RATIONAL TESTING

Interpreting and investigating proteinuria

Oliver T Browne, Sunil Bhandari

Department of Renal Medicine, Hull and East Yorkshire Hospitals NHS Trust and Hull York Medical School, Kingston upon Hull HU3 2JZ, UK
Correspondence to: S Bhandari
sunil.bhandari@hey.nhs.uk

Cite this as: *BMJ* 2012;344:e2339
doi: 10.1136/bmj.e2339

This series of occasional articles provides an update on the best use of key diagnostic tests in the initial investigation of common or important clinical presentations. The series advisers are Steve Atkin, professor, head of department of academic endocrinology, diabetes, and metabolism, Hull York Medical School; and Eric Kilpatrick, honorary professor, department of clinical biochemistry, Hull Royal Infirmary, Hull York Medical School. To suggest a topic for this series, please email us at practice@bmj.com.

Proteinuria with or without specific symptoms needs confirmation and quantification

A 46 year old woman who had been treated for an uncomplicated *E coli* urinary tract infection three weeks previously returns to check her urine, as advised by her GP, since her initial urine dipstick test showed traces of protein, blood, leucocytes, and nitrites. She is a lawyer with no important medical history or previous urinary infections. She has recently taken a non-steroidal anti-inflammatory drug (NSAID) for menstrual pain. She has had no recent urinary symptoms or episodes of visible haematuria. There is no family history of renal disease, hypertension, or diabetes, and she is not pregnant.

On physical examination, her blood pressure is 144/82 mm Hg, and there is no oedema. The urine dipstick test shows ++ result for protein but no blood.

What is the next investigation?

Who should be tested?

The prevalence of proteinuria in the general population is about 2% and is higher in elderly people and those with comorbidities.[1 2] Guidelines from the National Institute for Health and Clinical Excellence (NICE), based on systematic reviews of observational studies, recommend screening of groups considered at high risk of chronic kidney disease. These include patients with hypertension, diabetes, vascular disease (cardiovascular, peripheral, or cerebrovascular), a family history of renal disease, or multisystem disease with potential for renal involvement (such as systemic lupus erythematosus) and those taking nephrotoxic drugs.[3] Screening is by estimation of glomerular filtration rate from serum creatinine and calculating urine albumin:creatinine ratio (see box 1 for glossary of terms).

In this case, although the patient is asymptomatic, further evaluation is warranted, primarily because of the presence of proteinuria on a background of NSAIDs, to avoid missing significant renal pathology.

What to look for on physical assessment

Clinical history and examination often reveal few abnormalities unless features of multisystem disease are present or the degree of proteinuria is sufficient to cause physical signs such as frothy urine or peripheral oedema. In the current case further clinical assessment is likely to be negative, but a review of the patient's history for symptoms such as joint pains, weight loss, or fever may be relevant in a case of persistent proteinuria (see box 1) to rule out systemic disorders such as lupus; indeed, a rash may suggest a vasculitis.

Box 1 | Glossary of terms

Persistent proteinuria
The presence of dipstick positive proteinuria in two or more consecutive urine samples over a one to two week period. A previous urinary tract infection may theoretically affect the test, from the presence of leucocytes or alkalinisation of the urine leading to a false positive result for proteinuria. If the evidence is weak, the test should be repeated about two weeks later to check for persistent proteinuria. Repeat tests are more reliable for detecting renal pathology as they reduce the likelihood of transient proteinuria from exercise, stress, or fever.

Postural proteinuria
This is rare in people over 30 years old. Usually it is manifest only when a person stands erect for long periods and disappears on lying down. The diagnosis is made by collecting split urine specimens for comparison. The daytime specimen typically has an increased concentration of protein, whereas a night time specimen has a normal concentration.

Proteinuria and albuminuria
These are an abnormal presence of protein and albumin in the urine. The normal laboratory ranges are
Protein <150 mg/day/1.73 m^2
Albumin <100 mg/day/1.73 m^2.
The composition of urinary protein can be considered to be about 40% albumin and low molecular weight immunoglobulins (including IgA and light chains), 40% secreted proteins (such as Tamm-Horsfall protein, synthesised by the kidney tubule), and the remainder being other immunoglobulins. Dipstick results of + or greater indicate at least 30 mg/dL (equivalent to about 600 mg/day for a 2 litre urine volume) of proteinuria or albuminuria.

Protein:creatinine ratio and albumin:creatinine ratio
Increased ratios of protein or albumin to creatinine in the urine indicate raised protein or albumin as a result of glomerular pathology or hyperfiltration. They are calculated by dividing the urine protein or albumin concentration by the urine creatinine concentration (which is assumed to be excreted uniformly over 24 hours) to take into account differences in urine volume. Normal ranges are
Albumin:creatinine ratio <3.5 mg/mmol for men, <2.5 mg/mmol for women
Protein:creatinine ratio <15 mg/mmol.
An albumin:creatinine ratio of 30 mg/mmol is equivalent to a protein:creatinine ratio of 50 mg/mmol, and a 24 hour protein of 500 mg/day, while an albumin:creatinine ratio of 70 mg/mmol is equivalent to a protein:creatinine ratio of 100 mg/mmol and a 24 hour protein of 1000 mg/day.

bmj.com
Previous articles in this series
- Diagnosis of *Helicobacter pylori* infection (*BMJ* 2012;344:e828)
- Raised inflammatory markers (*BMJ* 2012;344:e454)
- Investigating recurrent angio-oedema (*BMJ* 2011;343:d6607)
- Investigation of "non-responding" presumed lower respiratory tract infection in primary care (*BMJ* 2011;343:d5840)
- Investigating mixed hyperlipidaemia (*BMJ* 2011;343:d5146)

Confirmation of proteinuria

Ideally evidence of persistent proteinuria should be confirmed with an early morning urine sample to exclude postural proteinuria, but this is rarely practical. Urine dipstick testing for protein can detect a urinary concentration of albumin of 100-200 mg/L but is relatively insensitive to other plasma proteins such as immunoglobulin light chains, which might be present in a patient with myeloma-associated renal disease. Dipstick urine analysis is a poor method of quantifying proteinuria. Indeed, over-estimation or under-estimation may be compounded if the result is read manually rather than by an automated urine dipstick analyser. False negative tests are often seen in dilute urine (specific gravity <1.005) and when protein other than albumin is present in the urine. False positives can be seen in concentrated urine, basic urine (pH >8), and in the presence of haematuria.

A midstream urine sample should be sent for culture to exclude urinary tract infection, but the presence of leucocytes and nitrites on urine analysis (81% sensitive and 59% specific, negative predictive value 93%) with symptoms and signs should direct initial therapy because of the potential for contamination of a midstream urine sample, resulting in variable sensitivity (60-100%) and specificity (49-100%) for infection.[4 5]

Quantification of proteinuria (albumin:creatinine ratio or protein:creatinine ratio)

A persistent positive urine dipstick test for proteinuria should prompt sending a sample for laboratory quantification by the ratio of albumin or protein to creatinine (see box 1). A systematic review has shown that testing of a random urine sample by protein:creatinine ratio had a sensitivity >90% to rule out pathology but low specificity (67%) to rule in a diagnosis.[6]

NICE recommends use of albumin:creatinine ratio because it has greater sensitivity (96.8%) than protein:creatinine ratio for low levels of proteinuria, whereas the Scottish Intercollegiate Guidelines Network (SIGN) recommends protein:creatinine ratio.[3 7 8] Historically, measuring proteinuria by means of a 24 hour urine collection was the gold standard, but this is impractical, subject to inaccurate sample collection, expensive, and inconvenient. Studies such as RENAAL have shown that both protein:creatinine ratio and albumin:creatinine ratio correlate well with 24 hour urine measurements (r=0.93 and 0.8 respectively).[9 10]

In the current case the patient's results for albumin:creatinine ratio and protein:creatinine ratio testing are 170 mg/mmol and 240 mg/mmol respectively, confirming significant proteinuria.

Box 2 | Summary of investigations for proteinuria

Basic investigations useful before referral to a nephrologist

Full blood count*
Biochemical profile or estimated glomerular filtration rate*
Protein:creatinine ratio or albumin:creatinine ratio*
Ultrasound scan of kidneys, ureters, and bladder*
Plasma viscosity
Lipids

Other potential specialist tests performed by nephrologist

Complement (C3, C4)
Immunoglobulins
Serum or urine electrophoresis
Rheumatoid factor
Antistreptolysin O titre
Antineutophil cytoplasmic antibody
Antinuclear antibodies
Antibodies to double stranded DNA
Antibody to glomerular basement membrane
Cryoglobulins

*Useful to carry out in primary care

Box 3 | Summary of individual criteria for referral to nephrologist*

Multisystem or collagen disease (such as systemic lupus erythematosus)
Resistant hypertension (>4 antihypertensive drugs including a diuretic)
Family history of renal disease (such as polycystic kidney disease)
Estimated glomerular filtration rate <60 µmol/L/1.73 m^2
Albumin:creatinine ratio >70 mg/mmol or protein:creatinine ratio >100 mg/mmol
Albumin:creatinine ratio >30 mg/mmol or protein:creatinine ratio >50 mg/mmol if associated non-visible haematuria
Nephrotic range proteinuria (protein:creatinine ratio >300 mg/mmol) and hypoalbuminaemia (<30 g/L) refer urgently

*Based on NICE guidelines[3]

Creatinine and estimated glomerular filtration rate

Serum creatinine and estimated glomerular filtration rate should be measured to assess renal function as suggested by NICE.[3] Other tests could be considered as detailed in box 2, but referral to a nephrologist for specific investigations to further characterise the pathology is warranted irrespective of estimated glomerular filtration rate (box 3).

Renal ultrasonography

Guidelines based on expert opinion suggest a renal ultrasound scan should be considered to confirm the presence of two kidneys (a single kidney occurs in 1 in 1000 people and may lead to hyperfiltration and proteinuria), measure kidney size, and identify structural abnormalities such as polycystic kidneys.

Relation between proteinuria and cardiovascular morbidity or mortality

Large epidemiological studies involving the general population, cohorts at high risk for chronic kidney disease (for example, with diabetes), and cohorts with known chronic kidney disease indicate that proteinuria is an independent risk factor for adverse cardiovascular events and faster progression of kidney disease.[11-14] The PREVEND (prospective study of a cohort of 40 845 in the general population) and ADVANCE (observational analysis of 10 640 patients with type 2 diabetes) studies found that both the degree of albuminuria and estimated glomerular filtration rate correlated with increased renal risk (up to 120-fold).[15] The rate of cardiovascular events more than doubled for every 10-fold increase in

LEARNING POINTS

- Proteinuria may indicate important glomerular or tubular pathology related to side effects from drugs such as NSAIDs
- In patients with persistently positive dipstick tests, proteinuria is best quantified by measuring albumin:creatinine ratio or protein:creatinine ratio
- Refer the patient if albumin:creatinine ratio >70 mg/mmol (or >30 mg/mmol if concomitant haematuria); protein:creatinine ratio >100 mg/mmol (or >50 mg/mmol if concomitant haematuria), or other renal pathology is present
- Urgent referral for heavy proteinuria (protein:creatinine ratio >300 mg/mmol) and a low serum albumin (<30 g/L)
- Proteinuria and impaired renal function (low estimated glomerular filtration rate) independently and additively correlate with risk of progression of renal disease and with cardiovascular events

baseline albumin:creatinine ratio (relative risk 2.48 (95% confidence interval 1.74 to 3.52)) and for every halving of baseline estimated glomerular filtration rate (relative risk 2.2 (1.09 to 4.33)).[16] In addition, these two measurements had additive effects on the number of events.

Outcome

Differential diagnosis

This patient has clinically significant proteinuria (protein:creatinine ratio >200 mg/mmol), which invariably indicates glomerular pathology rather than a tubular problem. The wide differential diagnosis could include minimal change glomerulonephritis, focal segmental glomerulosclerosis, membranous glomerulonephritis, and diabetic nephropathy. The temporal association with NSAIDs and the known association between NSAID use and minimal change glomerulonephritis suggest that this is the most likely diagnosis, although >85% of cases are usually idiopathic. Modest proteinuria (protein:creatinine ratio 100-200 mg/mmol) might indicate an alternative of tubular pathology such as drug induced interstitial nephritis from possible antibiotic use or acute tubular necrosis. In this case serum creatinine may be abnormal. Isolated proteinuria of <100 mg/mmol requires monitoring only.

The presence or absence of concomitant haematuria is diagnostically helpful since haematuria is uncommon in patients with minimal change glomerulonephritis, focal segmental glomerulosclerosis, or diabetic nephropathy. The absence of non-visible haematuria is less in favour of other possibilities such as IgA nephropathy, polycystic kidney disease, vasculitis, collagen or multisystem disease, and post-infectious glomerulonephritis. Therefore cessation of NSAIDs may lead to resolution, but referral should be made to a nephrologist (box 3).

Explain to the patient that, because there is significant protein in the urine, further specialist evaluation is required. Also mention that the patient's use of an NSAID might be responsible.

Withdrawal of the patient's NSAID led to complete remission within two weeks, as seen in most cases (over 90% recover within 2-6 weeks after stopping NSAIDs). If this did not occur a trial of corticosteroids might be considered and renal biopsy performed to exclude other pathologies.

Recommend avoidance of NSAIDs. Biannual monitoring of urinary protein is an important means of assessing response to therapy. Therefore, advise subsequent monitoring with dipstick testing (potentially by the patient) and suggest that the patient seek medical advice if oedema should occur as relapses tend to be abrupt and often clinically apparent. Identification of relapses allows early intervention and potential resolution in minimal change glomerulonephritis.

Contributors: All the authors have contributed according to ICMJE guidelines. OTB contributed to planning, drafting, and revising the article and to approval of the final version. SB conceived of the article; contributed to planning, drafting, and revising the article; and approved the final version. SB is guarantor for the overall content. Professors Eric Kilpatrick, Ian Watt, and Unna Macleod reviewed the article and gave advice on its content.

Competing interests: All authors have completed the Unified Competing Interest form at www.icmje.org/coi_discosure.pdf (available on request from the corresponding author) and declare that SB has received honoraria from Pfizer, Amgen, and Pharmacosmos for lectures (all created solely by SB and not influenced by the sponsor); no financial relationships with any organisations that might have an interest in the submitted work in the previous three years, no other relationships or activities that could appear to have influenced the submitted work.

Provenance and peer review: Not commissioned; externally peer reviewed.

Patient consent not required (patient anonymised, dead, or hypothetical).

1 Atkins RC, Briganti EM, Zimmet PZ, Chadban SJ. Association between albuminuria and proteinuria in the general population: the AusDiab Study. *Nephrol Dial Transplant* 2003;18:2170-4.
2 Garg AX, Kiberd BA, Clark WF, Haynes RB, Clase CM. Albuminuria and renal insufficiency prevalence guides population screening: Results from the NHANES III. *Kidney Int* 2002;61:2165-75.
3 National Institute for Health and Clinical Excellence. *Early identification and management of chronic kidney disease in adults in primary and secondary care. Clinical guidelines CG73*. NICE, 2008.
4 Car J. Urinary tract infections in women: diagnosis and management in primary care. *BMJ* 2006;332:94.
5 Jenkins RD, Fenn JP, Matsen JM. Review of urine microscopy for bacteriuria. *JAMA* 1986;255:3397-403.
6 Price CP, Newall RG, Boyd JC. Use of protein:creatinine ratio measurements on random urine samples for prediction of significant proteinuria: a systematic review. *Clin Chem* 2005;51:1577-86.
7 Hutchison A, O'Reilly D, MacCuish A. Albumin excretion rate, albumin concentration, and albumin/creatinine ratio compared for screening diabetics for slight albuminuria. *Clin Chem* 1988;34:2019-21.
8 Scottish Intercollegiate Guidelines Network. *Diagnosis and management of chronic kidney disease*. SIGN, 2008. www.sign.ac.uk/pdf/sign103.pdf
9 Guy M, Borzomato JK, Newall RG, Kalra PA, Price CP. Protein and albumin-to-creatinine ratios in random urines accurately predict 24 h protein and albumin loss in patients with kidney disease. *Ann Clin Biochem* 2009;46:468-76.
10 Lambers Heerspink HJ, Gansevoort RT, Brenner BM, Cooper ME, Parving HH, Shahinfar S, et al. Comparison of different measures of urinary protein excretion for prediction of renal events. *J Am Soc Nephrol* 2010;21:1355-60.
11 Brantsma AH, Bakker SJL, de Zeeuw D, de Jong PE, Gansevoort RT, Group ftPS. Extended prognostic value of urinary albumin excretion for cardiovascular events. *J Am Soc Nephrol* 2008;19:1785-91.
12 Consortium CKDP. Association of estimated glomerular filtration rate and albuminuria with all-cause and cardiovascular mortality in general population cohorts: a collaborative meta-analysis. *Lancet* 2010;375:2073-81.
13 Di Angelantonio E, Chowdhury R, Sarwar N, Aspelund T, Danesh J, Gudnason V. Chronic kidney disease and risk of major cardiovascular disease and non-vascular mortality: prospective population based cohort study. *BMJ* 2010;341: c4986.
14 Hemmelgarn BR, Manns BJ, Lloyd A, James MT, Klarenbach S, Quinn RR, et al. Relation between kidney function, proteinuria, and adverse outcomes. *JAMA* 2010;303:423-9.
15 Van der Velde M, Halbesma N, de Charro FT, Bakker SJL, de Zeeuw D, de Jong PE, et al. Screening for albuminuria identifies individuals at increased renal risk. *J Am Soc Nephrol* 2009;20:852-62.
16 Ninomiya T, Perkovic V, de Galan BE, Zoungas S, Pillai A, Jardine M, et al. Albuminuria and kidney function independently predict cardiovascular and renal outcomes in diabetes. *J Am Soc Nephrol* 2009;20:1813-21.

Accepted: 29 December 2011

A PATIENT'S JOURNEY

Superficial spreading melanoma

Penny D'Ath,[1] Penny Thomson[2]

[1]Department of Optometry and Visual Science, City University London, London EC1V 0HB, UK
[2]Barnet and Chase Farm Hospitals NHS Trust, Barnet Hospital, Barnet EN5 3DJ (penny.thomson@.nhs.net)
Correspondence to: P D'Ath p.j.d'ath@city.ac.uk

Cite this as: *BMJ* 2012;344:e2319
doi: 10.1136/bmj.e2319

This is one of a series of occasional articles by patients about their experiences that offer lessons to doctors. The *BMJ* welcomes contributions to the series. Please contact Peter Lapsley (plapsley@bmj.com) for guidance.

This patient was diagnosed with superficial spreading melanoma, which had spread to the lymph nodes. Treatment seems to have been successful, but she has been perturbed by some clinicians' reluctance to discuss prognosis

"You do understand this is cancer?" asked the dermatologist. It would be another few months before I fully understood the significance of this sentence and its implications. At that precise moment, it was just a mole: asymmetric, with irregular borders, of different colours, bigger than the diameter of a pencil, and elevated. In fact, everything the ABC rules of dermatology said it shouldn't be. My GP had said it would probably be fine, and I could leave it alone. Only it hadn't been fine, and it was lucky I hadn't left it alone. Except that it had been left alone long enough to march unrepentantly to my lymphatics, where it had settled comfortably into my sentinel node.

The dermatologist had just confirmed Google's tentative diagnosis of a superficial spreading melanoma. She mentioned survival rates. What? Along with the rest of the population, I thought that if it was malignant they would cut it out and that would be the end of it. Survival rate? This was a new concept, and one that I hadn't entertained.

My mole had a low mitotic rate (good), minimal inflammation (good), and no ulceration (good). On the other hand, it had a Breslow thickness of 1.8 mm and was Clark level IV, meaning that it was neither early nor thin. It was a mole with a mission. I found it incredible that something so thin—1.8 mm for goodness sake—could kill.

I returned one week after the wide excision and sentinel node biopsy to receive my results. The odds were heavily in my favour, as 80% of patients have no sentinel node involvement. The surgeon rather overplayed the fact that the wide excision was clear, and I could tell by his eager delivery of this result that the next one would be less favourable. I was right; the sentinel node was positive.

I returned for axillary clearance. The "likely" side effect of lymphoedema frightens me more than anything else. Not only cosmetically (who wants to look like the Michelin man?) but also functionally (I am very right hand dominant).

The results were good: 15 nodes removed, none cancer-

A DOCTOR'S PERSPECTIVE

When Penny first showed me the mole on her abdomen, I experienced that sinking feeling that a dermatologist feels when they are fairly sure that they are staring at a new presentation of a melanoma. I had been suspicious after taking the history: a longstanding mole that had changed shape and colour in a patient with very pale skin. Penny had been born in England but had spent much of her youth in sunny countries. When I asked her if she had ever worn any sun protection as a child, she laughed and said, "We wore nothing." Penny's brother had already been diagnosed with a melanoma. In addition, because of her seronegative spondyloarthritis, she had previously taken several immunosuppressive drugs, some of which are believed to increase the risk of developing cancer.

At this point it is difficult to know whether to be completely open and voice your suspicions or wait until you have histological confirmation. This is where I try to "feel" what the patient wants to know at this stage. I told Penny that the mole needed to be removed and offered to excise it at the end of the clinic. As she agreed straightaway, there was no need to heighten her anxiety by saying that it needed to be removed urgently. Neither of us talked about "melanoma." I removed the mole on her abdomen, giving it a small margin of surrounding normal skin. I noted that she had several other unusual looking moles and wondered if she had the dysplastic naevus syndrome, which increases a person's likelihood of developing melanoma.

The histology report was verified after 10 days and confirmed my clinical suspicion—a superficial spreading malignant melanoma with a Breslow thickness of 1.8 mm. The histology results were reviewed at the local and regional skin multidisciplinary team meeting, and further treatment and investigations were recommended.

I brought Penny back to the clinic and braced myself to give her news that I thought she would not be expecting. Do I just come out with the words "I am sorry but it is skin cancer" or do I work up to it slowly, firing "shots across the bow" as I was taught in my National Communication Course. After one consultation, how can you gauge how best a patient will take bad news?

I always break bad news in the clinics with our skin cancer nurse specialist present, as support for the patient. My previous consultation with Penny suggested that she would rather be told any bad news straight out—I hoped I had judged right.

When I explained the diagnosis to her, she looked almost relieved and said she had suspected that this would be the case. I explained that further surgery would be required to remove some more skin from around the scar, but that we would also recommend her having a sentinel lymph node biopsy from the draining lymph node basin. This staging investigation can be offered to patients who have had a melanoma removed with a Breslow thickness over 1 mm. I dictated a referral to the plastic surgeons who would perform the surgery at the regional skin cancer centre, and arranged to see Penny again after her surgery so that I could continue her skin surveillance and arrange any further investigations. I gave Penny the contact details of our cancer nurse specialist, who was also named as her key worker—the person to contact if she had any worries, fears, or delays in appointments.

I asked Penny if she had any further questions. Very rarely, in all the years that I have been giving bad news regarding skin cancer, has anyone asked me "How long have I got?"

I was relieved when Penny smiled again and said she had no more questions and that we would meet up again after her surgery.

Penny Thomson

RESOURCES FOR PATIENTS AND CLINICIANS

Macmillan Cancer Support (www.macmillan.org.uk/Cancerinformation/Cancertypes/Melanoma/Melanoma.aspx)—UK charity providing information on malignant melanoma, including how it is diagnosed, possible treatments and side effects, and how to get further support

Cancer Research UK (http://cancerhelp.cancerresearchuk.org/type/melanoma/about/)—UK charity providing information about melanoma, including survival rates and prognosis

British Association of Dermatologists (www.bad.org.uk/Portals/_Bad/Guidelines/Clinical%20Guidelines/RCP%20Melanoma%20Guidelines%202007.pdf)—Professional organisation providing information and guidelines on "prevention, diagnosis, referral and management" for melanoma

Goldstein BG, Goldstein AO. Diagnosis and management of malignant melanoma. *American Family Physician* 2001;63:1359-69 (www.aafp.org/afp/2001/0401/p1359.html)—Informative article in a peer reviewed journal

SkinCancer Net (www.skincarephysicians.com/skincancernet/)—US website from the American Academy of Dermatology. Provides information on melanoma, including staging (/staging.html) and recurrence (/melanoma_returns.html)

ous, stage IIIa regional metastasis. My upper arm and shoulder are numb, but this is a small price to pay. I was elated. I opened a bottle of champagne and got drunk. Then came the questions.

The plastic surgeon told me that the survival rate for people with my condition was above 90%. This couldn't be right. I had understood that it had been above 90% before they knew the cancer had marched triumphantly to the sentinel node and planted its flag. Thankfully, it hadn't started its ascent towards the summit, but was firmly ensconced in base camp at two separate locations.

The dermatologist told me survival was 67% at five years. One in three people would be dead in five years? "If that's the way you want to look at it," she said with exasperation in her voice, which rather suggested she wished I hadn't vocalised this. It wasn't that I wanted to see it that way; rather I couldn't really believe I might be staring my mortality in the face at the age of 41. I don't really believe I will die, because I am only 41 and it's only a mole. Also, I know the dermatologist won't let me die—even though, deep down, I understand that if the scud missile has me on its radar, there is nothing she can do. She explained that they knew there was no cancer where the mole had been (hence the wide excision) nor in the lymph nodes (hence the axillary clearance). What no one knew was whether there were any micrometastases in transit between the mole site and the lymph nodes. This was the piece of information that I lacked.

Perhaps I should have mentioned earlier that I am a "complicated" patient as there are various possibilities as to why I had this melanoma. The commonest cause is sun exposure, but I am definitely not an ardent sun worshipper. It is more probably because of the biologicals (biologically derived drugs) I take for my seronegative spondyloarthropathy, which increase the risk of tumour because of their immunosuppressant nature. Or perhaps it's because of family history; my brother had a melanoma. Or perhaps because I lived in west Africa as a baby and toddler in the days before sun cream (but also in the days when we had an ozone layer). Or maybe because I have that pasty Scottish tartan skin which my dermatologist informs me is Fitzpatrick type II. Or maybe a combination of all these factors.

Finally, I had an appointment with the oncologist, who delivered his monologue. Did I wish to go back on the biologicals that everyone seemed to see as the cause of my melanoma? I wasn't sure. My understanding is that melanoma at this stage is sneaky and aggressive and resistant to the usual chemotherapy channels available for other cancers. Treatment is usually adjuvant therapy in the form of interferon or clinical trials (such as bevacizumab). The oncologist concluded that, because I have arthritis and despite the rather disturbing fact that every other patient with stage IIIa melanoma is offered it, I should not be offered adjuvant therapy. He thought interferon would aggravate the arthritis as it stimulates the immune system, and I would not be eligible for the bevacizumab trial because of the arthritis. Did he ask me for my opinion? No, he discharged me and abandoned me to my fate. I left, not understanding the likelihood of recurrence or my chance of survival. I found the door effectively closed in my face with the same recurring thought: melanoma kills, arthritis does not.

To summarise the findings of my mole, my vocabulary now included terms such as superficial spreading melanoma, sentinel node, Breslow thickness, axillary clearance, lymphoedema, micrometastases, and adjuvant therapy, but I still didn't really understand the process of recurrence and survival rates. Not everyone with recurrence dies, right? And you can't die if you have no recurrence (excluding other causes like being hit by a bus), so why did these figures not add up? The oncologist had discharged me, which, to me, rather suggested that I was not worth saving.

I didn't actively seek a second opinion. I merely emailed my rheumatologist to update him. This was normal as my care was confusingly spread over three hospitals. The rheumatologist emailed back immediately saying he thought that I shouldn't automatically be excluded from further treatment because of my arthritis if this is what would normally happen. He then (bless him) referred me to the oncologists at his hospital.

Weeks later, I faced a new oncologist. She disagreed with the previous oncologists and enrolled me in the bevacizumab trial. This is really confusing. Two centres run identical trials, yet I am eligible for one and not the other? The cynic in me wonders if one of these centres is skewing its results. This oncologist didn't dodge my questions but agreed with me that I needed to know the facts so that I could make informed decisions. My feeling was that everybody was expecting the cancer to recur but nobody was saying it. Yes, she said, that is exactly what they were all thinking. With all the other factors (arthritis, family history) thrown in and a "significant chance of recurrence," she gave me a low five year survival probability (50%).

I haven't taken my biologicals for one year now but must decide if I wish to restart them. My rheumatologist informs me that there is "a small but measurable risk" in terms of melanoma. I cannot ignore the fact that I have chronic arthritis and must balance the quality of my life with the risk the biologicals pose. There may be no issue anyway as I may already be cured. I don't know. The excellent team who monitor me so closely don't know. But, I have every confidence that this team will "sherpa" me to the top of the five year mountain, where I will triumphantly plant my very own flag.

Competing interests: None declared.
Provenance and peer review: Not commissioned; not externally peer reviewed.
Accepted: 25 November 2011