

A CASE OF CLINICALLY UNDIAGNOSED CHRONIC LEUKAEMIA: UNEXPECTED SUDDEN DEATH OF A PATIENT WITH WHIPLASH INJURY

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ABSTRACT

This is the autopsy case of a 42-year-old male with whiplash injury who died suddenly and unexpectedly. The pathological diagnosis was chronic myelocytic leukaemia with involvement of the heart, spleen, lymph nodes, liver, kidneys and bone marrow. The immediate cause of death is considered to be heart failure resulting from leukaemic cell infiltration. In this case, the underlying leukaemia was masked by the persisting symptoms following whiplash injury, and left undiagnosed clinically.

INTRODUCTION

Blood dyscrasias such as leukaemias and anaemias may develop insidiously and cause unexpected death from some rapidly developing complication^{1) 2)}. I have recently encountered an autopsy case of clinically undiagnosed chronic myelocytic leukaemia in a patient with whiplash injury in whom unexpected sudden death resulted from heart failure evidently caused by massive leukaemic cell infiltration. The paucity of literature on such unexpected sudden death in the field of legal medicine has prompted this report.

CASE HISTORY

On September 13, 1971, a 42-year-old male sustained a whiplash injury while driving a car, and was admitted to an orthopaedic clinic. X-ray examination of the cervical spine revealed no abnormalities. After being discharged 1 month later, he was seen at intervals in the department of orthopaedic surgery of a university hospital because of persistent headache, dizziness, nausea and neck pain, and was treated conservatively on an outpatient basis. At the beginning of October, 1975, he began to complain, in addition, of malaise, loss of appetite, vomiting and constipation, but received no other treatment. On October 26, he was seized with discomfort in the chest, epigastric fullness and chilly sensation, and became unconscious. An ambulance arrived 3 hours later, when he was found dead.

GROSS FINDINGS

An autopsy was performed about 12 hours after death. Rigor mortis was established, but postmortem lividity poorly developed posteriorly. The skin was pale and slightly

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icteric. The conjunctivae were pallid. There were numerous petechiae over the entire body.

The abdominal cavity contained approximately 30 ml of blood-tinged ascites. The lymph nodes in general, particularly the mesenteric, were enlarged, and greyish-white on section.

A few petechial haemorrhages were seen over the pericardial surface. The myocardium was flabby and, on section, displayed well-demarcated pale-grey, firm nodules. There was a mild arteriosclerosis.

The spleen was firm and enlarged (430g); the capsule thickened; the pulp dark-red. The follicles were not discernible.

The liver weighed 2,100g, and the cut surface was firm and yellowish-pink.

The left and right kidneys weighed 300 and 280g, respectively. Numerous petechiae were observed on the renal capsules. Section through the kidneys exhibited pale-grey tumor nodules as in the myocardium. The corticomedullary junction appeared indistinct.

The brain showed no gross abnormalities except pallor.

The cut surface of the sternum revealed a pinkish-grey marrow.

HISTOLOGIC FINDINGS

The myocardium showed diffuse interstitial haemorrhages and massive infiltration by immature and mature granulocytes (Fig. 1). Myocardial fibres were degenerated, atrophic and partly disrupted or lost.

The splenic pulp was crowded with large numbers of myelocytes at all stages of maturation, with smaller numbers of nucleated red cells and megakaryocytes.

The microscopic picture of the lymph nodes was generally similar to that of the spleen, and all the cells ordinarily found in the bone marrow were present.

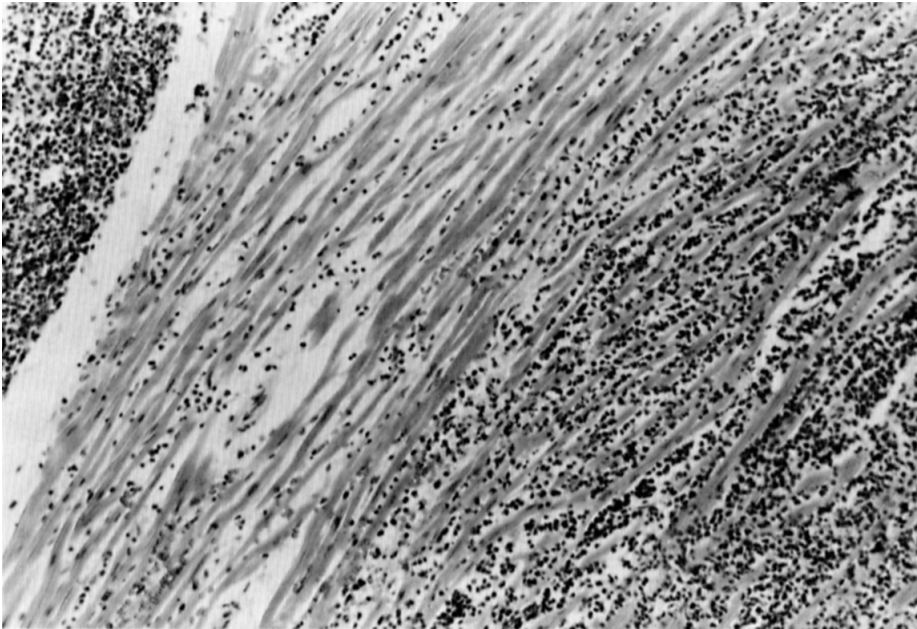


Fig. 1. Massive myelocytic infiltration with degeneration and disruption of myocardial fibres. Haematoxylin-eosin stain. X150.

The sinusoids and portal areas of the liver were massively infiltrated by myelocytes. The hepatic cells exhibited atrophy and degeneration, having fat globules in the cytoplasm.

There were extensive infiltrations of the kidneys, lungs, pancreas and other organs by immature and mature granulocytes.

In the bone marrow, nodular growths of myelocytes at all stages of maturation were noted. No megakaryocytes were seen. Normoblasts were sparsely present.

DISCUSSION

The case presented here showed the following pathological findings: (1) pallor, (2) haemorrhagic diathesis, (3) splenomegaly, (4) lymphadenopathy, (5) hepatomegaly, (6) infiltration of the heart, spleen, lymph nodes, liver, kidneys, bone marrow and other organs by myelocytes at all stages of maturation, (7) presence of megakaryocytes in the spleen and lymph nodes, (8) absence of megakaryocytes in the bone marrow and (9) degeneration and disruption of the myocardial fibres. On the basis of these findings, the deceased was diagnosed as having chronic myelocytic leukaemia.

The commonest cause of sudden death in leukaemias is cerebral haemorrhage, sometimes multiple^{1) 2)}. In other cases, heart failure may result from cardiac dilatation or myocardial degeneration caused by prolonged anaemia, coupled with coronary arteriosclerosis^{1) 3)}. Infiltration in the myocardium is a relatively infrequent cause of sudden death and leads to rupture of the ventricle or descending aorta³⁾.

In the present case, the immediate cause of death is considered to be heart failure resulting from leukaemic cell infiltration. It is likely that cardiac muscle was weakened by the infiltration process that caused degeneration and disruption of the myocardial fibres. Another possibility is that the infiltration involved a particularly sensitive portion of the myocardium, and resulted in cardiac arrest as in Adams-Stokes' syndrome.

It is noticeable that the fatal disease had not been clinically diagnosed. This is probably because the underlying leukaemia was masked by the persisting symptoms of headache, malaise, nausea and vomiting following whiplash injury. The present case suggests that one should never omit routine blood examination, even if the nature of trauma precludes the need for it.

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