Autopsy Incidence of Pulmonary Embolism in Tuberculosis

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Reports of the autopsy incidence of pulmonary embolism in non-surgical general hospital patients have resulted in increasing use of anticoagulant therapy in “medical” patients. The autopsy incidence of pulmonary embolism in patients dying with active tuberculosis has not been stressed in comparable fashion.

In a previous report I have discussed the necropsy incidence of active tuberculosis in a series of 634 consecutive autopsies performed over a five year period in an institution where both mental patients and general hospital patients were cared for, and where the average age at autopsy was unusually high (60 years). This same series of autopsies has also been used as a basis for reports on the incidence of pulmonary embolism due to prostatic thrombosis and the incidence of pulmonary embolism in coronary heart disease. The high incidence of pulmonary embolism and the frequent finding of active tuberculosis in the series suggested that this group of autopsies might well be analyzed from the viewpoint of the incidence of pulmonary embolism in subjects with active tuberculosis. Such an analysis should be helpful in evaluating the importance of prevention of pulmonary embolism in tuberculous patients and the possibility of the use of anticoagulant therapy in cases of tuberculosis complicated by thrombo-embolic phenomena or in certain selected cases where the possibility of thrombo-embolic complications seemed likely.

In the series of 634 consecutive autopsies, active tuberculosis was found in 111 subjects, an incidence of 17.5 per cent. These 111 cases form the basis of the present report, although it is recognized that this number is small from a statistical standpoint. Pulmonary embolism was found 27 times in these 111 autopsies (24.3 per cent). This incidence is slightly higher than the incidence of pulmonary embolism in the entire series (23.1 per cent). Four of these emboli were classed as massive (occluding one half or more of the pulmonary circulation) and 23 were classed as minor.

The four massive emboli were regarded as the immediate cause

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of death, and in three of these four cases the tuberculous lesions were of such nature that the patient might have recovered if the embolism had not occurred. The exact relationship between the minor emboli and the patients' deaths cannot be determined but lung infarcts occurred in two cases in this group and there seems no doubt that these minor emboli played at least a contributory role in some instances.

Due to the fact that the average age in the 111 cases of active tuberculosis was high (55 years), the possibility of coexisting heart disease as an etiologic factor in the production of the emboli was considered. Slight to moderate arteriosclerotic narrowing of the coronary arteries with no evidence of congestive heart failure was found in one of the four subjects with massive emboli. Of the 23 subjects with minor emboli, seven showed slight arteriosclerotic narrowing without congestive failure, two showed arteriosclerotic heart disease with congestive heart failure, and one subject died of coronary thrombosis while under treatment for tuberculosis. These figures would indicate that the tuberculous lesions requiring bed rest rather than associated heart lesions were responsible for the emboli in this group.

It should be mentioned that one massive and two minor emboli occurred in patients where the tuberculosis was active in areas other than the lungs. All of the rest of the emboli and all of the infarcts occurred in patients with active pulmonary tuberculosis. All of the patients were at bed rest.

SUMMARY

Pulmonary embolism was found 27 times in 111 consecutive autopsies in subjects with active tuberculosis (24.3 per cent). This compares with an incidence of pulmonary embolism of 23.1 per cent in a series of 634 autopsies in which the 111 cases of tuberculosis were found. Four of the emboli were classified as massive and 23 were regarded as minor. Lung infarction occurred four times.

This high incidence of pulmonary embolism in patients dying with active tuberculosis suggests that proper prophylactic measures to reduce the incidence of pulmonary embolism and the use of anticoagulant therapy such as dicumarol or heparin may play a definite role in the treatment of tuberculosis. Careful laboratory control of the prothrombin and coagulation times would be required in the use of dicumarol or heparin because of the possibility of pulmonary hemorrhage.

RESUMEN

En 111 autopsias realizadas en enfermos de tuberculosis pulmonar activa, embolia pulmonar estubo presente en 27 casos (24.3 por
ciento). Estos 111 casos de tuberculosis fueron hallados en una serie de 634 autopsias; obteniendo 23.1 por ciento de embolias pulmonares en el total.

Cuatro de las embolias eran masivas y 23 fueron menores. Infarto del pulmón ocurrió cuatro veces.

Este porcentaje alto de embolia pulmonar, en enfermos que mueren de tuberculosis pulmonar activa, sugiere el uso profilático de anticagulantes como ser decumero o heparina; los cuales, posiblemente, desempenen un papel importante, en el tratamiento de la tuberculosis. Deben hacerse prolijos estudios del tiempo de coagulación y protrombina, en los casos en que se use decumero y heparina, debido a la posibilidad de hemorragia pulmonar.

REFERENCES


