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Incidence, aetiology and epidemiology of uterine fibroids

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Uterine fibroids are the most common benign tumour of the female genital tract. However, their true prevalence is probably under-estimated, as the incidence at histology is more than double the clinical incidence. Recent longitudinal studies have estimated that the lifetime risk of fibroids in a woman over the age of 45 years is more than 60%, with incidence higher in blacks than in whites. The cause of fibroids remains unclear and their biology poorly understood. No single candidate gene has been detected for commonly occurring uterine fibroids. However, the occurrence of rare uterine fibroid syndromes, such as multiple cutaneous and uterine leiomyomatosis, has been traced to the gene that codes for the mitochondrial enzyme, fumarate hydratase. Cytogenetic abnormalities, particularly deletions of chromosome 7, which are found in up to 50% of fibroid specimens, seem to be secondary rather than primary events, and investigations into the role of tumour suppressor genes have yielded conflicting results. The key regulators of fibroid growth are ovarian steroids, both oestrogen and progestogen, growth factors and angiogenesis, and the process of apoptosis. Black race, heredity, nulliparity, obesity, polycystic ovary syndrome, diabetes and hypertension are associated with increased risk of fibroids, and there is emerging evidence that familial predisposition to fibroids is associated with a distinct pattern of clinical and molecular features compared with fibroids in families without this prevalence.

Key words: fibroids; familiality; growth factors; genes; lifetime risk.

First described in 1793 by Matthew Baillie of St George's Hospital, London, uterine fibroids are the most common benign tumour in women, being clinically apparent in up to 25% of all women[1] and up to 30–40% of women over 40 years of age. However, a large proportion of fibroids remain undiagnosed since most are asymptomatic. The aetiology of uterine fibroids and their biology are poorly understood. Several risk factors have been identified, such as ethnicity, nulliparity, genetics and hormonal factors, yet little is known regarding the reasons for the heterogeneity in behaviour of fibroids and the symptoms attributable to them. Why is it that fibroids are solitary, small and

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slow growing in some women, and multiple, large and fast growing in other women? Why is it that there is a prevalence of uterine fibroids in some families but not in others? Traditional teaching is being updated, and molecular research is continually improving our understanding of the epidemiology of this debilitating condition which has significant morbidity and socio-economic impact.

INCIDENCE

The population incidence of uterine fibroids is not known because most studies have evaluated the incidence in women seeking medical help. Reports based on clinical diagnosis or diagnostic tests underestimate the true incidence, since they only relate to women with symptoms or those who come into contact with health professionals. On the other hand, reports based on histology alone may overestimate the true incidence as they relate to women with symptoms in whom non-surgical treatments have failed, most of whom may therefore have fibroids. In one such study which included histological assessment of hysterectomy specimens, the incidence of uterine fibroids was 33% based on clinical assessment, 50% based on ultrasound and 77% based on histological assessment.[2] The reported incidence therefore ranges from 30% to 70% in premenopausal women and increases with increasing age.[3] A population-based study of members of an urban health plan aged 35–49 years, selected at random, reported uterine fibroids in 59% of Black women and 43% of White women with no previous diagnosis of fibroids, with the incidence reaching over 75% and 65%, respectively, in those aged 45 years or more.[4] This study estimated that most women have a lifetime risk of developing uterine fibroids of nearly 70%. However, many of these studies were cross-sectional and whilst they confirm the incidence at any point in time, they do not offer any insight into the trends of diagnosis with age, in contrast with longitudinal studies. Studies with a cohort design also have the limitation that participants are not usually screened at entry.

An alternative method of reporting incidence is to assess the rates of new diagnoses per year in women followed-up in longitudinal observational cohort studies such as the Nurses Health Study or the Black Women's Health Study in the USA, with reported rates of 12.7 per 1000 woman-years and 29.7 per 1000 woman-years, respectively.[3,5]

AETIOLOGY

Although the aetiology of uterine fibroids is unknown, there have been some major advances in understanding of the pathogenesis of this condition. As with all tumours, there are two distinct stages in development: transformation from a normal to an abnormal cell; and growth and proliferation of the abnormal cells. Although fibroids may be multiple or solitary in a woman's uterus, studies have established that, irrespective of size, each fibroid develops from one single cell (the monoclonal development of fibroids concept). In hysterectomy specimens from women with multiple fibroids, glucose-6-phosphate dehydrogenase iso-enzyme (A or B) activity was identical in each cell of a particular fibroid tumour, exhibiting a single electrophoretic pattern even when as many as eight samples from the same tumour were analysed.[6] This has been confirmed using polymerase chain reaction involving the differential inactivation of X-linked phosphoglycerol-kinase gene from fibroid specimens.[7] All the cells from each fibroid tumour were found to have a single type of active allele, again

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