, Panwarfin, Prothromadin.

**References**


**Giant Meckel’s Diverticulum**

Harold L. Endlich, MD, Heinz L. Kafka, MD, and Leonard G. Powaser, MD

Regardless of its size, Meckel’s diverticulum may escape detection for long periods of time. It must be considered in every case of gastrointestinal dysfunction of an obscure nature. The diverticulum described in this case report is of interest because of its unusually large diameter and unusual position, as well as its prolonged symptomatic history.

**Report of a Case**

An 11½-year-old white girl was referred because of chronic anemia. Interrogation revealed that she had been afflicted with chronic vomiting since birth. During infancy, the attacks of vomiting occurred several times daily. At the age of 6½ months she was studied at another hospital, where roentgenographic studies of the stomach and small intestine failed to demonstrate any abnormality. Thereafter, it was assumed that the vomiting was on a functional rather than a mechanical basis. The frequency of episodes of vomiting gradually decreased; when the patient reached the age of six, they occurred approximately once a month. The attacks were preceded by cramping abdominal pain, and relief occurred upon vomiting.

On admission to the hospital July 7, 1964, the patient’s height was 57½ inches (145.8 cm), weight, 82 lb (37.2 kg), and blood pressure, 100/58 mm Hg. The results of physical examination were essentially normal. The abdomen was soft, not distended, and no tenderness or masses could be palpated.

Laboratory examination revealed the following values: hemoglobin, 9.2 gm/100 cc; hematocrit, 32%; white blood cell count, 7,500/cu mm, with normal differential count, and reticulocyte count, 2.2%/cu mm. Urinalysis showed normal values. Electroencephalogram, with the patient awake and asleep, was normal. Stools were positive for occult blood, but negative for enteric pathogens, ova, and parasites. Skeletal x-rays showed bone age compatible with chronologic age.

Roentgenograms of the colon showed a malrotation of the right side of the colon with the cecum lying high in the epigastrium. Gastroentoduenal and small-intestinal studies revealed an enormously dilated loop high in the left upper quadrant, interposed between the spleen and the greater curvature of the stomach. The marked dilatation of this loop caused displacement and compression of the stomach (Fig 1). The abnormal segment appeared larger than the stomach and emptied slowly but completely after 24 hours. The peculiar configuration and mucosal pattern of the segment suggested an enteric cyst, resection, or Meckel’s diverticulum.

At laparotomy, a huge Meckel’s diverticulum measuring 15 cm in diameter was found on the antimesenteric border of the ileum, approximately 100 cm from the ileocecal junction (Fig 2). Extensive adhesions were present in the small-bowel mesentery, and it was evident that the loop had repeatedly undergone volvulus with intermittent obstruction. It had enlarged to the point where it completely incorporated a segment of ileum within its boundaries. Many greatly enlarged lymph nodes were present in the mesentery.

Nonrotation of the small bowel was present, with complete failure of attachment of the mesentery of the small bowel. From the Department of Radiology, Holy Cross Hospital, San Fernando, Calif (Dr. Endlich) and the departments of pediatrics (Dr. Kafka) and surgery (Dr. Powaser), Rose-Loos Medical Group, Van Nuys, Calif.

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