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 confounded 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 x 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 left testis was made and the patient underwent surgery. The hernial sac included an atrophic testis (2.0 x 1.5 x 1.0 cm) and another small bean shaped mass (3.0 x 2.0 x 2.0 cm), which was studied histopathologically.

The testis showed a positive string sign on the cut surface. The other mass had a grey-white cut surface, with an intact capsule. Sections of the testis showed seminiferous tubules with thickened basement membranes containing inactive germ cells and some having only Sertoli cells. These features were consistent with cryptorchid testis. Sections from the bean shaped mass showed a kidney-type structure, with cortex and medulla. The glomeruli and tubules were seen in their developmental stages, with immature mesenchymal tissue interspersed in between (fig 1). These features were consistent with a diagnosis of supernumerary kidney because the patient had two normal kidneys in the abdomen.

A supernumerary kidney is a rare congenital anomaly. About 70 such cases have been reported in the international literature. However, to the best of our knowledge, this is the first reported case of a supernumerary kidney in an inguinal hernia. The kidney usually lies within the renal fascia, caudal to the normal organ. Because the kidney was located below the second lumbar vertebra in this case, we prefer to classify it as an ectopic supernumerary kidney.

In humans, the metanephros or permanent kidney begins to develop early in the 5th week of intrauterine life. It arises from two different sources: the renal blastema, which gives rise to the renal cortex and medulla, and the ureteric bud, which gives rise to the collecting system. The metanephros begins to differentiate from the caudal end of the nephrogenic cord (metanephric mesoderm), which is thought to be caused by the formation of two ureteric buds arising from different positions in the uroblast, which reach the metanephric blastema at such divergent positions that aberrant divisions result in two kidneys on one side. The supernumerary kidney is smaller, can be hypoplastic, and is usually well organised histologically. This case has been reported for its rarity and also because of its unusual presentation with cryptorchid testis as inguinal hernia.

V Kusuma, M Hemalata, B V Suguna
Department of Pathology, Kempegowda Institute of Medical Sciences (KIMS), VV Puram, Bangalore 560004, India; jreddy@nimhans.kar.nic.in

References

Oncocytic carcinoma 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 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 x 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 cytookeratin 20. Positive staining was seen for cytookeratin 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 carcinoma 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.

Figure 1 Haematoxylin and eosin stain of the neoplastic tissue (original magnification, ×50).

Figure 2 Tissue stained positive for synaptophysin (original magnification, ×50).
Previous reports of these tumours have been in the lung,4 nasopharynx, thymus, and in one report, the kidney.5 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 oncocytic type. The appearance of the tumour was somewhat innocuous, but early excision biopsy averted potentially more serious consequences later on.

Acknowledgement

Dr P Hamden (St James University Hospital, Leeds, UK) reviewed the pathological findings.

E 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 cells” 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 diagnostically unconvincing. However, the problems are not apparent to readers unfamiliar with the background or pitfalls of this specialised topic.

The development of a vascular network plays a crucial role in the development and function of normal tissues and organs, in addition to tumour growth and metastasis. Understanding how tumours acquire their vasculature is indispensable for developing novel therapeutic approaches. However, the vascularisation of tumours is very complex, consisting of sprouting, vessel cooption, glomeruloid angiogenesis, mosaic vessel formation, vascular mimicry, and intussusceptive angiogenesis.1–3 Furthermore, there is emerging evidence that putative angioblasts, also known as EPCs, might persist in adult life and contribute to the vascularisation of tumours.4–6 EPCs have been isolated from peripheral blood and bone marrow. Similar to embryonic angioblasts, EPCs have the capacity to proliferate and differentiate into mature endothelial cells (ECs). To date, no clear definition exists as to when an EPC turns into a mature, fully differentiated endothelial cell in vivo. Early EPCs (localised in the bone marrow or immediately after migration into the circulation) are CD133+/CD34+/VEGF-R2+/VEGFR-2+ (vascular endothelial growth factor receptor 2 positive) cells, whereas circulating EPCs are positive for CD34/VEGFR-2/CD331/Vcadherin, lose CD133, and begin to express von Willebrand factor. In general, it is widely accepted that the loss of CD133 reflects the transformation of early circulating EPCs into more mature endothelial-like cells.7–9 Hilbe et al identified early EPCs by CD133 labelling not in peripheral blood or bone marrow, but in the endothelial tubes of NSCLC tissue. The key evidence for their identity came from immunohistochemical studies.7

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 labeling for CD33 and EC markers nor immunoelectron microscopic examination was performed. Because the cellular boundaries cannot be seen in the figures provided, it is unclear what types of cells are CD133+. Second, CD133 is not exclusively expressed on early—but not circulating or committed—EPCs. In addition to being expressed on haemopoietic stem cells, CD133 also serves as a marker for non-haemopoietic progenitor cells, such as neural stem cells, embryonic stem cell lines, and adult stem cells with a pluripotent differentiation capacity.7 Furthermore, CD133 was found to be expressed on tumour cells of epithelial origin.5 The possibility that the CD133+ 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, why 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.

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 detected on routine mammography. She had no risk factors for breast cancer. There was no history of breast trauma or surgery, renal disease, or parathyroid problems. The patient had no insidious signs and symptoms, such as a 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 fibroendotheloma and typical benign microcalcifications. The atypical calcification was present in a medium to high density clustered calcification pattern in a medium to high density clustered pattern in a medium to high density clustered pattern, and was situated within a medium to high density clustered pattern. This pattern is usually caused by calcium phosphate, and is typically associated with malignancy, compared with low density amorphous calcifications, which are caused by calcium oxalate, and are associated with benign conditions.1–3

Wire localised excision of the clustered calcifications was performed and the specimen 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.

References


This atypical pattern, though a diagnostic dilemma, requires further histopathological assessment. It has been termed as an atypia by several authors, including Lehtokumpu, Härkönen, and Räsänen, who described an atypical pattern of calcium deposition in the pulmonary arteries. The atypical pattern may be reflective of the underlying pathogenic mechanism.

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

A Saxena
Department of Pathology, Royal University Hospital, 103 Hospital Drive, Saskatoon, SK, S7N 0W8 Canada; saxena@skwash.ka.ca

I C Waddell
Department of Radiology, Victoria Hospital, Prince Albert, Saskatchewan, S9V 3T4 Canada

R W Friesen
Department of Surgery, Victoria Hospital

R T Michalski
Department of Pathology, Victoria Hospital

References


Extramedullary haemopoiesis

Extramedullary haemopoiesis usually occurs in association with haematological disorders—in particular, myelofibrosis—and normally occurs in the reticulo-endothelial system, involving the liver, spleen, and lymph nodes. The myocardium 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 59-year-old woman underwent a right radical nephrectomy and the specimen measured 30 × 6 × 6 cm. A 2.5 × 2.0 × 2.0 cm circumscribed 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. 3,4 A renal cell carcinoma associated with a perirenal liposarcoma and extramedullary haemopoiesis has been documented.3 A superficial, spindle cell lipoma from the neck with extramedullary erythropoiesis has also been reported.4 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.5 Foci of haemopoiesis or erythropoiesis have been described adjacent to recent, acute myocardial infarcts, thought to be a manifestation of altered cytokine production.6 Isolated megakaryocytes are a normal occurrence in the capillaries of the lung.7 They have been cited to occur in sentinel lymph nodes,8 although in lymph nodes they are usually present as part of a 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,9 foci of associated erythropoiesis appear to be unusual.

J D Coyne
Wythenshawe Hospital, Wythenshawe, Manchester M20 8UR, UK; johnncoyne@doctors.org.uk

References


CALENDAR OF EVENTS

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

Diagnostic Histopathology of Breast Disease
9–13 May 2005, Hammersmith Hospital and Imperial College, London, UK
Further details: Wolfson Conference Centre, Hammersmith Hospital, Du Cane Road, London W12 ONN, UK. (Tel +44 (0)20 8383 3117/3227/3245; Fax +44 (0)20 8383 3248; e-mail wcc@ic.ac.uk)

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 7351 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
Further details: ACP Central Office, 189 Dyke Road, Hove BN3 1TL, UK. (Tel +44 (0)1273 775700; e-mail info@pathologists.org.uk)

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