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Dr Kaye comments as follows:

The confirmation by Dr Van de Vyver and co-workers that aluminium can be demonstrated in marrow cells using the aluminon stain and verified using a microprobe analytical technique is gratifying. The combined storage of iron and aluminum is not unexpected, although it was not seen in our material. This is probably because our patients tend to be kept mildly iron deficient and marrow iron stores are usually meagre. One of our patients with clinically severe aluminium bone disease was treated with desferoxamine for 8 months, after which a repeat bone biopsy showed disappearance of trabecular aluminium staining but persistence in the marrow, implying different chemical reactivity of aluminium in the two sites.

I have not systematically looked at the reason(s) for the apparently less satisfactory staining in methyl methacrylate or epon embedded material and this could be due to fixation or other factors. Whatever the explanation it is further evidence of a difference between the reactivity of the marrow and mineral aluminium deposits. The authors observations are very interesting and amplify the results previously reported.

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Acute myeloid leukaemia in a patient with congenital antithrombin III deficiency

Congenital antithrombin III deficiency is well recognised as a rare cause of venous thrombosis. The occurrence of acute myeloid leukaemia in a patient with congenital antithrombin III deficiency has not previously been reported.

A 45 year old woman presented in July 1982 with a five week history of tiredness, spontaneous bruising, and gingival swelling. She had a history of postoperative venous thrombosis on two occasions and a striking family history of thrombotic disease: her father and paternal uncle had had major thrombotic episodes while in their twenties. The maternal side of the family was unaffected.

At presentation fever (39°C), pallor, and widespread bruising were noted, but no lymphadenopathy, hepatosplenomegaly, or sternal tenderness was found. Haemoglobin concentration was 10.0 g/dl, white cell count $5.5 \times 10^9/l$, and platelet count $290 \times 10^9/l$. Blood film showed 95% blast cells. Histology and cytochemistry of a marrow aspirate led to a diagnosis of acute myeloid leukaemia (M1).¹

Chemotherapy, comprising daunorubicin, cytosine arabinoside, and thioguanine (DAT) was started. After the first pulse of chemotherapy, blast cells were no longer apparent in the peripheral blood and progress was uneventful. Eighteen days after admission sudden severe pleuritic chest pain developed, and there was pain in the right calf. Electrocardiogram was normal, but chest x ray examination showed opacification of left upper and right lower zones, an elevated right hemidiaphragm, and a small right sided pleural effusion. Pulmonary embolism was diagnosed, although the patient was unfit for isotopic scan. There was no evidence of disseminated intravascular coagulation on baseline coagulation screening. Heparin by continuous intravenous infusion was cautiously instituted in a dosage of 10 000 IU every 12 h in view of the pronounced thrombocytopenia, and monitored according to the partial thromboplastin time with kaolin. Platelet concentrates were given every 12 h throughout this period.

Despite these measures the patient developed recurrent chest pain and haemoptysis with subsequent hypotension and acute renal failure, and she died 33 days after admission. Necropsy revealed multiple small and medium sized pulmonary emboli, bilateral pulmonary infarcts, and multiple thrombi in the calf areas of the right leg.

An amidolytic antithrombin III assay performed on a blood sample taken before heparinisation using a commercially available chromogenic substrate (Kabi S 228) showed a substantial reduction in antithrombin III at 43% (normal range 80-115%). Samples taken later from the patient's mother showed normal antithrombin III levels (90%). No other relatives were available for study.

Acquired antithrombin III deficiency can occur as a result of disseminated intravascular coagulation in a number of conditions.^{2,3} The absence of disseminated intravascular coagulation in this case together with the striking past and family history of thrombosis strongly support a diagnosis of congenital antithrombin III

Laser microprobe mass spectrum originating from one of the aluminon negative bone marrow macrophages of patient A. The brown colour of these cells suggested considerable iron storage.