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Fifty years of woodworkers' nasal adenocarcinoma in High Wycombe

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Abstract

Background. Since adenocarcinoma of the ethmoid sinuses was first described as an occupational disease in the woodworkers of High Wycombe, over the 50 subsequent years there has been a gradual decrease in the numbers to none over the last 12 years. Although this mirrored the decline in local industry, it seems the causative factor was first seen and then disappears over a 50-year period.

Methods. A total of 146 cases have been traced historically over this time as well as personal experience of 33 cases; these cases are reviewed and success with a new modality of treatment is discussed.

Conclusion. This paper outlines how the disease was initially recognised, both its diagnosis and treatment development. It also describes how both the appearance and disappearance were seemingly caused by changes in manufacturing practice.

Main Article

Mr J W R Capper takes responsibility for the integrity of the content of the paper

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Introduction

In 1968, Esme Hadfield, consultant ENT surgeon in High Wycombe, presented a paper to the Royal Society of Medicine, which showed that workers in the hardwood industry of High Wycombe and the surrounding areas had a very high incidence of adenocarcinoma of the ethmoid sinuses.[1] She became aware of this association for the first time in 1964, taking time to substantiate it, before presenting at the Royal Society of Medicine and writing subsequent papers in the *British Medical Journal*, *The Lancet* and elsewhere.[2–5] She was honoured with a Hunterian Lectureship in 1969.[6] After a distinguished career, she retired in 1986 and I was fortunate enough to succeed her.[6]

In my first year in post, I saw five new cases of this disease. Bearing in mind that most clinicians in the UK were likely to see as few as two cases in their 'clinical life-time', this was somewhat unusual. Hadfield had described the disease and I was very keen to find an effective treatment. Little did I know that, in time, the disease was to disappear, in a similar manner to its original appearance.

Background

Historically, High Wycombe, which has good transport links to London, was surrounded by some beautiful beech tree woods, many still present today. This wood was utilised in the manufacture of chairs, and the town became known as the centre of chair-making in England,[7] producing, at one stage (circa 1877), 5000 chairs a day. Originally made partly in the woods themselves by 'bodgers' (turners who made chairs of beech wood), chair production moved in to small factories close to the railway, and then to intensive factories at the beginning of the twentieth century. The concentrated space and subsequent 'new and improved' methods of manufacture were to prove very hazardous to the workers themselves.

Hardwood dust is very fine, with exceedingly small particles of around 5 μ; in comparison, so-called softwood particles are very coarse and heavy. Hardwood dust was so fine that it was even used to bulk out paint and, because of their highly incendiary nature, used in fireworks.

Dust is produced in purely manual work. Since high-speed machinery was introduced, in the 1920s and 1930s, the dust, particularly from sanding, was everywhere in factories, piling up in any corner as well as under and over the machinery.

There was a progression of improved mechanisation, starting, in the 1920s, with the use of belt drives, then electric motors with the formation of the National Grid in 1935, until, certainly the most aggressive technology, pneumatic and hydraulic feeds in the 1940s. The level of dust extraction or dissipation in no way kept up with these developments. The increased dust levels acted as an extreme fire risk but, more importantly, the choking levels of dust suspended in the air were inhaled by the workers with the nose trapping the dust.

If you were to examine a woodworker, in their workplace or at the end of a shift, even in the late 1980s, you could see a large deposit of dust, particularly on the anterior end of the middle turbinate. It is this dust, possibly containing a more specific heat product, that

seems to be either the causative factor or an associated agent or promoter of adenocarcinoma, with the disease usually presenting years after initial exposure.

Hadfield and others showed squamous metaplasia and decreased mucociliary transport of the nasal lining as a result of contact with the dust.[8] However, unlike blue asbestos fibres in the lung found in a pre-malignant form, there has never been a demonstration of long-term deposits of dust in the nasal tissue or evidence of any pre-malignant histological change in the woodworkers' noses. In my time, any woodworker seen in the clinics or having any routine nasal surgery was carefully examined with biopsies where possible. For a limited time, when past employment as a woodworker was known, ethmoid specimens were taken by myself during coincidental post-mortems. No evidence of any pre-malignant change was found as a result of this, or following examination in routine clinics or by means of the nasal survey (see below).

The glues, polishes, solvents or constituents of sandpaper seem not to be relevant, nor does exposure to exotic or unusual wood types. All cases occurred in patients who had been exposed to the local beech wood, many exclusively.

Presentation

Once the risks were known, Esme Hadfield and Ronald McBeth set up a nasal survey, with help and sponsorship from the British Furniture Manufacturers Association, to examine, once a year, all the current workers and 'pensioners' of their firms in High Wycombe. This resulted in increased awareness amongst the workers and the general practitioners. Almost all cases, in my experience, presented from general practice referrals rather than the survey.

A unilateral blocked nasal passage, often with serosanguinous discharge, was the presenting symptom of adenocarcinoma of the ethmoid sinuses, with an obvious fleshy, often dark or haemorrhagic polyp protruding from under the middle turbinate where this had not been destroyed. In later stages, extension occurred superiorly and laterally towards the brain and eye, with, ultimately, central nervous system involvement that proved fatal.

The survey, as well as raising awareness of this disease, aimed to identify any possible contributing cause, such as: snuff-taking (common because of the fire risk), a deviated septum affecting laterality, specific trades or exposure to different woods where subsequent malignancy occurred. There was no common factor other than exposure to hardwood dust inside the factory itself. The disease did not occur in external workers, such as timber yard sawyers or kiln workers only exposed to wet wood and no real fine dust. It occurred in every trade within the factory, such as French polishers, sanders, inside sawyers, lathe workers, and in one person whose job it was to gather the dust every day and take it to the nearby paint factory.

There was a long latency of disease onset between the dust exposure and disease presentation. This was very evident in two patients in my time. Their exposure to dust was for 2 and 5 years respectively; these individuals then both left the industry and presented with a tumour 30 and 25 years later.[9] This latency in disease onset has been remarked on in other papers.[10] Interestingly, exposure does not need to be prolonged, and the disease can develop when away from the promoter or causative agent. Furthermore, when examining the natural history of both the earliest and the last cases to present with the disease, the latency sheds a very different view on the chronology.

Materials and methods

A total of 146 new cases with accompanying clinical notes were assessed from 1950 to the present day, all in High Wycombe or the surrounding area. Hadfield collected data from 41 cases prior to her confirmed interest in 1965 (she was appointed in 1956), and so earlier cases could have been underreported where case notes were not available. From 1986 to the present day, a total of 33 cases presented.

From this group of cases, some facts emerge. Firstly, there was no evidence of distant metastasis in any of the patients, with death occurring by local invasion of the frontal lobes. Secondly, the age of presentation increased with time, with several patients aged in their fifties in the earlier groups, with age then gradually rising, particularly in the last three decades. However, the most important finding is the steep reduction of new cases presenting from 1996 to 2006, with no cases presenting in the last 12 years.

Looking at the numbers, five new cases per year was common in the 1960s (Figure 1). As this is such an unusual disease, it seems unlikely that the incidence had been missed by clinicians prior to Hadfield, nor had the disease entered the folklore of the chair-making community, particularly the typical frog-eyed appearance of a terminal untreated case.

Results

Treatment during Hadfield's time varied a great deal. Her view (as expressed to me) was that if a patient had the disease they would die with it or, more likely, from it. She had tried radiotherapy, both external beam and implants. She had used various surgical approaches, ranging from external nasal, transantral to transpalatal approaches, with little success. Radical surgery combined with orbital exenteration was attempted, and even local iridium implants. Hadfield seemed to accept that any clearance resulted in incomplete removal of microscopic amounts of the disease, which subsequently returned slowly but surely, ultimately leading to death. The logical principal of surgical margins in this area was very hard to achieve.

Hadfield did persuade the Department of Health to accept the disease as an industrial disease (Industrial Injuries Disablement Benefit disease code D6) and therefore compensable with a pension. This was some comfort to the patients with what was almost a death sentence.

In 1986, the radiotherapy dose was increased to 5500 rads (from 2000 rads) following aggressive debulking. On recurrence, salvage surgery in the form of craniofacial resection was performed, with the help of Tony Cheeseman, at Charing Cross Hospital. The results were not very good, although there were some successes – of the 15 patients treated with a view to cure, only 1 out of 7 undergoing craniofacial resection after failed radiotherapy, 6 after radiotherapy alone, and 1 who had complete local removal of a very small lesion and then a lateral rhinotomy, were free of disease. In other words, 8 out of 15 patients appeared cured, dying of other causes. Three patients underwent no treatment because of other morbidities, and died soon after presentation.

The three biggest problems were: (1) orbital complications secondary to the radiotherapy (a dry painful red eye that could be quite severe); (2) the difficulty of identifying recurrence at an early stage; and (3) the apparent increase in rapid spread into the frontal lobe with any further recurrence following craniofacial resection with a graft to replace the cranial floor.

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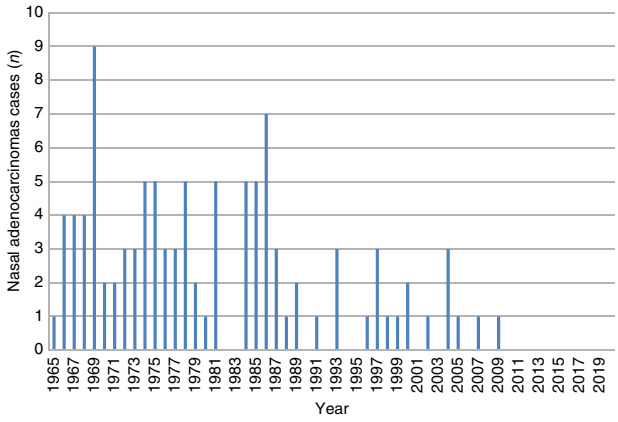

Fig. 1. The numbers of new diagnoses of nasal adenocarcinomas in woodworkers in the High Wycombe area between 1965 and 2020.

Combined with this, there was no room for sensible margins of resection, despite better visualisation.

After 10 years of this protocol, Paul Knegt, at the Royal Society of Medicine (where Hadfield had presented her initial findings 20 years earlier) presented his series of nasal cancer cases.[11,12] His cases included a large number of adenocarcinomas. After learning of Sato's initial work in Japan,[13] Knegt undertook radical clearance of the nose; this consisted of clearing the medial wall of the nose, the sinus walls to their lateral margins and to the eye and bony roof of the nose via a transantral approach, and then sterilising the remaining cavity with 5 per cent fluorouracil (5-FU) cream (Efudex; Valeant Pharmaceuticals, Aliso Viejo, California, USA) to mop up any remaining tumour cells. This was coupled with regular debridement and repeated 5-FU application over six weeks.

This treatment seemed to avoid the side-effects of the radiotherapy on the eye, on what was a fairly radio-resistant tumour, leaving this modality available as a future treatment if necessary. It also opened up the whole area where tumour might recur to direct inspection. Resection occurred at the margins of visible tumours, sparing the orbit when, in the past, it might have been sacrificed. The problem of adequate tumour margins was countered by the 5-FU, which sterilised the resection margins. Finally, the skull base was left intact where possible, with the aim of further delaying invasion of the meninges and brain.

Starting in 1996, all patients underwent this treatment over the next decade, with hugely gratifying results. Of the patients treated by radiotherapy and salvage surgery, 7 out of 15 died from disease; in contrast, only 1 out of 11 died from disease in the 5-FU group.[14]

However, the treatment had its own problems, despite the results. The cavities crusted badly for some time, often taking a year or more to epithelialise. Although the crusting eventually settled, regular removal and inspection was undertaken, usually under anaesthesia, until the crusting had ceased. Differentiating between normal granulations under the crusting and tumour recurrence was difficult, which made careful examination and repeated biopsies important until the crusting had settled. Knegt recommended a permanent oroantral fistula for inspection. Admittedly, this made inspection easier, but it was very unpopular because of oronasal regurgitation and was stopped quickly. Support of the 5-FU nasal pack was difficult for each week it was in place; a Whitehead's varnish ribbon gauze pack was used in the primary surgery, to hold it in place and control early oozing. A compressed foam tampon pack was used for support of follow-up packs placed weekly for six weeks.

Headache and occasional visual disturbance were symptomatic problems that settled once the packing was removed. The introduction of the powered microdebrider made a huge difference to the precision of tumour removal.

Surprisingly, in the long term, a huge cavity caused little inconvenience to the patient once the crusting had settled. Cosmetically, there was no external sign or deformity. Functionally, the cavity was of no consequence. In time, inspection was conducted during a quick out-patient visit. Even in the case of recurrence, a repeat full course of 5-FU seemed to work in most cases.

Neither magnetic resonance imaging nor computer-assisted tomographic imaging seemed to help in the early assessment of recurrence.

Following on from the paper on a 20-year experience of woodworker's adenocarcinoma of the ethmoid sinuses,[14] little has changed in the 1996–2006 figures, with no further recurrences or retreatments. Only one patient is still alive and free of disease, but those who died were disease-free.

There have been two new cases since the original study: one patient presented in 2008, aged 86 years, and died disease-free three years post-treatment, and another younger patient, who presented aged 62 years, died of disease in his frontal lobes after only one year post-treatment. Both were treated by transantral surgery and 5-FU application. There have been no new cases in the last 12 years.

Discussion

I saw five cases in 1986 at High Wycombe,[9] and an average of at least two new cases a year presented for a while after. In time, however, there was a drop off in the numbers, with no cases after 2008. It is believed that the decline in incidence mirrored the decline of the woodwork industry in High Wycombe. There are still factories around, but these exist in much smaller numbers, and most have impressive amounts of space, with, importantly, better and effective ventilation. However, even as many manufacturers sourced their production elsewhere, often overseas, or went out of business, the skilled staff did not tend to migrate with the work and stayed in the area.

As mentioned earlier, it is known that there is a definite latency of up to 30 years before the onset of the disease. When this latency is considered, the decline in incidence appears to be the result of some other factor.

Almost all the factories in High Wycombe changed over time, progressing from manual to belt-driven tools that had much lower levels of dust than the subsequent mechanical multipliers, to electric machinery, and, finally, to pneumatic or hydraulic feed mechanisms that powered high-speed work with increasing levels of dust and heat products.

Legislation had been brought in to reduce the levels of dust in the atmosphere. In 1988, the Control of Substances Hazardous to Health Regulations specified the allowable levels of dust in the factories.[15]

Even before the Control of Substances Hazardous to Health Regulations, dust extraction had been introduced, gradually but effectively, in all the workshops. This extraction was introduced firstly in small ways with vacuum devices close to the dustier areas and machines, and evolved to fully enclosed systems for sanding and cutting. Today's factories, such as Ercol in Princes Risborough, have become a model of 'dust

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efficiency', with almost all the dust captured and recycled for energy production. Dust-filtering masks were also introduced and, after an initial reluctance, all workers used them. This pre-dated the Control of Substances Hazardous to Health Regulations, and their use became universal by the mid-1980s.

It appears, with the latency of onset taken into account, that there was a decline in the causative factor from the 1970s and early 1980s, well prior to the decreased reduction in presentation. The statistical case for this is well argued in a paper by Rourke *et al*., published in 2014.[16]

Hadfield retrospectively traced patient's notes back to the early 1950s once she became aware of the problem. Obviously, there may have been cases earlier but not picked up. There was no folklore or previous reference to the disease amongst the thousands of woodworkers of High Wycombe. It is unlikely that pre-war clinicians would have missed the unusual number of patients involved. It seems highly likely that this connection would not have been noticed if the numbers were not as high in such a small community, continually, for more than two decades before.

From this, it appears that the causative factor of woodworker's adenocarcinoma was likely to have been present from as early as the 1930s and probably in sufficient levels necessary to initiate the cancerous change, which, after a variable latency, would present as the full-blown disease. Taken with the recent decline in the number of cases, it appears there could have been a 50-year window of pre-cancerous exposure to the dust levels and, subsequently, a 50-year window of the disease presenting.[16]

The development of high-speed machinery appears to be the catalyst for the onset of this epidemic. The improved reduction of dust inhalation seems to have resolved it. The actual agent is still a mystery.

- A total of 146 cases of nasal adenocarcinoma in woodworkers over 50 years are discussed
- Cause of disease was exposure to hardwood dust and heat products
- The period of exposure was 1930s to 1980s
- Exposure was reduced by exhaust ventilation and masks
- Treatment with 5-fluorouracil seems the best option

It is entirely possible that this disease, its cause, and even this article, will be of historical interest only. It is also possible, and mildly ironic, that the treatment protocol developed may not have a wider application.

Conclusion

Rarely does a disease process occur in a population within such a small geographical area. Because of the beech woods around High Wycombe and its proximity to London, furniture manufacture was concentrated here. However, over the last 50 years, a high rate of adenocarcinoma of the ethmoid sinuses appeared in factory workers. This paper outlines how the disease was initially recognised, its diagnosis and the treatment development, and discusses how both the appearance and disappearance were seemingly caused by changes in manufacturing practice.

Competing interests. None declared

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