Cardiac Tamponade in Systemic Lupus Erythematosus

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PERICARDITIS and peripheral effusion are frequent accompaniments of systemic lupus erythematosus. Cardiac tamponade, however, has been only rarely described. For example, Williams and Soutter stated that they have never seen such a case, and Taubenhaus et al. barely alluded to its occasional occurrence. Recently, we saw an instance of this phenomenon. The details of this interesting case are briefly outlined below.

The patient was a 34-year-old Puerto Rican woman who presented herself with weakness and pedal edema. Workup disclosed hypo-albuminemia, proteinuria, borderline hypercholesteremia, mild azotemia, borderline hypertension, generalized shotty lymphadenopathy, microscopic hematuria, and red blood cell cylinduria. Over the next 2 months, she developed unexplained transitory fevers, bilateral pleural effusions, alpha, and gamma hyperglobulinemia, a fleeting facial erythematous rash, mild arthralgias, progressive azotemia, and anemia without evidence of hemolysis. L.E. preparations, however, were persistently negative on 15 occasions. Lymph node and muscle biopsies were also negative. The pleural fluid was clear yellow, with a total protein content of 2.4 Gm. per cent, a specific gravity of 1.021, a total cell count of 340 cells per mm.\(^3\), and a differential count of 82 per cent segmented polymorphonuclear leukocytes, 16 per cent lymphocytes, and 2 per cent monocytes. No L.E. cells were seen, and an L.E. preparation of the pleural fluid was negative. When the blood urea nitrogen reached 127 mg. per cent, a painful pericardial friction rub appeared, and the chest x-ray and electrocardiogram suggested pericarditis with effusion. In the next few days, the patient became progressively weaker and lapsed into profound shock with no blood pressure and a rapid thready pulse. Pericardiocentesis was performed through the fourth left intercostal space in the parasternal line, with the withdrawal of 450 ml. of blood-tinged fluid. A smear of the pericardial fluid revealed almost all erythrocytes, and no L.E. cells were noted. The supernatant fluid had a protein content of 3.4 Gm. per cent and a specific gravity of 1.026. An L.E. preparation of the supernatant fluid was negative. Immediately after the pericardiocentesis, the patient regained consciousness, the blood pressure reappeared at 135/95, the pulse became slow and forceful, and the skin turned warm and dry. Steroids were then administered but were discontinued after a few days when psychosis supervened. Without medication, a partial remission ensued. The blood urea nitrogen dropped to mildly azotemic levels. She gradually regained strength, the hemoglobin level stabilized, and soon she was well enough to ambulate freely on the ward. Eight weeks after the remission began, the L.E. preparation became positive, severe anemia again developed, the direct Coombs test became positive, and agglutinins against homologous blood (type O, Rh negative) appeared, so that cross-matching for transfusion became difficult. Terminally, the uremia exacerbated, and she died with subarachnoid bleeding and bilateral bronchopneumonia.

Necropsy revealed the wire-loop lesion, fibrinoid thrombi, and hematoxylin bodies in the kidneys, onion-skin changes in the arterioles of the spleen, and chronic adhesive pericarditis, pleuritis, and peritonitis. The endocardium did not show Libman-Sachs endocarditis. There were new and old focal hemorrhages in the cerebrum and cerebellum, necrotizing pancreatitis, hemosiderosis of the liver and spleen, and bilateral bronchopneumonia.

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Summary and Conclusions

A case of systemic lupus erythematosus is described in which acute cardiac tamponade occurred due to a serosanguineous pericardial effusion of high protein content and high specific gravity, and in which dramatic and immediate improvement occurred following pericardiocentesis. Other interesting features of the case were the persistently negative L.E. preparations throughout all but the terminal part of her course, a positive direct Coombs test, the development of hemagglutinins to homologous blood on cross-matching, and a temporary but striking remission in her signs and symptoms, probably of spontaneous origin.

References

Thomas Addison

In the year 1855 Thomas Addison, one of the greatest clinical observers which this country has produced, published a description of The Constitutional and Local Effects of Disease of the Suprarenal Capsules. In the preface to his pamphlet—one of the classics of medical literature—after pointing out that the pathologist may sometimes be able to throw much light upon the functions of diseased organs, and that we are apt to forget how much of our physiological knowledge has been revealed by casual observations of the effects of disease, he goes on to speak of the adrenals, and adds: “It is as a first and feeble step towards inquiry into the functions and influence of these organs, suggested by pathology, that I now put forth the following pages.” Yet that “feeble step” was destined to be epoch-making; for Addison’s description of the disease which goes by his name was the starting-point of our present knowledge of the functions of the endocrine glands.

Let us pause, then, to consider how Addison set to work. He noticed that a certain group of symptoms was apt to occur in association—namely, anaemia, debility, remarkable feebleness of the heart’s action, irritability of the stomach, and a peculiar dingy or smoky discoloration of the skin—and that in every case in which this syndrome was met with extensive disease of the adrenals was found post mortem. So constant was this association that, in the later cases of the series, the disease of the adrenals was predicted during the lifetime of the sufferer.—Sir Archibald Garrod (B.M.J., 1926). The Quiet Art: A Doctor’s Anthology. Compiled by Dr. Robert Coope. Edinburgh & London, E. & S. Livingstone Ltd., 1952, p. 68.
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