
Giant Hemangioma of the Adrenal

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Adrenal hemangiomas are a well-known entity, but giant adrenal hemangiomas are uncommon. We report on a patient with a symptomatic giant right adrenal hemangioma that was initially presumed to originate from the liver. The clinical presentation and management are described.

Patient Description

A 45 year old healthy woman complained of early satiety, right upper quadrant discomfort and fullness of 6 months duration. She denied fever, weight loss, jaundice or other systemic complaints. Initial workup revealed a mild elevation in liver enzymes, and a subsequent computed tomography scan of the abdomen revealed what was believed to be a giant liver hemangioma [Figure]. Because of continuing abdominal discomfort the patient was

taken to the operating room for exploration. Intraoperatively, a large 10 cm firm cystic mass was identified under the right liver lobe. After mobilization of the right liver lobe, it became clear that the mass did not violate the liver capsule but instead arose from the right adrenal gland. A right adrenalectomy was performed en bloc with removal of the mass.

Gross pathologic examination showed a 10.8 cm ovoid mass with multiple cystic spaces filled with fresh organizing thrombus arising from the adrenal gland. Histologic examination showed large hemangogenous pockets lined with flattened endothelial cells. The tumor compressed the adrenal remnant, which showed normal histology. The findings were consistent with an adrenal hemangioma. The patient had an uneventful recovery with no further symptomatology.



CT scan of the abdomen, revealing a large mass compressing the right lobe of the liver.

Comment

Hemangiomas of the adrenal gland are rare. The first clinical case was reported in 1955 by Johnson and Jeppesen [1]. Since then, approximately 50 cases have been described, with fewer than 10 having a diameter larger than 10 cm.

While most hemangiomas are found incidentally, larger masses – such as the one described here – may present with flank or abdominal pain, early satiety, or may be palpated on physical examination. Symptomatic adrenal hemangioma has been reported to be associated with pregnancy [2]. Most adrenal hemangiomas are unilateral, benign and non-functioning, although bilateral tumors have been reported in cadavers [3]. Hypertension and coexistence with malignant hemangioendothelioma have also been described [4,5]. Our patient had a unique presentation of elevated liver enzymes caused by liver compression.

Diagnosis is usually made on imaging studies. Post-contrast CT reveals patchy areas of peripheral enhancement. T1-weighted images on magnetic resonance

imaging demonstrate a non-homogeneous mass, and T2-weighted images classically show high signal intensity of the mass.

Liver hemangiomas do not require resection unless they are symptomatic or found to be of large size in women of childbearing age. There are insufficient data regarding the natural history of adrenal hemangiomas to make similar recommendations. The lesions have been reported to coexist with malignancies and may be symptomatic; we therefore advocate surgical resection for all diagnosed lesions.

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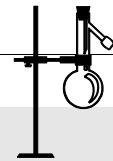
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Research Projects

Novel Therapeutic Approaches for the Treatment of Leukemia Based on Chemokine/Chemokine Receptor Interactions

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Background: Chemokine/chemokine receptor interactions regulate immune cell trafficking in the body. Natural killer (NK) cells are key effector cells mediating the graft-versus-leukemia (GVL) effect. Resting NK cells express the chemokine receptors CXCR1, CXCR3, CXCR4, CCR1 and CCR5.

Objectives: Our study focuses on manipulating the trafficking of activated NK cells to the bone marrow (BM) in order to augment the GVL effect of donor-transfused NK cells.

Methods: Purification of NK cells was

done using magnetic bead separation. Chemokine receptor regulation and function was studied using flow cytometry analysis and transwell migration assays. Characterization of BM-expressed and induced chemokines was done by polymerase chain reaction, immunohistochemical staining and ELISA assays, in both human specimens and using animal models.

Results: Interleukin (IL)-2 stimulation of NK cells induced up-regulation of the chemokine receptors CXCR3 and CCR5 and down-regulation of CXCR4 and CXCR1. Correspondingly, CXCL10 (CXCR3

ligand) induced the migration of activated NK cells through transwells coated with a BM endothelial cell line. Following interferon-beta, IL-18 and IL-12 injections, CXCR3 ligands (CXCL9, CXCL10) expression in the BM was significantly increased relative to peripheral blood.

Conclusions: NK cell activation stimulates their response to CXCR3 ligands. Induction of CXCR3 ligand production in the BM via cytokines or via gene therapy delivery strategies may in the future facilitate the recruitment of donor-infused activated NK cells to the BM.

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