Letters to the Editor

Pentavalent technetium-99m dimercaptosuccinic acid scintigraphy is useful in diagnosis and localization of neuroblastoma

Sir,

Pentavalent technetium-99m dimercaptosuccinic acid (Tc-99m-V-DMSA) has proved to be useful for localization of medullary carcinoma of thyroid and phaeochromocytoma. As neuroblastoma is also believed to arise from neural crest, we explored the usefulness of Tc-99m-V-DMSA scintigraphy in this condition. Forty-two patients with histologically proven tumours were scanned by the method described by Ohta. Scintigrams using a conventional gamma camera were taken 60–120 minutes after intravenous administration of 10 mCi Tc-V-DMSA. The distribution of tumours was; neuroblastoma 17, soft tissue sarcoma 9, non-Hodgkin’s lymphoma 6, renal cell carcinoma 2, invasive thymoma 2, and one case each of metastatic germ cell tumour, adrenal cortical tumour, metastatic Wilms’ tumour, bronchogenic carcinoma, metastatic adenocarcinoma of stomach to femur, and anaplastic carcinoma of thyroid. All patients had gross clinical disease at the time of the procedure. All 17 patients with neuroblastoma and the patient with anaplastic carcinoma showed avid uptake. In our study the scintigraphy had a sensitivity and specificity approaching 100% and 98%, respectively, for neuroblastoma (Figure 1).

Figure 1 Photograph of a Tc-99m-V-DMSA scan showing avid uptake with central necrosis in a patient with neuroblastoma in lower abdomen.

The usefulness of this scanning in neuroblastoma has not been reported before. As it is one of the more common tumours of childhood, the implications of our observation are significant and manifold. (a) Most importantly, the technique may help in differentiating various malignant round cell tumours. Not uncommonly pathologists on routine histology find it difficult to differentiate neuroblastoma from other malignant round cell tumours such as soft tissue sarcomas and lymphomas. The characteristic light microscopic demonstration of Homer–Wright neural rosettes may not always be possible in neuroblastoma. The electron microscopy, immunochemistry and urinary catecholamine estimation may help in the diagnosis in a few more cases. However, a sizable group will be left with a diagnosis of a ‘malignant round cell tumour’ that cannot be characterized further. Significantly, 14 of our patients who had either lymphoma or soft tissue sarcoma (all malignant round cell tumours) showed negative uptake. (b) The technique could be used to localize and monitor the response to treatment. (c) Tc-99m-V-DMSA scanning is much cheaper, simpler and less time-consuming that 131I-iodobenzylguanidine scanning that is currently used in neuroblastoma. (d) Its potential as an adjuvant in the treatment of such a refractory tumour as neuroblastoma could be investigated with the use of a new agent such as rhenium-186 (a technetium analogue) with beta and gamma emission.

Departments of Nuclear Medicine, Medical Oncology and Pathology, All India Institute of Medical Sciences, New Delhi 110 029, India

References


Nifedipine and thrombocytopenia

Sir,

Thrombocytopenia from nifedipine seems to be a rare event.

A 64 year old woman affected by diabetes mellitus and high blood pressure was hospitalized because of gingivitis and petechiae.

Apart from a platelet count of 20,000/mm³, peripheral blood and coagulation screen were normal. Bone marrow
aspirate was cellular with all lines represented, but the meagakaryocytes showed maturation arrest. A drug-related thrombocytopenia was suspected her medications were nifedipine and gliclizide, which were stopped. The patient had started taking nifedipine 20 days before and had been on treatment with gliclizide for 5 years.

Prothrombin time, partial thromboplastin time and thrombin clotting time were normal, but the fibrinogen level was elevated to 700 mg/dl and the concentration of fibrin degradation products was less than 10 μg/ml. Liver function tests were normal.

We executed the Dixon test to investigate the presence of antibody anti-nifedipine and gliclizide, the first test only being positive. Ten days after hospitalization, platelets were normal. We suspect that nifedipine may be the cause of thrombocytopenia in this case, even though this is rarely described in medical literature.

The pathogenesis of thrombocytopenia from nifedipine is unknown.

On the basis of the foregoing, we suggest that thrombocytopenia may occur as a result of exposure to nifedipine.

Enrico Breda
Luigi Temperilli
Anna Maria Sidoti
Fulvio Pauselli
Department of Internal Medicine,
Ospedale Fatebenefratelli 'S. Giovanni Calibita',
Isola Tiberina, 00186 Roma,
Italy.

References

Massive intra-abdominal bleeding complicating bone marrow aspiration and biopsy in multiple myeloma

SIR,

Bone marrow aspiration and biopsy have traditionally been considered safe procedures even when severe coagulopathies or thrombocytopenia are present. We recently encountered a patient with multiple myeloma who had fatal intra-abdominal haemorrhage following iliac crest bone marrow aspiration and biopsy. This experience suggests that the risk of complications from bone marrow aspiration or biopsy in diseases causing severe bone destruction should be re-evaluated.

An 81 year old woman with IgA myeloma refractory to chemotherapy was admitted with epistaxis, gum bleeding, lethargy and abdominal pain. Haemoglobin was 6.7 g/dl, white cell count 4.97 x 10^9/l, platelet count 49 x 10^9/l, serum creatinine 160 μmol/l, and coagulation screen normal.

To determine the aetiology of the pancytopenia, iliac crest aspiration and biopsy were unsuccessfully attempted bilaterally. A right posterior iliac crest bone marrow biopsy was then obtained with a Jamshidi – Swan needle. A hypercellular marrow full of plasma cells and lymphocytes was found with few normal cells. No amyloid was found.

Several hours after the procedure, the patient became hypotensive. During the next few hours, ecchymoses appeared in the right lower quadrant. A computed tomographic scan revealed a large haemoperitoneum with widespread lytic lesions particularly in iliac bones. Angiography revealed bleeding overlying both sacro-iliac joints. There was no obvious bleeding from the viscera. Selective embolization of the right ilio-lumbar and internal iliac arteries achieved only partial haemostasis. She died on the ninth hospital day.

This outcome of bone marrow biopsy and aspiration has not been described before in multiple myeloma. In this case, the temporal association implicates bone marrow aspiration or biopsy of bone damaged by widespread lytic disease as the likely cause of fatal haemorrhage. It is of note that bleeding was found also on the left iliac crest, which only underwent an aspiration attempt using a 16 gauge sternal aspiration-type short needle.

The haemorrhagic diathesis of myeloma, a potential result of coagulation factor inhibition, hyperviscosity, vascular abnormalities or impaired platelet function, could have contributed to bleeding in this patient although thrombocytopenia was not severe. In view of normal tests of coagulation, it appears unlikely that significant coagulopathy secondary to fibrin polymerization defects or to acquired inhibitors existed. Despite the biopsy findings, amyloid infiltration of blood vessels preventing vasoconstriction is not excluded.

Retroperitoneal haemorrhage following bone marrow biopsy has been reported in two previous patients, both of whom survived. In these cases, osteoporosis and renal osteodystrophy were the predisposing factors. Although the complication rate from repeated marrow aspirations of healthy iliac crests as in bone marrow donors is small, the risk with diseased bone may be greater. Our case suggests that when severe bone disease is present, the indication and value of bone marrow aspiration or biopsy should be carefully considered.

Sanjeev Gupta
Michael L. Meyers
John Trambert*
Henny H. Billett
Departments of Medicine and *Radiology,
Albert Einstein College of Medicine,
Bronx, NY 10461, USA.

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E. Breda, L. Temperilli, A. M. Sidoti and F. Pauselli

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