Ectopic supernumerary kidney presenting as inguinal hernia

Supernumerary kidney is one of the least common forms of congenital renal abnormality and is usually discovered when it presents complications. The diagnosis of supernumerary kidney is confined to a mass of renal tissue that has no parenchymatous connection with the definitive kidney. The published literature on supernumerary kidney is scarce. Here, we report a case that presented as indirect inguinal hernia.

A 36 year old man suffering from chronic asthma presented with a painful swelling in the left inguinal region, which he first noticed one month previously. The swelling measured 3.0 × 4.0 cm, was situated on the medial aspect of the inguinal ligament, and was reducible with positive cough impulse. The scrotum on the left side was empty. An abdominal scan showed normal organs, including two normal kidneys. A diagnosis of indirect inguinal hernia with undescended testis (2.0 × 1.0 cm) and an oncocytoma and are recognised by their appearance has ultrastructural similarities to small filling defect in the bladder was also incidentally reported. Urinalysis revealed no abnormality. A rigid cystoscopy was performed. This revealed a raised swelling with normal appearance of the overlying mucosa, superior to the midpoint of the interureteric ridge. The lesion was approximately 1 × 1.5 cm in diameter. This was completely resected to muscle and sent for histology.

Histopathological examination revealed a neoplasm composed of large cuboidal and columnar cells, with abundant granular eosinophilic cytoplasm and large nuclei, with occasional macronucleoli (fig 1), which formed tubules, cords, and cribriform areas. The stroma was oedematous and contained lakes of mucin. The tissue stained negatively for prostate specific antigen and cytokeratin 20. Positive staining was seen for cytokeratin 7, synaptophysin (fig 2) and chromogranin. Therefore, the tumour was classified as a carcinoid tumour of the oncocytic variety.

The patient underwent a subsequent computed tomography scan of his chest and abdomen, and no other tumour sites were found. A 24 hour urine collection for 5-hydroxyindoleacetic acid did not suggest residual tumour.

Oncocytic carcinoid tumours are rare. They are a variant of carcinoid tumour whose appearance has ultrastructural similarities to an oncocytoma and are recognised by their abundant eosinophilic and granular cytoplasm. Oncocytic changes are often seen in the salivary and thyroid glands; they are thought to be of a degenerative nature. It has been suggested that the change within a carcinoid tumour may result from local environmental changes, possibly ischaemia.

References


Oncocytic carcinoid tumour of the bladder

We report the case of a 77 year old man who underwent an ultrasound scan for lower abdominal pain. He had no significant medical history. The scan identified a small inguinal hernia, explaining his discomfort. A
Previous reports of these tumours have been in the lung,2 nasopharynx, thymus, and in one report, the kidney.3 The tumours are malignant and capable of metastatic spread. They can also result in a carcinoid syndrome, so that full resection is recommended.

This case is worthy of note in view of its rarity. Carcinoid tumours of the bladder have been reported sporadically, but this is the first report of an oncotypic type. The appearance of the tumour was somewhat innocuous, but early excision biopsy averted potentially more serious consequences later on.

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References


Endothelial progenitor cells in non-small cell lung cancer

We read with interest the article by Hilbe et al concerning the contribution of endothelial progenitor cells (EPCs) to the vasculature in non-small cell lung cancer (NSCLC).1 In their study, the authors conclude that “increased numbers of CD34 positive EPCs can be found in NSCLC tissue and these cells seem to contribute to the formation of capillaries”. Although it is interesting and worthy of further study, in our view, the evidence presented in their paper is unconvincing. However, the problems are not apparent to readers unfamiliar with the background or rigorous examination.

In our view, there are three problems with the arguments put forward by Hilbe et al. First, the presumed localisation of EPCs on serial frozen sections is not convincing because neither multiple microvessel labelling for CD34 and EC markers nor immunoelectron microscopical examination was performed. Because the cellular boundaries cannot be seen in the figures provided, it is unclear what types of cells are CD34+.

Second, CD34 is not exclusively expressed on early—but not circulating or committed—EPCs. In addition to being expressed on haemopoietic stem cells, CD34 also serves as a marker for non-haemopoietic progenitor cells, such as neural crest cells, embryonic stem cell lines, and adult stem cells with a pluripotent differentiation capacity.1 Furthermore, CD34 was found to be expressed on tumour cells of epithelial origin.3 The possibility that the CD34+ cells in the NSCLC tissue are not ECs was not explored.

Third, a convincing argument for the presence of EPCs in the NSCLC tissue depends on the unequivocal identification of this cell type. However, Hilbe et al did not use more than one early stem cell marker to detect EPCs. Their method differs from several earlier studies that used different antibody combinations.

The involvement of alternative vascularisation mechanisms—including vasculogenesis—in the tumour blood supply has broad biological and medical importance. We found the message emerging from the Hilbe study a valuable contribution to our knowledge of the vasculogenesis in tumour tissue. Our critical comments are intended simply as a reminder that the extent of these phenomena is still unclear, and can only be determined by rigorous examination.

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References


Monckeberg medial calcific sclerosis mimicking malignant calcification pattern at mammography

Monckeberg medial calcific sclerosis (MCS) is a ring-like calcification of the vascular media of small to medium sized vessels without associated intimal thickening. Almost exclusively, MCS is the underlying condition in what is referred to as breast arterial calcification (BAC) detected at mammography. BAC is a relatively common finding. The classic radiographic pattern of BAC is the “railroad track” pattern, which appears as linear parallel calcifications, and is a reflection of the circumferential pattern of calcification in MCS; it is easily interpreted as benign.

We recently encountered an atypical microcalcification pattern of MCS mimicking malignancy in a 64 year old woman with no history of breast cancer. There was no history of breast trauma or surgery, renal disease, or parathyroid problems. The patient had non-insulin dependent diabetes mellitus. Coronary artery disease was present as identified by an episode of retrosternal chest pain and a stress test showed ST segment elevation in the electrocardiogram. No palpable abnormalities were present in the breast or axilla.

This atypical pattern was present together with popcorn-like calcification of a hyalinised fibrodenoma and typical benign microcalcifications. The atypical calcification was present in a medium to high density clustered calcifications in a curved and branching pattern. This pattern is usually caused by calcium phosphate, and is typically associated with malignancy, compared with low density amorphous calcifications, which is caused by calcium oxalate, and are associated with benign conditions.1

Wire localised excision of the clustered calcifications was performed and subsequent radiographs showed that suspicious microcalcification clusters were included in a block. Sections corresponding to suspicious microcalcifications had Monckeberg medial calcific sclerosis in small to medium sized vessels. These were both ring-like classic circumferential areas of calcification and discontinuous calcification foci in arterial media.
This atypical pattern posing a diagnostic dilemma requires exclusion for histopathological assessment. It has been reported earlier, and is probably caused by non-circumferential and discontinuous foci of calcification in the vascular media; these calcific microliths are probably seen in the early stages of development of MCS, and may mimic linear, curved, or branching patterns of microcalcification clusters indicating malignancy. The pathogenesis of MCS/BAC is thought to be related to several factors, including age-related change, diabetes mellitus, chronic renal failure, and coronary artery disease. Pecchi et al. showed that the presence and severity of BAC strongly correlated with the extent of coronary atherosclerosis, as determined by the amount of coronary calcium detected by multislice computed tomography, and BAC may indeed be a surrogate marker of coronary artery disease. Although coronary artery disease is almost always the result of intimal atherosclerosis, a disease different from MCS, the association may be reflective of shared pathways of calcium deposition.

In summary, this report highlights the atypical calcification pattern of Monckeberg medial calcific sclerosis mimicking malignant calcifications in breast requiring exclusion for diagnosis. This benign vascular calcification may also be a marker of coronary artery disease.

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References

Extramural haemopoiesis
Extramural haemopoiesis usually occurs in association with haematological disorders—in particular, myelobrosis—and normally occurs in the reticulo-endothelial system, involving the liver, spleen, and lymph nodes. The eyes and other organs are less often affected. In addition, single lineage haemopoiesis may occur, although it does not usually form mass-like lesions. This report describes a focus of erythropoiesis occurring in a renal cell carcinoma. A 55 year old woman underwent a right radical nephrectomy and the specimen measured 30 x 6 x 6 cm. A 2.5 x 2.0 x 2.0 cm circumscibed nodule was present. Microscopic examination showed a clear cell renal carcinoma, nuclear grade 2, with central cystic degeneration. A single, extremely small focus of erythropoiesis was present within a central small capillary, consisting of approximately 20 nucleated red blood cells (fig 1). A preoperative haemoglobin concentration was normal, at 132 g/litre (normal range, 115–185). Extramedullary haemopoiesis has been reported in the kidneys, usually associated with idiopathic myelofibrosis. A renal cell carcinoma associated with a perirenal liposarcoma and extramedullary haemopoiesis has been documented. A superficial, spindle cell lipoma from the neck with extramural erythropoiesis has also been reported. Extramedullary haemopoiesis also occurs in hepatic angiomylipoma (but not in renal angiomylipoma) and in other hepatic tumours, an occurrence thought to be related to the hepatic sinusoidal endothelium. Foci of haemopoiesis or erythropoiesis have been described adjacent to recent, acute myocardial infarcts, thought to be a manifestation of altered cytokine production. Isolated megakaryocytes are a normal occurrence in the capillaries of the lung. They have been cited to occur in sentinel lymph nodes, although in lymph nodes they are usually present as part of microscopic foci of erythropoiesis and granulopoiesis. Extramedullary haemopoiesis usually occurs in tissues with a milieu that supports the proliferation of primitive haemopoietic bone marrow elements. Filtration of clonogenic bone marrow cells within supportive tissues is one pathogenetic mechanism considered in the pathogenesis of extramedullary haemopoiesis, whereas the migratory nature of megakaryocytes may explain their presence in aberrant sites in the absence of extramedullary haemopoiesis. Although this case may represent a transitory erythropoietic focus, a rare erythropoietin induced occurrence of erythropoiesis within a renal cell carcinoma is perhaps a more plausible explanation. Although it has been reported that 74% of renal cell carcinomas show strong erythropoietin immunolocalisation, foci of associated erythropoiesis appear to be unusual.

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References

Figure 1 (A) Low power appearance of renal cell carcinoma with focus of erythropoiesis. (B) Intracapillary erythropoiesis.

Full details of events to be included should be sent to Maggie Butler, Technical Editor JCP, The Cedars, 36 Queen Street, Castle Hedingham, Essex CO9 3HA, UK; email: maggie.butler2@btopenworld.com

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Practical Pulmonary Pathology
26–29 July 2005, Royal Brompton Hospital, London, UK
Further details: Professor B Corrin, Brompton Hospital, London SW3 6NP, UK. (Fax +44 (0)20 7 351 8293; e-mail b.corrin@ic.ac.uk)

Association of Clinical Pathologists’ National Scientific Meeting
16–17 June 2005, Royal College of Physicians, London, UK
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