Table 1 - The cases performed in each group.

Operation	Group A (negative suction)	Group B	*P*-value
CABG	69	189	
CABG + MV Repair	8	11	
CABG + MVR	1	2	
CABG + AVR	0	1	
Total (%)	78 (28)	203 (72)	
Blood loss (ml) mean ± SD	870 ± 270	630 ± 215	$p<0.05$
Re-opening (%)	10 (12.8)	9 (4.4)	$p>0.05$
Pericardial effusion (%)	2 (2.5)	9 (4.4)	$p>0.05$
Mortality (%)	4 (5.1)	7 (3.4)	$p>0.05$

CABG - Coronary artery bypass grafting, MV Repair - Mitral valve repair, MVR - Mitral valve replacement, AVR - Aortic valve replacement.

on rates of re-opening for bleeding, development of pericardial effusion, and an overall mortality. It can be seen from our data, that the rates of residual effusions were higher in the control group; however, did not reach statistical significance. Although, there were more re-opening in the negative suction group, this also did not reach statistical significance. Despite the limitation of not being a randomized study, nonetheless, it shows that negative suction applied to the chest drains after CABG increase Mediastinal drainage, however, had no effect on re-opening rates and overall mortality.

Acknowledgment. *We wish to thank all the nurses in the Queen Alia Heart Institute, Intensive Care Unit, King Hussein Medical Center, Amman, Jordan, for their help in making this study possible.*

Received 14th February 2006. Accepted 6th August 2006.

From the Department of Cardiac Surgery, Queen Alia Heart Institute, Amman, Jordan. Address correspondence and reprint requests to: Dr. Basel Harahsheh, Department of Cardiac Surgery, Queen Alia Heart Institute, Amman, Jordan. Tel. +795773838. Fax. +962 (6) 5715944 E-mail: basel_md@hotmail.com

References

1. Munnell ER, Thomas EK. Current concepts in thoracic drainage systems. ***Ann Thorac Surg*** 1975; 19: 261-268.
2. Akowuah E, Ho EC, George R. Less pain with flexible fluted silicone chest drains than with conventional rigid tubes after cardiac surgery. ***J Thorac Cardiovasc Surg*** 2002; 124: 1027-1028.
3. Visser CA, Van Wezel HB, Koolen JJ. How soon should drainage tubes be removed after cardiac operations? ***Ann Thorac Surg*** 1989; 48: 540-543.
4. Obney JA, Barnes MJ, Lisagor PG. A method for mediastinal drainage after cardiac procedures. ***Ann Thorac Surg*** 2000; 70: 1109-1110.
5. Svedjeholm R, Hakanson E. Postoperative myocardial ischemia caused by chest tube compression of vein graft. ***Ann Thorac Surg*** 1997; 64: 1806-1808.

Amyloid-depositing plasmacytoma of cervical spine masquerades as a granulomatous inflammatory reaction

Ibrahim S. Tillawi, *MBBCh.*

A case of plasmacytoma involving the lower cervical vertebral body is presented, in which the tumor resulted in lytic boney changes in the fourth cervical vertebral body with outward extension leading to the development of a large paravertebral soft tissue mass. Aspiration of the soft tissue component of the mass, showed a number of eosinophilic amorphous variable-sized clumps, inflammatory cells including large numbers of plasma cells, spindled shaped cells, multinucleated giant cells and blood, all suggesting that the lesion is benign in nature. Three pathologists, who independently had the lesion aspirated and cytopathologically examined, gave discrepant diagnosis, ranging from inflammatory granulomatous reaction to a highly lethal small cell variant of osteogenic sarcoma, not very much different in that from the differential diagnostic list given by the radiologist. This led the neurosurgeon to request frozen section assistance towards a definitive therapeutic surgical intervention; frozen section revealed the true nature of the lesion, which consisted of a plasmacytoma associated with secondary granulomatous reaction due to amyloid produced by the tumor cells. This communication emphasizes the need for the pathologists to be aware, not only of the characteristic appearance of amyloid on cytological preparations, but of its inherent capability of producing a granulomatous reaction; if its presence is overlooked, or not considered in the appropriate context, the pathologic diagnosis will change.

A 43-year-old previously healthy male presented with a right sided brachialgia, progressive in nature, of few months duration. Clinical examination revealed the presence of the right sided weakness of ipsilateral elbow extension, without concomitant signs of myelopathy and flexor plantar responses. Neck movement was also markedly restricted. Computed tomography scan, revealed the presence of a heterogenous irregular expansile and infiltrative large boney mass that destroyed the pedicle of the fourth cervical vertebral body and extended into the surrounding adjacent soft tissues, which was interpreted that it can similarly be inflammatory or neoplastic in nature. The mass was a fine needle aspirated by 3 pathologists who independently gave 3 different opinions after examining 3 different samples each obtained individually from a separate site from the large soft tissue mass. The aspirations generally showed variable degrees of admixtures of irregularly shaped eosinophilic amorphous clumps, inflammatory cells including large numbers of plasma cells, many "spindled" shaped cells, multinucleated giant cells, and blood. One pathologist opinion was that of a benign reactionary granulomatous process. The second pathologist thought that it might represent a small cell variant of osteogenic sarcoma of bone; amyloid fragments seen were thought to represent fragments of necrotic bone and "malignant osteoid". The third pathologist considered the lesion to represent an aneurysmal bone cyst, based on the presence of blood, giant cells, and a scattering of "inflammatory cells included among which are plasma cells". Subsequently, exploration was carried out, and excision of the most peripheral skeletal muscles, in addition to almost the entirety of the infiltrative mass along with a major portion of the centrally placed and almost totally collapsed, the fourth cervical vertebral body. Frozen section interpreted by an independent fourth pathologist, found that the lesion is a plasma cell neoplasm with associated amyloid (amyloid tumor) production and secondary granulomatous inflammatory cell reaction. Retrospectively, the cellular composition of the slides obtained from all 3 aspirations, revealed the presence of amorphous eosinophilic clumps, inflammatory cells including large number of plasma cells, spindled shaped cells, multinucleated giant cells, and blood in various proportions and densities. Two pathologists overlooked the presence of amyloid and regarded the seemingly unrelated elements of the lesion as either an inflammatory process or an aneurysmal bone cyst with or without a giant cell tumor component. The third pathologist, considered the lesion to represent small cell variant of osteogenic sarcoma, as he mixed up amyloid with malignant osteoid and necrotic fragments of malignant osteoid. The surgically excised specimen revealed heterogeneously represented sheets and nests of plasma cells of variable maturational stages and sizes, between large masses of congophilic amyloid, and scattered large number of multinucleated giant cells, some of which containing very tiny amyloid material within their cytoplasm. In many occasions, the scalloped edges of the islands of amyloid were rimmed by spindled shaped cells, which characteristically had a cartwheel nucleus of plasma cells, and as shown later stained with CD138. Necrosis was not seen anywhere in the tumor. In the soft tissue component, one also observes irregular skeletal muscle fibers separated by dense plasma cell infiltrates and Congo red-positive amyloid. The diagnosis made an extramedullary plasmacytoma with amyloid production (amyloid tumor) with secondary granulomatous reaction. CD138 as well as kappa were evident in the same cells, which were simultaneously negative for lambda. On admission, complete blood count, erythrocyte sedimentation rates, serum calcium, phosphorus, beta 2-microglobulins, protein electrophoresis, and urine electrophoresis were all within the normal range. The initial bone marrow biopsy and aspiration revealed the presence of less than 5% plasma cells, and interpreted as a cellular marrow. The patient subsequently received radiotherapy to the region of the fourth cervical vertebral body spine and 4 cycles of chemotherapy (myleran and prednisolone).

Radiology is pathology-see-through; the list of diagnostic considerations will greatly aid the pathologist when it comes to finalize the pathological report. In the differential diagnosis of a fine-needle aspiration biopsy of soft tissue masses resulting from primary bone lytic lesions, a wide range of diagnostic considerations should be included depending on the predominating constituent cells in the aspirated material. Usually, some technical variables interfere in the final cytologic al diagnosis; one such factor includes the uniformity of the cytological spread, which truly depends on the cellular composition of the pass used, and this invariably influences the diagnostic accuracy in any case. Overlap conditions are of paramount importance to be segregated and identified; that by itself, primarily depends on several factors. Lesions that do include a heterogenous and unequal admixture of giant cells, spindled cells, stromal vascularized fibroconnective tissue cells, and few other unrelated inflammatory cells with an inconspicuous amount of amyloid, will end up being diagnosed as either inflammatory or aneurysmal bone cyst on fine needle aspiration biopsy (FNAB) in this clinicopathologic setting, as was the case in this patient. Additionally, accurate identification of the true nature of amyloid on cytologic preparations and recognizing their presence is equally challenging. Although, previous reports and major textbooks have detailed their cytologic

features, partly, the rare incidence of such tumors makes it a potentially difficult diagnosis and a diagnostic pitfall.[1,2] Conversely, the straightforward? diagnosis of extramedullary plasmacytoma with or without associated amyloid deposition, requires primarily the presence of a rather uniform preparation of invariably large number of discohesive plasma cells of stages of maturation and sizes, with an associated background of lymphoglandular bodies as well as occasional lymphocytes, and as minimal as possible stromal cells.[3] It is such, that without these constituent cells, one cannot make the diagnosis of extramedullary plasmacytoma. Other factors that also interfere in the diagnosis of plasmacytoma includes; 1. The degree of differentiation of plasma cells 2. Relative frequency and proportion of distribution of the accompanying inflammatory/ reactionary and stromal cells 3. Demonstrating unequivocal monoclonality in the examined cells.[3] On the other end of the "neoplastic spectrum", one should also consider other neoplasms, whose constituent discohesive neoplastic cells greatly mimic plasma cells in various other neoplasms, when the later assumes other shapes or grades of differentiation that may mimic plasma cells. Amelanotic melanoma, or aggressive non-Hodgkin's lymphoma is such examples, and it depends on how the cytopathologist can easily make the distinction between these cell types sometimes; that will most likely require immunohistochemical staining.[3] In one of the largest studies on FNAB of extramedullary plasma cell tumors, there was no emphasis made on the role of the giant cell as a cellular component of these neoplasms, especially with special reference to its close association and relationship to the amyloid deposition. Similarly, intracytoplasmic amyloid observed within the cytoplasm of the giant cells did not receive any emphasis.[3] The presence of a certain proportion of a giant cells in lytic bone lesions will primarily aid in the separation of a main giant cell tumor of bone from "reactionary" presence of giant cells in primary bone lesions; the combination of clinical, radiological, and the complete cellular composition alongside with giant cells help in the final distinction. With reference to the small cell variant of osteogenic sarcoma, especially if the needle passes into a zone of minimal osteoid formation, or other sarcoma composed mainly of small cells, can still pose a problem in the differential diagnosis. Amyloid can sometimes be confused (especially in scanty amounts and if the cytologic setting is right), with osteoid.[4,5]

Received 5th June 2006. Accepted 30th August 2006.

From the Division of Hematopathology, Department of Pathology, Riyadh Military Hospital, Riyadh, Kingdom of Saudi Arabia. Address correspondence and reprint requests to: Dr. Ibrahim S. Tillawi, Division of Hematopathology, Department of Pathology, Riyadh Military Hospital, Riyadh 11154, Kingdom of Saudi Arabia. E-mail: ibrahimtellawi@hotmail.com

References

1. Gutmann EJ. Granulomatous inflammation related to amyloid deposition in a focus of multiple myeloma. Report of a case with diagnosis by fine needle aspiration biopsy. ***Acta Cytol*** 1995; 39: 793-797.
2. Yakulis R, Dawson RR, Wang SE, Kennerdell JS. Fine needle aspiration diagnosis of orbital plasmacytoma with amyloidosis. A case report. ***Acta Cytol*** 1995; 39: 104-110.
3. Nicol KK, Ward WG, Savage PD, Kilpatrick SE. Fine needle aspiration biopsy of skeletal versus extraskeletal osteosarcoma. ***Cancer*** 1998; 84: 176-185.
4. Powers CN, Wakely PE Jr, Silverman JF, Kornstein MJ, Frable WJ. Fine needle aspiration biopsy of extramedullary plasma cell tumors. ***Mod Pathol*** 1990; 3: 648-653.
5. Klein MJ. Differential diagnostic points in giant cell lesions. ***Pathology Case Reviews*** 2000; 5: 305-312.

Maternal and fetal thyroid stimulating hormones and the fetal indices of maturation, growth, and development

Oluwole O. Adedeji, *D.PHIL,*
George K. Oyakhire, *FRCOG.*

The thyroid stimulating hormone (TSH) occupies a central position in the hypothalamic-pituitary-thyroid axis, which regulates and controls the secretion of thyroid hormones.[1] The developing fetus depends on the thyroid hormones for neurological maturation, growth, and development.[2,3] Thyroid hormones are supplied by the mothers to the fetus through transplacental transfer until mid-gestation when the fetus begins to produce the hormones.[3] The placenta is permeable to thyrotropin releasing hormone, tetra-iodothyronine (T_4), and tri-iodothyronine (T_3), however, TSH does not cross the placenta. In fact, the fetal hypothalamic-pituitary-thyroid axis develops relatively independent of maternal influence,[4] and TSH production is determined by the thyroid hormone status. The maturation of negative feedback of thyroid hormone synthesis occurs by approximately mid-gestation, and elevated TSH concentrations were observed in infants as early as 2 weeks.[3] An abnormal TSH is usually the first indication of thyroid dysfunction. Hence, TSH screening for thyroid diseases has been recommended.[1,3] There is a growing interest in relative influence of maternal and fetal TSH on the growing fetus. Therefore, the objective of this study was to determine the correlation between maternal and fetal