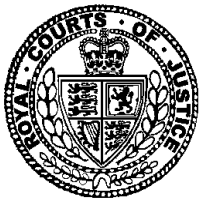

Neutral Citation Number: [2020] EWHC 416 (QB)

Case No: QB-2018-000827

IN THE HIGH COURT OF JUSTICE
QUEEN'S BENCH DIVISION

Royal Courts of Justice
Strand, London, WC2A 2LL

Date: 28/02/2020

Before:

MR JUSTICE STEWART

- -

Between:

TRACEY KING **(as Personal Representative of the Estate of KEVIN KING, Deceased)**	**Claimant**
- and –	
SOUTH TEES NHS HOSPITAL FOUNDATION TRUST	**Defendant**

- -
- -

Mr A Axon (instructed by **Armstrong Foulkes**) for the **Claimant**
Mr Q Fraser (instructed by **DAC Beachcroft**) for the **Defendant**

Hearing dates: 17th, 18th and 19th February 2020

- -

Approved Judgment

MR JUSTICE STEWART:

Introduction

1. On 2nd February 2017 Mr Kevin King ("the deceased") died due to lung cancer. The Claimant is his widow. The claim she brings is for damages consequent upon his death. The deceased was aged 56 years at the time of his death, his date of birth having been 15th October 1960.

2. On 22nd May 2016 the deceased attended the accident and emergency department of James Cook University Hospital complaining of a number of symptoms. The diagnosis made was of a deep vein thrombosis in the left leg. A number of investigations took place including a chest x-ray. The chest x-ray was reported on 26th May 2016. It identified a 3cm. focal area of increased opacification in the left mid zone which had not been obvious on the previous examination of 28th January 2014. Pathology and even malignancy were described as being "difficult to exclude". A further assessment with CT scanning was suggested.

3. No further action was taken, however, following this report.

4. Thereafter the deceased's health deteriorated. He suffered from cough, hoarseness, lethargy, loss of weight, reduced appetite and breathlessness.

5. On 25th November 2016 a further chest x-ray was performed. This showed that the opacity described on the previous image of 22nd May 2016 had increased significantly in size. The deceased was urgently referred to a consultation with a respiratory physician who saw him on 6th December 2016. A CT scan was performed. On 14th December 2016 a PET scan was performed. Because of the findings of these scans and the clinical situation, the deceased began chemotherapy in January 2017 but deteriorated quickly and died on 2nd February 2017.

The issue for determination

6. The Claimant's case, as pleaded in paragraph 17 of the Particulars of Claim, is that the May 2016 chest x-ray should have been acted upon and a diagnosis of lung cancer would have been made in June 2016, some 6 months prior to the actual diagnosis. Breach of duty in this regard is admitted in paragraph 9 of the defence.

7. The issue to be determined by the court is how far advanced the cancer was in June 2016.

The Expert Evidence

8. The expert evidence from the Consultant Clinical Oncologists is contained in the following documents:

    i) Report from Doctor Lester dated 10th February 2019;

    ii) Report from Doctor Peake dated March 2019;

    iii) Joint statement of Doctor Lester and Doctor Peake dated 7th August 2019.

iv) Letter from Doctor Peake dated 22nd December 2019, replied to by Doctor Lester on 9th February 2020.

9. Counsel for the Defendant provided a helpful simple glossary of terms. I am told it is on an agreed document. This is attached to this judgment as Appendix A.

10. In the joint statement the oncologists agree that the size of the primary tumour in June 2016 was approximately 3cms. They disagree on the N staging of the cancer in June 2016.

11. Doctor Lester's opinion was that the deceased had no regional lymph node metastasis (N0). Doctor Peake's opinion is that the deceased had at least metastasis in the ipsilateral mediastinal and/or subcarinal nodes (N2). It follows from this that Doctor Lester's view is that the deceased had T1c N0M0 (Stage IA3) or T2aN0M0 (Stage IB) localised squamous cell lung cancer in June 2016. Doctor Peake agrees with the T stage. However, his opinion is that the deceased had N2 Stage IIIA disease in June 2016.

12. If Doctor Lester's view is accepted then the oncologists agree that treatment would have been with surgery. Had he been treated with either surgery or chemotherapy, he would not have died in February 2017. He would have been expected to survive some 8 years from the date of surgery. Therefore his life expectancy would have been until June 2024.

13. If Doctor Peake's opinion is correct then the oncologists agree that surgery would not have been offered in June 2016. Treatment would have been with radiotherapy and chemotherapy. The deceased's life expectancy would have then been a survival of 29 months from date of diagnosis, such that the deceased would have been expected to live until November 2018.

14. There is also agreement on the probable effects of the lung cancer, had it been properly treated in June 2016. The parties have agreed based on the alternative hypotheses,

    i) If Doctor Lester is correct and the deceased had had surgery in June 2016, he would have taken 3-6 months fully to recover from the operation. On the balance of probabilities, he would then have had 7 years good quality life before the cancer recurred. When the cancer recurred, he would have declined in performance and function in the months leading to his death.

    ii) If Doctor Peake is correct then, following chemotherapy and radiotherapy in June 2016, the deceased would have been more short of breath as a result of radiation toxicity to the lung. He probably would have had 1.5 to 2 years of good quality life before the cancer recurred. When the cancer recurred, he would have declined in performance and function in the months leading to his death.

15. Having set out relevant matters in the joint statement of experts, it is useful to incorporate, by way of explanation, Doctor Lester's introduction which explains lung cancer staging. He said this in his report:

> "Lung cancer is staged in the same way as other cancers using the Tumour, Node, Metastasis (TNM) classification. T describes the size of the original (primary) tumour and whether it has invaded nearby tissue, N describes nearby (regional) lymph nodes that are involved, M describes distant metastases (spread of cancer from one part of the body to another). Once the T, N and M are determined for a particular cancer, they are combined and an overall stage of 1, 2, 3 or 4 is assigned. Stage 1 cancers are small, with no spread to the nodes or distant metastases and have the best prognosis. Stage 1 lung cancers are further sub-divided into stage 1A1, 1A2, 1A3, 1B, depending on primary tumour size. Stage 4 cancers have spread to other parts of the body (M1) and have the worst prognosis. Stage 2 and 3 cancers consist of various combinations of TNM between stage 1 and stage 4. Stage 1 and 2 cancers are commonly referred to as "localised disease", and stage 3 and 4 cancers as "locally advanced or metastatic disease". The stage of disease is important as it guides both treatment and prognosis; the earlier the stage at diagnosis, the greater the chance of cure and the better the prognosis. It is well established that cancers grow with time. As they grow, the potential for spread increases; cancers progress from an early stage to a more advanced stage, which decreases the chance of cure and reduced life expectancy …"

The experts' experience

16. Doctor Lester has been a consultant clinical oncologist since 2003. He describes himself as a respected leader in his fields of interest. His practice is based in South East Wales. He has clinics in two hospitals in Cardiff. He remains a practising consultant doing some six clinics a week. On average he sees about 250 new lung cancer patients per year.

17. Doctor Peake's experience was examined in more detail. He had provided, apparently at the request of the Claimant's solicitors, a curriculum vitae. Doctor Peake had been appointed a consultant in Birmingham in 1997 dealing with two cancer clinics outside the main central hospital. In one of them he was the only oncologist. He had checked his records and from then until 2011 he had seen some 120-140 new lung cancer patients a year. In 2011 he started a new clinic and gave up some of the lung cancer patients to a colleague. In 2015 he stopped seeing new lung cancer patients.

18. From 2011 lung cancer ceased to be one of Doctor Peake's main interests. Since 2016 Doctor Peake has mainly taken on a more management role. His last publication was in 2016. Up to 2016 his main academic interest was more related to sarcomas. He had, however, been a principal investigator collecting data for a controlled trial of a drug in patients with small cell lung cancer. The data was collected whilst he was at Sandwell Hospital prior to 2015. Doctor Peake's hospital was one of 92 which contributed data to the trial.

The stage of cancer at June 2016: Doctor Lester's Report

19. In December 2016 the deceased's lung cancer was T3N3M0 stage 3C.

20. As to the T stage, Doctor Lester said that the primary cancer was measured at approximately 3cms. on the x-ray in May 2016. Absent any additional radiological imaging at this time, this is the most accurate estimate available of primary tumour size. A T1c tumour measures greater than 2cms., but 3cms. or less in greatest dimension. T2a is a tumour measuring greater than 3cms., but 4cms. or less in greatest dimension. On that basis Doctor Lester's opinion is that the T stage was T1c or T2a in June 2016.

21. As to the N stage, N0 is no regional lymph node metastases, N1 indicates metastasis in ipsilateral hilar lymph node or nodes and N2 metastasis in ipsilateral mediastinal lymph nodes. Doctor Lester says that the 26th May 2016 x-ray did not show any clear evidence of nodal disease. He says that the likelihood of N0 disease (i.e. no nodal spread) with a primary squamous cancer of this size is 72.6% based on published series of surgically resected cancers. Therefore, on the balance of probabilities the deceased had N0 disease in May 2016.

22. Doctor Lester obtains the 72.6% figure from a paper namely Oda M et al.: The extent of mediastinal node metastases in clinical stage 1 non-small-cell lung cancer: The role of systematic nodal dissection. Lung cancer 1998; 22: 23-30 (Oda et al).

23. Finally, Doctor Lester records that which is common ground, namely that the M stage was M0, namely no distant metastases.

24. In summary therefore, his opinion according to TNM version 8 (the 8th edition of the UICC Classification of Malignant Tumours) was that Mr King had T1cN0M0 stage 1A(3) or T2aN0M0 (stage 1B) localised squamous cell carcinoma of the lung in June 2016. He also relied upon the following:

    i) The average survival of patients with untreated stage 3 or 4 disease (locally advanced or metastatic) at diagnosis is approximately 5.03 months. Therefore, on the balance of probabilities the deceased had localised disease in June 2016 as he survived 8 months until February 2017. The average survival of patients with untreated early stage (stage 1 and 2) lung cancer is approximately 12 months. Doctor Lester suggests that the reason the deceased lived less than the average estimated 12 months survival is a combination of two factors, namely:

        a) He was in relatively poor health before chemotherapy started. On 13th January 2017 he was described as being performance status 2. In medical terms this is not very fit. This poor fitness was a burden of the cancer, not pre-existing health problems. It is well established in lung cancer that poor fitness equates with poorer outcomes and a shorter life.

        b) The chemotherapy may have shortened the deceased's life. He had grossly deranged liver function tests and developed a chest infection within a few days of the chemotherapy. Both of these are recognised side effects of chemotherapy treatment and will have contributed to the deceased's shorter than expected survival.

(In support of the survival statistics, Doctor Lester quotes Wao, H et al. Survival of patients with non-small-cell lung cancer without treatment: a systematic review and meta-analysis. Systematic reviews 2013, 2:10 (Wao et al)).

ii) A paper published in 2016 which he describes as the "largest and most robust published research on the outcomes of treated lung cancer patients". This paper is the IASLC Lung Cancer Staging project: proposals for revision of the TNM stage groupings in the forthcoming (eighth edition) of the TNM classification for lung cancer. Goldstraw P. et al. J Thorac Oncology 2016 Jan; 11 (1): 39 – 51 (Goldstraw et al). Figures derived from table 6 of Goldstraw et al suggest that patients presenting with a tumour measuring 2cms. – 3cms. had an 80.4% chance of N0 and those with a tumour measuring 3cms. – 4cms. a 68% chance of N0.

Doctor Peake's Report on Staging of the tumour

25. Doctor Peake says there is significant uncertainty regarding the likely staging of the deceased's disease as at June 2016, but, on the balance of probabilities, it would not have been N0, stage 1 disease. At diagnosis in December 2016 the deceased had locally advanced disease invading the main bronchus and extensive mediastinum disease, T3M3. The PET scan reported a primary tumour to be 4.5 x 3cms. and the subcarinal nodal mass measured 5 x 3.5cms. The chest x-ray of May 2016 reported a 3cms. lesion. Although it is not possible to make a direct comparison between these measurements, Doctor Peake says this suggests there had been a limited increase in size over 6 months. Whilst accepting the possibility that the nodal disease grew more rapidly than the primary tumour, on a balance of probabilities lymphadenopathy would have been detectable on CT and/or PET scan following the chest x-ray in May 2016, and therefore the deceased would have had at least N2, stage IIIA, disease at that time. Doctor Peake says that given the extent of nodal metastases at diagnosis in December 2016, it would have been very unlikely that this could have developed from node negative disease over a 6-month period, even if this had been a rapid progressive tumour which he believes it not to have been. I attach to this judgment as Appendix B relevant extracts from the radiological information available.

The medical literature in more detail.

Oda et al

26. The objective of the Oda paper was to determine the extent of lymph node metastases in clinical stage 1 non-small-cell lung cancer (NSCLC). The method was by performing a retrospective review of 524 patients with clinical stage 1 NSCLC who underwent lobectomy with systematic nodal dissection. The authors point out that nodal status is the most important factor influencing the prognosis in NSCLC. Clinical stage 1 lung cancer is not always pathological stage 1 cancer, therefore systematic nodal dissection is necessary even in clinical stage 1 NSCLC so as to clarify the correct nodal status. [I shall return later in this judgment to the difference between clinical and pathological stages]. In Kanazawa University Hospital, Japan between January 1980 and December 1996, 582 patients with clinical stage 1 NSCLC underwent resectional surgery. Among the 524 patients who underwent lobectomy or pneumonectomy with systematic nodal dissections, of these patients 154 had

squamous cell carcinoma. Table 4 classifies the nodal status according to tumour size in such patients. For those with a tumour size of 3.1cms. and over, 52 were N0, 8 were N1 and 8 were N2. The table records that of those patients 72.6% were N0, 13.7% N1 and 8% N2. In fact, it appears that there are some arithmetical errors in Table 4. The correct figures are 76.5% were N0, 11.8% were N1 and 11.8% were N2.

27. Doctor Peake says that the Oda paper is not relevant as it considers the incidence of pathological node involvement in a cohort of patients fully staged with a CT scan and that the data cannot be extrapolated to assess the likelihood of nodal disease on CT scan following chest x-ray.

28. Doctor Lester accepts that Oda required CT scanning as an entry criterion for being in the cohort. Therefore, strictly the deceased was not in the same position as those in the cohort; we do not know that the figures necessarily apply to him for that reason. There is no published literature on patients without a CT scan, as not to do such scanning would be substandard care.

29. However, although all the cohort in Oda table 4 were clinically N0 (ie: as shown on scanning), some 85% of them were pathologically N0 also. This figure approximates to the agreed oncologists' evidence that about 90% of patients who are clinically N0 turn out to be pathologically N0. The figure for tumours of more than 3cms in Table 4 is 76.5%. Therefore analysis of Oda et al does support, statistically, that a patient such as the deceased who on x ray had a 3cm tumour would probably have been N0 clinically in June 2016.

Wao et al.

30. Wao et al conducted a systematic review and meta-analysis of the natural history of patients with confirmed diagnosis of lung cancer without active treatment. Specifically, they estimated overall survival when no anti-cancer therapy had been provided. The methods used were to search electronic databases and abstract proceedings, to review the bibliographies of included articles and contact experts in the fields. As to results they included in the meta-analysis seven cohorts studied (4418 patients) and 15 randomised control trials (RCTs) (1031 patients). All studies assessed mortality without treatment in patients with NSCLC. As appears from table 2, the cohort studies dealt with disease stage 1 and 2. The RCTs dealt with disease stage 3 and 4. The figures extracted are on page 7 of the paper. Of the 4125 patients in the cohort studies, the pooled mean survival was 11.94 months. In the RCTs there were 1031 patients. The pooled mean survival for those patients was 5.03 months.

31. Doctor Lester accepted that on the face of it, the deceased survived more in line with stage 3 and 4 patients than stage 1 and 2 patients. He also accepted that the patients in the Wao paper, untreated for their cancer, were just as likely as the deceased to have had ill health as a consequence of their cancer before they died. He said, however, that this has to be looked at in conjunction with the fact that the deceased, unlike the Wao patients, had received chemotherapy. In reality, revising the two points made in his report, there was only really one point namely the effect of the chemotherapy treatment on the Claimant who was already in poor health due to his cancer.

32. Doctor Lester said that if a person's pre-existing general health is not optimal at the time of the commencement of the chemotherapy, the physiological insult is such that

the body may not be able to handle it. The deceased would have been given chemotherapy with a view to attempting to extend his life and to help with his symptoms. However, chemotherapy comes with a risk to the individual.

33. There was consideration of the performance status of the deceased as appears from his medical records. Performance status runs from 0 (no real impairment of function) to 5 (death). If somebody has a performance status of 0 or 1 then chemotherapy is indicated. At performance status 2, there is some contention. In the UK, perhaps 25% of patients have performance status 2. It is therefore not unreasonable to treat such patients with chemotherapy. Performance status is subjective and something of a blunt tool. If for example the performance status is 2 and this is as a result of the cancerous disease, there may be more hope for a successful outcome than if the reduced performance status is due to a pre-existing condition e.g. diabetes.

34. Looking at the deceased's performance status as contained in his medical records:

    i) There is an entry of 25th November 2016 which says that the deceased had a history of near to 1.5 stones weight loss over the preceding two months, together with night sweats.

    ii) On 6th December 2016, when seen by the consultant respiratory physician, the deceased's performance status was said to be 0. Stopping at that point, Doctor Lester said looking at these two notes together, the deceased was clearly having symptoms. He hesitated to say it but the deceased might have been having good days and bad days.

    iii) On 13th January 2017, when seen by the consultant oncologist, the deceased's performance status was 2.

    iv) On 18th January 2017 the deceased received this first and only cycle of chemotherapy.

    v) Between 20th January and 25th January 2017, the deceased was admitted with shortness of breath and other symptoms following the first cycle of chemotherapy. He was found to have deranged liver function tests. A clinical note of 25th January 2017 recorded that Dr. Zubair (oncologist) "thought this could also be chemotherapy related." Doctor Lester said that shortness of breath can be caused by the burden of the cancer and/or the chemotherapy. The second cycle of chemotherapy planned for 8 days after the first was cancelled. He was discharged on 25th January 2017, then re-admitted on 28 January 2017.

35. Doctor Lester said that he could not be sure as to the cause of death, but it would be difficult to say that the chemotherapy was not a contributory cause of death. In his report he said that the chemotherapy may have shortened the deceased's life. In his 9th February 2020 letter he said that the chemotherapy – related toxicity "significantly shortened his life". Doctor Lester agreed that his opinion was in fact more in line with his report than his letter. His use of language in the letter was slightly loose.

36. Nevertheless, Doctor Lester said that an opinion on any situation such as this one is based on clinical impression of treating patients over the years. When treating

patients with chemotherapy, he always considered early death as a side effect. Doctor Lester said that 80% of those who die as a result of the chemotherapy treatment die after first cycle. In the deceased's case it would be about the right time that he died after the first cycle, because most people die within the first week or two of the administration of the first cycle. The deceased here survived 15 days. That does not make the contribution of chemotherapy any the less likely.

37. Doctor Lester said that if the liver function tests are deranged then, if one organ fails, this has a knock-on effect. The deranged liver function tests are often a consequence of the chemotherapy and suggest that the chemotherapy on balance contributed to the earlier death.

38. Doctor Lester said it is speculative whether the deceased would, absent the chemotherapy, have lived one month or three months or more. He assumed that whoever decided to give the deceased the chemotherapy decided that he would benefit from the treatment, and therefore considered that without the treatment the deceased's life expectancy would have been a few months and that the chemotherapy might extend this.

39. Doctor Lester did accept that given that the life expectancy in the Wao paper for stage 3 and 4 was 5.03 months, then if as Doctor Peake suggests, the deceased had stage 3A in May/June 2016, then perhaps one would expect an untreated life expectancy of somewhat better than 5.03 months. Doctor Peake, for his part, said that although few people diagnosed as Stage 3 or 4 last longer than 6 months, if untreated, the deceased would have been on the better side of Stage 3.

40. Doctor Peake was not persuaded that the deceased was performance status 0 in December 2016. He based this substantially on the Claimant's witness statement in which she indicates that her husband was not able to do day to day tasks by this stage. Although the deceased continued to work through the latter half of 2016, she said, in paragraph 8, that he was so tired when he returned from work that she had to do everything around the house, including the tasks he had always done. Nevertheless, whether the deceased was performance status 0 or performance status 1 he became performance status 2 and therefore entered the terminal phase. Patients struggle to cope at first and then start to become ill and there is a snowball effect. Further, Doctor Peake referred to entries in the records that the deceased may have been suffering from unrelated cholecystitis as at 20th January 2017 and this may have been a different reason for the deranged liver function tests. Given Doctor Peake's belief that it would be very unusual for cholecystitis to be caused by chemotherapy administered only two days before, he thought this was a potential different reason for the deranged liver function tests and one which was unrelated to the chemotherapy.

41. Having set out the evidence between the consultants in some detail on this point, nevertheless there was a substantial measure of agreement that the chemotherapy was probably a factor in the Claimant's death but it would be very difficult to estimate by how much the administration of the first cycle of chemotherapy had accelerated the death.

Goldstraw et al

42. The International Association for the Study of Lung Cancer (IASLC) International Staging Project collected a new database of 94,708 cases donated from 35 sources in 16 countries around the globe. This data was analysed. The relevant table for the purposes of this case is table 6. There is a breakdown in terms of size of tumour and the N0, N1, N2 and N3 findings. For a T1 tumour between 2 and 3cms. the total sample size was 5731 of which 4606 (80.4%) were N0, 492 (8.6%) were N1, 596 (10.4%) were N2 and 37 (0.6%) were N3. For T2 tumours of 3-4 cms. the corresponding figures on a total sample of 9387 were 6382 (68%), N0, 1250 (13.3%), N1, 1666 (17.7%) and N2, 89 (0.09%), N3.

43. Doctor Lester accepted that as at December 2016 the primary tumour was 4.5 cms. and the subcarinal lymph node mass was 5 cms. He was taken to Table 6 of Goldstraw which indicates that for somebody with a primary tumour of 4-5 cms., only 35 out of 2780 people will have N3 nodal involvement. It was therefore suggested to him that the incidence of N3 nodal involvement with that size primary tumour is only 1.3% (35/2780). Doctor Lester did not accept that. He pointed out that Table 6 was based on people whose tumours had been surgically resected. As agreed in the joint statement, a surgeon would not expect to resect if there was N3 nodal disease present. This is because it would be dangerous to do so. Thus, a large number of 4-5 cms. primary tumours with N3 nodal disease would be excluded from Table 6. This explains why, counter-intuitively, even though the primary tumour increased substantially in size on the Table 6 figures, the N0 nodal involvement remained over 50% in each size category. As explained by Doctor Lester this makes perfect sense. Therefore Table 6 is not a proper basis for the suggestion, put to Doctor Lester, that the incidence of N3 nodal disease with a primary tumour of 4-5 cms. is only 1.3%.

44. It further followed from Doctor Lester's evidence on Table 6 that the smaller tumours there described give rise to more robust figures, because the smaller the tumour the less likely it is that any nodal disease will be present. Therefore the 68% N0 finding for a tumour of 3-4 cms. (the deceased's primary tumour as at May 2016) is unaffected by the selection bias of whether members of the cohort were fit for surgery.

45. Doctor Lester was then asked about Table 2 on the basis that a proposed category T/M tumour, categorised as T3, showed only 77 N3 nodal disease cases out of a total of 1449. This approximates to 5%. This Table categorises the patients by clinical classification, rather than by histology after surgical resection, as in Table 6. Doctor Lester pointed out that under the "Methods" section of the Goldstraw paper, it states "for cases which chemotherapy was received before surgery (yp cases), only clinical stage was considered." He said that it appeared from that these were patients who were receiving chemotherapy with a view to surgery. He said that if there is some equivocation as regards the nodal status then chemotherapy prior to surgery can be indicated. If the tumour shrinks then surgery may well take place. If it grows, then surgery may well not be indicated. Thus Table 2 is an atypical subset in that it only selects people who have been considered as possible candidates for surgery. If surgery is not indicated, as is generally the case with N3 nodal disease, patients in that category would not be incorporated into Table 2. He suggested that the reason why this clinical classification table was included in the paper is that pre-surgery chemotherapy may "muddy the waters" of tumour classification by proposed T/M categories and nodal disease involvement after subsequent surgery and histological

analysis. Chemotherapy may have, pre-operatively, significantly reduced the size of the tumour.

46. Doctor Lester added that there may be some difficulty in comparing the proposed T/M categories in Table 2 with the categories in Table 6. The T1 and T2 categories are probably analogous but for T3 and T4 the categorisation is not just driven by size. The description in Table 6 is one based on size only, whereas Table 2 categorises by the proposed T/M.

Doctor Peake's letter 22 December 2019 – The Wang Paper

47. Further to the reasoning given in his report and the joint statement dated August 2019, Doctor Peake disclosed a letter dated 22nd December 2019. This included a paper from Wang et al. published in Lung Cancer: 2012 October; 78 (1): 51-56. Doctor Lester responded by letter dated 9th February 2020. The central part of Doctor Peake's further evidence is that he says that most commonly described model of tumour growth is the exponential model in which the tumour volume (or mass) increases by a constant fraction in equal intervals of time. Although this is an over-simplification, it is generally accepted as a good approximation for much of the tumour's natural history. The model describes a tumour starting as a single cell and then dividing into 2, then 4, 6, 8 etc. The time of doubling up cells is constant and is called the volume doubling time. The volume doubling time can be calculated from serial measurements of tumour diameter. If the diameter of a tumour doubles, volume increases eight-fold. On the chest x-ray in May 2016 the primary tumour measured approximately 3cms. in diameter. On the PET scan, at diagnosis it measured 4.5cms maximum in diameter. Using these measurements the doubling time of the tumour was 117 days, which is consistent with the published data on rates of growth of lung cancer. Doctor Peake says therefore the tumour was growing at a typical rate for lung cancer and in keeping with clinical experience.

48. Doctor Peake says that the Wang paper reported that the rate of growth of lymph nodes is similar to that of the primary tumour. If the lymph node disease grew at a similar rate to the primary disease, then in May 2016 the subcarinal mass would have measured approximately 3.3cms. diameter. Even if the nodal disease was growing at the twice the rate of the primary tumour, it would have measured greater than 2cms in May 2017. Doctor Peake says that there are a couple of caveats, namely (i) it is likely that metastatic disease starts as a small clump of cells, rather than a single cell and (ii) the nodal disease may not be a single lymph node but could have started as a number of different lymph nodes that have coalesced to form a single mass. Despite this, Doctor Peake says it is almost inconceivable that the deceased would have progressed from N0 to N3 with a 5cm nodal mass within 6 months, particularly given the rate of growth of the primary. Further, in Doctor Peake's experience of treating hundreds (probably more than a thousand) patients with non-small-cell lung cancer over the past 25 years, he does not recall ever seeing a patient whose nodal disease progressed so rapidly, and definitely not in whom the tumour grew at a typical rate.

49. As to this last point Doctor Peake accepted that after first scanning, patients would generally either be treated (if treatable) or not scanned again if not susceptible to treatment. However, he said there are instances of patients having two scans. He gave examples such as if a person has incurable cancer and is asymptomatic or relatively asymptomatic then the patient is often given the choice of opting for

palliative chemotherapy at that point or, when the symptoms worsen, considering chemotherapy at a later point. At that later point further scanning may well take place. Nevertheless, it would clearly be a relatively small minority of patients who had scanning some months apart. However, in his experience he said that one would normally see primary and secondary tumours progressing at approximately the same rate.

50. Doctor Lester criticises the use of the Wang paper in his letter of 9 February 2020. He says:

i) The paper is based on an analysis of 12 patients who had nodal disease in the mediastinum diagnosis and had a second scan, a median of 43 days after the initial diagnostic scan. He says it is not appropriate to state that "*the growth of lymph nodes is similar to that of the primary tumour"* based on a study of only patients who were followed up for 6 weeks. It is too small a group of patients followed up for too short a period of time for any robust conclusions to be made. In the discussion section of the paper, the authors admit to the limitation that "*the estimation of the doubling time was based on only 2 pre-treatment PET/CT images rather than multiple serial scans, therefore the calculations would be more susceptible to variation due to technical factors in the volumetric and metabolic measurements."* Doctor Lester says that the fact that there were not multiple serial scans means that the calculations were susceptible to variation error. In oral evidence he added that the study was not designed to look at volume doubling time of nodal disease. To extrapolate what the Defendant sought to extrapolate from Wang, he described as "bad science". It is not possible to scan to watch tumours grow. To do so would be negligent. Alternatively, if treatment is not indicated, further scanning should not be done just for research purposes.

ii) There is no reference in the paper to the view that growth of lymph nodes is similar to that of the primary tumour. Doctor Lester says he has never come across the concept in 20 years of treating lung cancer patients. He is not aware of any other academic paper that support this view. A linear relationship between the growth of the primary cancer and the growth of nodes simply does not reflect what is seen in clinical practice. Nodes and primary cancers will often grow and respond to treatment at entirely different rates. Nodes can be significantly larger than the primary cancer at diagnosis, indicating a much shorter volume doubling time for the nodes compared to the primary cancer. That is why, in this case, it is perfectly plausible to find a 5cm nodal mass and there be no nodal disease 6 months earlier. The rapidity of growth in the deceased's case is consistent with lung cancer and is why early diagnosis and treatment is vital. Doctor Lester explained in oral evidence that when he had used the terminology "perfectly plausible", he meant that he would not raise an eyebrow at such a finding. He would not regard it as out of the ordinary.

51. Doctor Lester was extensively cross-examined on this point. He said that looking at the size of the primary tumour may indicate how long it has been present but, as to nodal mass, this sort of estimation was very disputable. He accepted that obviously a 5 cm nodal mass would not grow in a day but it is impossible to say whether it would grow in one month or anything up to five years. Although, broadly speaking, small cell carcinomas grow faster and patients have a lower life expectancy, he would not

be surprised, in non-small-cell carcinomas with nodal mass growth from 0-5 cms. in a week or two. Nor would he be surprised if there was a growth from 0 nodal mass to 5 cms. over 6 months with steady growth in between. He said that 6 months is a relatively long time. He has seen such growth from 0 cms. to 5 cms. in 6 months. He said that lung cancer is very unpredictable. It is a very aggressive disease and can grow very quickly. Volume doubling time in lung cancer is not a clinical matter. It is an academic matter. In practice an oncologist does not work out the volume doubling time of secondary tumours.

52. Doctor Lester accepted that the primary tumour doubling time in the deceased's case was not unusual. He said it was not possible to know if the doubling time concept applies to secondary tumours. He was not aware that there was any predictable growth time for secondaries such as implied by this concept. Further, the assumption in primary lung cancer is that a single cell doubles and then the resulting two cells double again and so on. As regards lymph nodes, disease may not necessary be visible. Also, the node is lymph tissue designed to react to foreign invaders, one of which may be cancer. Therefore, it will react in a completely different way to the primary tumour. For these reasons there is no literature on the point.

    Doctor Lester said that, in any event the volume doubling time for secondary tumour activity would not have to be a few days for a secondary to go from 0 to 5 cms. in 6 months.

53. Doctor Peake accepted that it was not the purpose of the Wang paper to draw conclusions about the growth between primary and secondary tumours. This is an observation of a finding. He accepted as caveats the limitations in the paper to which Doctor Lester had drawn attention. Nevertheless, he said it was the only literature which made any statement upon correlation between primary and secondary tumour growth. The relevant sentence in the paper is "12 patients had non-contiguous primary and metastatic lymph node sites. There was no significant difference in volumetric growth …" The paper also discusses the fact that tumour volumetric measurements increased remarkably in a short interval (the median interscan interval was 43 days).

54. Doctor Peake was taken to a text book chapter headed "The growth rate of tumours", the author of which was G.Gordon Steel. At page 11 it says "within any one tumour type there is a wide range of volume doubling time. For instance, the range of values for lung metastasis of adenocarcinoma is shown in figure 2.5. Some double their volume in a week, some in a year or more, and the median is around 90 days." Doctor Peake said it was unusual but not impossible for metastasis to double in a week.

Bronchoscopy

55. In the deceased's medical records there is a bronchoscopy report dated 7th December 2016. This report shows that there was a tumour overgrowth in the medial wall of the right main bronchus. Doctor Lester said he assumed this was referring to the subcarinal mass, not the primary tumour. Cancerous cells grow along the line of least resistance. Therefore, in the deceased's case, they would have had to erode the wall of the bronchus. Doctor Lester said it was pure speculation as to how long the mass had been there. It was not possible to say that the fact that it had eroded the wall was

an indication of time. It may well be an indication of the aggressiveness of the tumour.

56. Neither Doctor Lester nor Doctor Peake had noted the bronchoscopy reports in their expert reports. It was first raised by Doctor Peake very shortly before trial and was the subject of oral evidence only. In the first report Doctor Lester had not noted the size of the subcarinal mass. He accepted that consideration of the growth rate of nodal disease and the bronchial mass were aspects worthy of consideration. Nevertheless, it was not possible to base any findings upon them in terms of the length of time they had been present and/or the speed of growth.

57. Further when asked about the bronchoscopy, Doctor Lester made these points:

    i) He relied not only on the statistical evidence in the papers but also on his clinical experience. He said that the majority of patients he saw with cancers of the size which the deceased had in May 2016 did not have nodal disease. His clinical findings were therefore in keeping with the statistical bases in the literature.

    ii) He said it was important that the N stage makes no reference to the size of the nodal disease or if it is for example eroding the bronchus. Thus, in the accepted classification, neither of these factors is listed as being relevant. This is because it is simply not known whether they do or do not have any relevance.

58. There was evidence as to the potential way secondary tumour had spread to the right main bronchus. I do not need to go into detail upon this. There was agreement that the strong probability that the large mass of lymph nodes had invaded the wall of the bronchus from the outside to the inside. The squamous cell carcinoma had gone through the wall of the bronchus which is a fibrous wall which would provide resistance to penetration. Doctor Peake relied upon this for his opinion that this was an unusual finding and indicated that secondary tumour had been there for a considerable period of time. He said that this reflected the length of time the metastatic disease had been present. Doctor Lester disagreed, saying that it is in the nature of the squamous cell carcinomas that they are invasive. As distinct from other tumours, invading tissues in this manner is what they do. He disputed Doctor Peake's evidence that the bronchoscopy confirmed his previous statement that the metastatic disease had been present in the lymph nodes in May/June 2016 and would have been detectable on CT or PET scanning.

Legal Authorities on the use of statistical evidence

59. The parties drew my attention to certain citation from authority in which the Courts have considered the approach to evidence based on statistics. I have considered: *Gregg v Scott* [2005] 2AC 176; [2005] UKHL 2, Lord Phillips at [153]; *Sienkiewicz v Greif* [2011] 2AC 229; [2011] UKSC 10, Lord Rodger at [163] and *JD v Mather* [2012] EWHC 3063 (QB) at [35].

60. Perhaps the most relevant dictum for this case is *Wardlaw v Farrar* [2004] PIQR P19; [2004] EWCA Civ 1719. Brooke LJ said:

> "35.. I do not understand this. When the judge had to consider, on the balance of probabilities, whether Dr Farrar's negligence (and the consequent delay in her admission to hospital) was causative of Mrs Wardlaw's death the judge had to take into account all the relevant evidence, and the rival cases that were being put forward at the trial in relation to this evidence. The failure of anti-coagulant therapy, when it was tried, to prevent the formation of a massive pulmonary embolism (which had not been present on 22nd September) was inevitably a material piece of evidence. While judges are of course entitled to place such weight on statistical evidence as is appropriate, they must not blind themselves to the effect of other evidence which might put a particular patient in a particular category, regardless of the general probabilities."

Discussion

The question for the court

The first point to make clear is that the question I have to determine is whether on the balance of probabilities the deceased would have been N0 as of May/June 2016. As appears from this judgment there are two potential bases for describing nodal involvement. One is clinical, based on scanning. The other is pathological, based on post-operative histological examination. A substantial amount of evidence was given on this point, mainly because Doctor Peake, when considering the Wang paper and the tumour doubling time for the lymph node disease had made calculations based upon pathological presence of nodal metastasis. On histological examination, secondary tumours which are microscopic can be detected. For CT scanning, or PET scanning which is more sensitive, the secondary tumour has to be of a certain measurable size – for example a CT scan may not show the disease if it is less than 1 cm in diameter. Nevertheless there is approximately a 90% correlation of clinical staging on PET scanning and pathological staging of tumours. The experts' joint statement is such that if the deceased was N2 clinically, then he would not have had surgery and his life expectation would have been some 29 months. If on clinical data, i.e. scanning he had been N0 then there would be some people who would not therefore be N0 pathologically. Nevertheless the life expectation of the cohort who are N0 clinically is such that they would probably survive a median of 8 years from diagnosis.

Oda and Goldstraw – statistical evidence

61. In determining whether the deceased would have probably been N0 clinically as at May/June 2016, Doctor Peake accepted, albeit with some qualification, that the Oda and Goldstraw papers demonstrated as a matter of statistics that people with a primary the size of the deceased's primary tumour would probably be N0 clinically. He further accepted that it followed that once the May 2016 x-ray had been done, had he been asked what the probabilities were that the deceased was N0, he would have said

that, based on those papers, he probably was. The papers suggest a 67%-75% statistical likelihood of the deceased having been N0 in May/June 2016.

62. Had the Defendant acted non-negligently then there would have been scanning at that stage. Such scanning was not carried out until December 2016.

63. In line with the authorities I am entitled to rely upon the statistical evidence. What I must not do is fail to take into account and weigh in the balance the evidence we have which is relevant to the deceased as an individual. There is also merit in the point made to me by Mr Axon that medical literature is generally published so as to inform medical practice, not to determine legal concepts of causation. Therefore it must be approached with some caution. Doctor Peake says that the subsequent evidence is such that the deceased would have been in the minority group in the Oda and Goldstraw papers, namely he would have been a person who had at least N2 nodal involvement. Doctor Lester disagrees and says that the points of distinction relied upon by Doctor Peake are not convincing and should not persuade the court that the deceased's disease was other than in line with the statistical majority in those two papers.

64. Against that backdrop and the detail of the evidence which I have set out above, I now evaluate the evidence, reminding myself that the Claimant must prove the case on the balance of probabilities.

Mr King's actual survival period – the Wao paper

As mentioned previously in this judgment, Doctor Lester considered that the actual period the deceased had survived was more consistent with him having been a stage 1 patient. The defendant submits that the survival rates in the Wao paper are more consistent with the deceased having been a stage 3 patient as at June 2016. I do not propose to revisit the evidence which I have summarised earlier in this judgment. In short:

i. There is a reasonable basis for arguing that the deceased's survival period of eight months was slightly closer to the mean survival rate of stage 3/4 patients; perhaps even more so given that one would expect stage 3 patients to have a somewhat longer survival rate than the total cohort of stage 3 and stage 4 patients.

ii. There was a probable contributory effect of the first cycle of chemotherapy, though this is far from certain. In any event neither of the oncologists was able to give any real estimation as to by how much (if any) the chemotherapy had accelerated death.

65. The problem is that there are so many variables and possibilities that it is not possible to draw any safe conclusion from the period which the deceased actually survived. Therefore, in conjunction with the evidence in the Wao paper, his actual survival does not assist me at all as to the deceased's disease stage in June 2016.

66. The defendant submitted that there were two aspects of this evidence which had a generally undermining effect on the quality of Doctor Lester's evidence. These were:

i. Doctor Lester's statement, that the deceased was in a better position than the cohort in the Wao paper because he was in relatively poor health before chemotherapy, was in error. I accept that on this point Doctor Lester did make a mistake. The deceased, prior to his death was in no different position in terms of estimating life expectancy than the untreated patients in the Wao cohort.

ii. In his report Doctor Lester said that the chemotherapy may have shortened the deceased's life, whereas in his 9 February 2020 letter he said that the deceased died of chemotherapy-related toxicity which significantly shortened his life. This was what Doctor Lester acknowledged in cross examination was loose language.

67. Having considered these two points, although they have merit in themselves, they do not in my judgment have any real effect in undermining the quality of Doctor Lester's evidence as a whole.

The growth of secondary disease – the Wang paper

As previously stated Doctor Peake's primary reason for contending that the deceased would not have been N0 as at June 2016, and therefore would have been in the minority of patients with a 3cm primary tumour, was the size of the nodal disease on PET scanning in December 2016. He based his opinion, first on clinical experience. He then searched the literature and discovered the Wang paper. I shall deal with that paper before I consider the dispute between the oncologists based on their clinical experience.

68. Doctor Peake accepted that there were substantial caveats about the Wang paper. Nevertheless he relied upon it as evidence that the growth of lymph nodes is similar to that of the primary tumour. Therefore, if the lymph node disease grew at a similar rate to the primary disease, then in May 2016 the subcarinal mass would have measured approximately 3.3cms in diameter. Thus the deceased would not have been N0 on scanning. In my judgment the Wang paper cannot be relied upon as evidence of any weight to show that lymph nodes grow at a similar rate to primary tumours. This is because:

(1) The paper is intended to demonstrate that lung cancers develop quickly in a short period.

(2) There were only 34 patients in the report as a whole. Only 12 of these had squamous cell carcinoma. This cannot be a statistically significant number.

(3) 12 patients were (incidentally) noted as having non-contiguous primary and metastatic lymph node sites with no significant difference in volumetric growth. It is not known whether any of these were patients with squamous cell carcinoma.

(4) The median interscan interval in the Wang cohort was 43 days. The period of growth being considered in the deceased's case is some 6 months.

69. In addition, Doctor Peake, in his 22 December 2019 letter, said that the most commonly described model of tumour growth was the exponential model in which the tumour volume increases by a constant fraction in equal intervals of time. He accepted this was over-simplification of tumour growth but said that it was generally accepted

as a good approximation for much of the tumour's natural history. This hypothesis was challenged by Doctor Lester. Broadly speaking primary tumours usually grow in a straight line (or nearly so), though even some primary tumours show irregular growth with sudden increase or decrease in growth rate not being uncommon. This evidence is found in the Steel document. Nevertheless I remind myself that, albeit in lung metastases of adenocarcinoma, Steel says that some such metastases double their volume in a week, some in a year or more, and the median is around 90 days. This supports Doctor Lester's opinion that it is inappropriate to draw any conclusions that secondary tumours grow by doubling in anything like the same way or at the same rate as primary tumours.

70. Even on Doctor Peake's hypothesis that secondary tumours do double in a similar way to primary tumours, and therefore one can work back from the secondary tumour found on scanning in December 2016, counsel agreed by the close of the case that the doubling time of such a secondary tumour would be 26-30 days from the date on which a lymph nodal mass would become clinically suspicious as malignant. This contrasts with what Doctor Peake said in his letter. He said it is almost inconceivable that the deceased would have progressed from N0 to N3 disease with a 5cm nodal mass within 6 months, because this would require a tumour doubling time of a few days for the lymph node disease. The basis for this was assuming that Mr King had no pathological nodal disease in June 2016. What should have been considered was whether he had any clinically apparent nodal disease. If one takes the latter ie: nodal disease measuring approximately 1cm or more, then one arrives at the 26-30 day calculation based on doubling.

71. I therefore reject Doctor Peake's estimations of the growth of nodal disease because: (i) they were based on nodal disease growth being essentially by way of doubling – which I find to be unproven; (ii) even if nodal disease doubling could be calculated, the doubling time would have been 26-30 days and not a few days; (iii) Doctor Peake himself caveated his calculation on the further basis that metastatic disease probably starts as a small clump of cells rather than a single cell; also the nodal disease may not be a single lymph node but could have started as a number of different lymph nodes that have coalesced to form a single mass. These, in my judgment are more than caveats. Separately, and particularly with the other two points, they result in the court having to reject any estimation of growth based on doubling times.

Clinical experience and evaluation of evidence

72. I have arrived so far at the position that the statistical evidence has not been affected one way or the other by such evidence as there is in the Wao or Wang papers or by Doctor Peake's evidence based on theoretical doubling of nodal disease. What remains to be evaluated, therefore, is the evidence based on the oncologists' experience. Doctor Lester was not at all surprised if there was growth from 0 nodal mass to 5cms over 6 months with steady growth in between. Doctor Peake regarded it as very unlikely that this development could have taken place over a 6 month period. I do not propose to repeat their evidence as set out previously in this judgment.

73. Mr Fraser, for the defendant, criticised, albeit in very measured terms, Doctor Lester's evidence. I have dealt with and rejected as in any way undermining Doctor Lester's evidence as a whole, Doctor Lester's report in respect of survival periods. The main point which is made against Doctor Lester is that he formed an initial opinion based

erroneously on the Oda paper which he has subsequently endeavoured to maintain. In other words that he has been affected, subconsciously, by confirmation bias. Further that Doctor Lester did not consider the size of the lymph nodal disease mass in his report or comment: the joint statement on whether any inference could or could not be drawn from the size of the lymph nodal mass. He only replied on this point in his letter of 9th February 2020. I do not regard any of these points as detracting from the quality of Doctor Lester's evidence. I make these comments:

i. Doctor Lester has been consistent that there is no inference to be drawn from the size of the lymph nodal disease mass. Therefore it is perhaps unsurprising that he did not comment upon it in his report, Further he referred to the PET scan finding, albeit without the specific measurement. He recorded "large left hilar mass with left upper load obstruction with extensive large mediastinal nodes… with likely staging T3 N3 N0." It is clear from his evidence that he did not consider this capable of being information from which any inference could be drawn as to the nodal disease 6 months earlier.

ii. There was no actual error in relying on the Oda paper in Doctor Lester's first report. Perhaps he should have made it subject to a caveat. Nevertheless, my analysis of that paper and, perhaps more importantly, of the Goldstraw paper, shows that as a matter of statistics Doctor Lester was not in error. Indeed the statistical evidence on the N0 status of T1 tumours did not end up being seriously disputed.

74. The defendant suggested that Doctor Lester was somewhat evasive in parts of his cross examination and some suggested examples were given. I do not accept that he was in any way evasive, even in a subconscious manner as Mr Fraser submitted.

75. I do not wish to be unduly critical of Doctor Peake's evidence. He was clearly doing his best to assist the court. If anything, however, I believe it was his evidence that was affected to some extent by confirmation bias. His initial view undoubtedly was that the size of the nodal disease was such that "there is significant uncertainty regarding the staging of Mr King's disease if diagnosed 6 months earlier, however, in my opinion, on the balance of probabilities it would have not have been N0 stage 1 disease." In his letter of 22 December 2019 he regarded it as "almost inconceivable that Mr King would have progressed from N0 to N3 with a 5cm nodal mass within 6 months."

76. In addition the following points can be made:

i. I have rejected the volume doubling hypothesis for the reasons already given. Further, his calculations based on that hypothesis were, as stated, founded on an inaccurate starting point for the purposes of this case (ie based on pathologically present as opposed to clinically present disease).

ii. When Doctor Peake suggested in his letter that in his experience of treating hundreds (probably more than a thousand) patients with non-small-cell lung cancer over the past 25 years, he did not recall ever seeing a patient whose nodal disease progressed so rapidly, and definitely not in whom the primary tumour grew at a typical rate. As Mr Axon submitted this statement did not

bear scrutiny. At best it applied to a minority of patients who were scanned twice, having previously deferred palliative chemotherapy.

iii. From my earlier summary of clinical experience, Doctor Lester's experience is more extensive and more up-to-date in relation to the treatment and management of lung cancer.

iv. If there is any consistency in the rate of growth of squamous cell secondary tumours (contrary to the impression given in respect of adenocarcinomas in the Steel chapter) then it is perhaps surprising that such evidence has not been the subject of any literature. Doctor Peake accepted, when asked, that it would have been perfectly possible for somebody to collect data on patients who had been twice screened, and analyse that data so as to enlighten the medical profession in relation to growth of squamous cell metastases. From his standpoint, Doctor Peake was surprised by the lack of literature (not including the Wang paper). Doctor Lester, on the other hand, said there was simply no evidence from which to draw any inference about the speed of growth of such metastases. This I accept.

77. I have previously set out the evidence as to bronchoscopy. Doctor Peake considered the bronchoscopy evidence as consistent with his viewpoint. Doctor Lester was of the view that squamous cell carcinomas are aggressive in themselves and the penetration of the bronchus did not cast any light on the length of the period of growth of the nodal disease. In final submissions neither counsel really relied upon the bronchoscopy results as really taking the matter any further. Depending upon which oncologist's evidence the court accepted, the bronchoscopy result could be explained accordingly.

Summary

78. Having carefully considered all the evidence, in my judgment the position is:

- The statistical evidence, on the clear balance of probabilities, suggests that a person with a 3cm primary tumour has a 68-75% probability of being N0.

- None of the other literature provides a sound basis for undermining that evidential starting point, taking account of the known progression of the deceased's disease and his ultimate demise. I accept Doctor Lester's opinion that the results of the December 2016 PET scan are not inconsistent with the deceased being N0 in June 2016. As indicated in this judgment where the two oncologists differ, I prefer the evidence of Doctor Lester.

- On analysis as at June 2016 the deceased would have been in the majority of patients who, with a 3cm primary tumour, are N0.

79. I therefore find for the claimant on the one issue before the court.

Appendix A

GLOSSARY OF TERMS

Carcinoma: a cancer arising in the epithelial tissue of the skin or of the lining of the internal organs

Squamous cells: these cells form the surface of the skin and lining of hollow organs in the body and line the respiratory and digestive tracts

Lymphatic system: an organ system in vertebrates that is part of the circulatory system and the immune system. It is made up of a large network of lymphatic vessels, lymphatic or lymphoid organs, and lymphoid tissues. The vessels carry a clear fluid called lymph towards the heart

Lymph nodes: small, bean-shaped masses of tissue scattered along the lymphatic system that act as filters and immune monitors, removing fluids, bacteria, or cancer cells that travel through the lymph system

Hilar lymph nodes: lymph nodes in the hilum of the lung that receive lymph from the pulmonary nodes, and drain to the tracheobronchial nodes

Hilum: the hilum, on the medial side of each lung, is where the main bronchus, pulmonary arteries, bronchial arteries, and nerves enter the lung and where the pulmonary veins, bronchial veins, and lymphatic vessels leave the lung

Mediastinum: a membranous partition between two body cavities or two parts of an organ, especially that between the lungs.

Supraclavicular (scalene): superficial lymph glands and lymphatic vessels of head and neck.

Subcarinal: beneath the carina. In the body the trachea (windpipe) divides into two branches almost in the middle of the chest. The area beneath this branching is the subcarinal region

Appendix B

X-ray, taken on 22 May 2016 and reported on 26 May 2016

"CXR:
No interval change since CXR dated 28/01/2014.
Normal cardiac and mediastinal contours.
There is a 3 cm focal area of increased opacification in the left mid zone and this was not obvious on the previous examination of 28/01/2014. Pathology and even malignancy is difficult to exclude and further assessment with CT is suggested"

X-ray, taken and reported on 25 November 2016

"History: Left-sided chest pain, shortness of breath, recent infection, exclude pneumothorax

PA chest: the 3 cm left mid-zone opacity described on the previous image of 22/05/2016 has increased significantly in size. There is now associated volume collapse. The right lung appears clear. An urgent CT scan/chest physician referral is advised"

CT scan, taken on 6 December 2016 and reported on 7 December 2016 [446, folder 3]

Clinical history: 2 WEEK RULE:::::TO BE SEEN IN 2 WEEK RULE CLINIC-PLEASE BOOK THE SCAN FOR THE 6/12/2016-?LUNG CANCER-REFERRED BY A+E: WORSENING SOB WITH PRODUCTIVE COUGH-SIGNIFICANT WEIGHT LOSS 1.5 STONES OVER 2 MONTHS WITH REDUCED APETITE [sic] AND NIGHT SWEATS-SMOKER 15-20 CIGARETTES PER DAY FOR 40 YEARS. –CXR: PERSISTENT AND WORSENING OPACIFICATION OF LEFT MZ/LZ? LUNG CANCER

CT thorax and upper abdomen post IV contrast.

Comparison made with the prior chest x-ray dated 25/11/2016.

Left-sided large ill-defined hilar/perihilar heterogenous tumour mass infiltrated the anterior mediastinum with significant luminal narrowing and infiltration of the adjacent left main bronchus as well as encased the bronchovascular structures at the left hilar region associated with the large segmental collapse of the left upper lobe and multiple enlarged mediastinal and hilar lymphadenopathy some of them with central necrotic changes particularly at thoracic inlet, paratracheal, precarinal and sub carinal regions with 22-24 mm max SAD. Small sub 11 mm right hilar lymph nodes.

Significant bronchiectasis in the left lower lobe with small shadowing in the left base.
Right lung clear.
Small amount pericardial effusion. No pleural effusion. Multilevel degenerative changes/DDD in the visualised spine.
No evidence of nodules, sinister pathology or metastasis in the visualised upper abdomen.
PET scan/lesion biopsy has to be assessed as a further investigation tool. The above mentioned finding in the left lung compatible likely with bronchogenic carcinoma infiltrated ipsilateral hilar and mediastinal regions with multiple pathological hilar and mediastinal lymph nodes. Additional findings as detailed above.
MDT discussion is advised.
Priority gold star."

PET scan, taken on 14 December 2016 and reported on 16 December 2016 [445, folder 3]

INDICATIONS
Probable left lung tumour, provisional stage IIIB. Left hilar mass with extensive mediastinal contralateral lymphadenopathy-also RMB overgrowth likely from stage nodes.
…
FINDINGS

There is a large left hilar mass with the abnormal area metabolically measuring approximately 4.5 x 3.3 cm and is markedly FDG avid with a SUV max of 26. There is narrowing of the left main bronchus and there is collapse of most of the left upper lobe with a further more peripheral small nodular component of FDG uptake. There are minor changes in the left lower lobe which are probably inflammatory. The right lung field is clear. There are multiple mediastinal nodes including a large subcarinal mass measuring 5 x 3.5 cm (SUV max 26.2), high right paratracheal, anterior mediastinal, lower left paratracheal, pre tracheal and a node anterior to the right main bronchus. More inferiorly there is a node adjacent to the inferior left lateral oesophageal wall.

…

IMPRESSION

1. Large left hilar mass with left upper lobe obstruction with extensive large mediastinal nodes but no distant metastatic disease with likely staging T3 N3 M0, The high FDG uptake is an independent poor prognostic factor."