

given to prevent possible new extramedullary haemopoiesis.

**Pier Paolo Piccaluga, Carlo Finelli, Ernesto Vigna, Claudio Agostinelli, Francesco Bacci, Stefania Padini, Cristina Papayannidis, Claudio Laterza, Giovanni Martinelli, Stefano A Pileri, Michele Baccarani**

Institute of Haematology and Medical Oncology "L and A Seragnoli", University of Bologna, Bologna, Italy

Correspondence to: Dr Pier Paolo Piccaluga, Institute of Haematology and Medical Oncology "L and A Seragnoli", S Orsola Malpighi Hospital, University of Bologna, Via Massarenti, 9-40138 Bologna, Italy; [ppicca@med.unibo.it](mailto:ppicca@med.unibo.it)

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## Obesity-related glomerulopathy: another nail in the coffin of the epidemic of end-stage renal disease

The clustering of insulin resistance, dysglycaemia, dyslipidaemia, hypertension and central obesity represents the major features of metabolic syndrome. These clusters of factors may share common aetiology, each of which is a risk factor for cardiovascular disease. The metabolic syndrome seems to affect between 10% and 25% of adult populations worldwide. Several studies have described the association between metabolic syndrome, and diabetes and cardiovascular disease.<sup>1</sup> Although obesity is often associated with diabetes and hypertension, which are two of the most common risk factors for the development of end-stage renal disease (ESRD), it has been suggested that obesity in itself is an independent risk factor.

The prevalence of obesity-related glomerulopathy (ORG), which may lead to end-stage renal disease, has increased 10-fold over the past 15 years as a consequence of the spread of the obesity epidemic. The increasing prevalence of ESRD, with its associated high annual mortality and rates of cardiovascular complications, is a worldwide problem.<sup>2</sup> In the US alone, the prevalence of ESRD has more than doubled in the past decade and the population living with ESRD is projected to increase to 650 000 persons by the year 2010, with associated Medicare expenditures of \$28 billion.<sup>3</sup> Identifying new and potentially modifiable risk factors for ESRD is critical in order to devise effective, population-based preventive strategies.

Massive obesity has been shown to produce nephrotic syndrome, and it has been reported that proteinuria and segmental glomerulosclerosis can be present in obese patients, even in the absence of diabetes.<sup>2</sup> In addition, a large-scale study including 6818 renal biopsies from 1986 to 2000 showed a 10-fold increase in renal lesion, such as glomerulomegaly and focal segmental glomerulosclerosis, which were associated with obesity.<sup>2</sup> ORG was recently defined morphologically as glomerulomegaly with or

without focal segmental glomerulosclerosis. The syndrome constitutes the triad of morbid obesity, marked proteinuria without oedema and normal serum albumin concentration. It can occur in any degree of obesity but is more common in the morbidly obese group—that is, body mass index >40 kg/m<sup>2</sup>. It often presents as proteinuria on urinary dipsticks, followed by the confirmation of gross proteinuria of up to 32 g/dl. ORG should be diagnosed by excluding the presence of hypertension or undetected type 2 diabetic renal diseases.<sup>2</sup> The pathogenesis is unknown (thought to be due to low renal nitric oxide production) and most of the available information comes from studies in Zucker fa/fa rats which often die from ESRD. These rats are a genetic model of obesity that results from a mutation in the leptin receptor gene.<sup>4</sup> Homozygous Zucker fa/fa rats exhibit most of the metabolic picture seen in human obesity, including hypercholesterolaemia, hypertriglyceridaemia, hyperinsulinaemia and proteinuria.

Although the condition is said to be benign, a small proportion of patients will progress towards end-stage renal failure requiring replacement therapy, a further addition to the cost. The condition is readily reversible and ameliorated with weight loss, an important consideration in the management of this condition. In association with increasing prevalence of chronic kidney disease, this will inevitably result in increasing proportions of deaths from cardiovascular disease as well as increased prevalence and associated consequences of other complications of chronic kidney disease. A concerted, global initiative is required to deal with the ORG epidemic.

**Mohamed H Ahmed**

Department of Chemical Pathology, Southampton General Hospital, Southampton, UK

**Atif A Khalil**

Department of Nephrology, Royal Liverpool University Hospital, Liverpool, UK

Correspondence to: Dr M H Ahmed, Department of Chemical Pathology, Mail point 6, Level D South Academic Block, Southampton General Hospital, Tremona Road, Southampton SO16 6YD, UK; [elziber@yahoo.com](mailto:elziber@yahoo.com)

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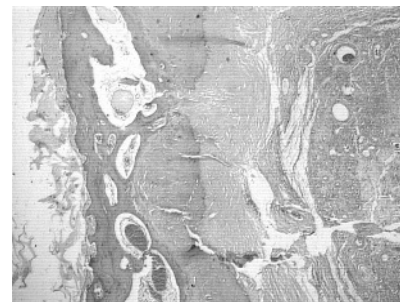
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## Osseous metaplasia in ovarian tumours: a case with serous cystadenoma

In a recent paper in the *Journal of Clinical Pathology*, Godbole *et al*<sup>1</sup> reported a case of osseous metaplasia in a benign ovarian cyst in association with cloacal anomaly. These



**Figure 1** The cyst wall is lined a single layer of cells and contains abundant osseous tissue. Ovarian stroma is seen on the right part of the picture. Original magnification,  $\times 25$ .

authors stated that only four published cases with osseous metaplasia in ovarian tumours exist, three in the literature,<sup>2–4</sup> and the one they presented. The former three cases were associated with papillary serous carcinoma, thecoma and endometrioma in a supernumerary ovary, respectively. According to their data, Godbole *et al* concluded that osseous metaplasia is more common in anatomically abnormal ovarian tissue in women of reproductive age. However, a thorough search we made in the literature revealed that to date, the total number of cases with osseous metaplasia in an ovarian tumour reported in the literature has reached 10,<sup>1–10</sup> (one of them<sup>5</sup> was actually added after the report of Godbole *et al*, whereas one more case is described herein).

Our case concerns a 37-year-old woman who was subjected to hysterectomy with the right adnexa, owing to multiple leiomyomas. The right ovary measured 7 cm in diameter, and presented an osseous lesion that was hard in consistency and 3 cm in diameter. Histologic examination, after decalcification, showed a serous cystadenoma with extensive, circumferential osseous metaplasia of its wall (fig 1). The cyst wall was lined by a single layer of flattened or cuboidal cells, whereas the osseous tissue consisted of mature lamellar bone. The ovary was searched thoroughly to exclude the possibility of an underlying teratoma. The rest of the pathology of the present specimen revealed only the aforementioned uterine leiomyomas. This is the first reported case where osseous metaplasia has occurred in a benign ovarian serous cystadenoma.

Bone formation in the ovary, with the exception of developing in the setting of a mature cystic teratoma or a heterologous mixed mesodermal tumour, is exceedingly uncommon. Excluding the previous cases, so far, the ovarian neoplasms most commonly associated with osseous metaplasia are mucinous cystadenomas (three cases),<sup>6–8</sup> followed by serous cystadenocarcinomas (two cases).<sup>2,9</sup> A minority only seems to occur in anatomically abnormal ovaries (two cases). The most plausible explanation for bone formation in an ovarian neoplasm is a metaplastic process of the multipotential stromal cell. No prognostic significance has been attributed to this unusual phenomenon.

**D Miliaras**

Laboratory of Histology & Embryology, Medical School, Aristotle University of Thessaloniki, Greece

**M Ketikidou**

Department of Pathology, National Security Foundation (IKA), Thessaloniki, Greece



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Mohamed H Ahmed and Atif A Khalil

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