The cost of ignoring acute kidney injury

Andrew Lewington1 and Peter Hall2

1Renal Medicine, Leeds Teaching Hospitals NHS Trust, Leeds, UK and 2Leeds Institute of Health Sciences, University of Leeds, Leeds, UK

Correspondence and offprint requests to: Andrew Lewington; E-mail: andrew.lewington@leedsth.nhs.uk

The National Confidential Enquiry into Patient Outcomes and Death (NCEPOD) Adding Insult to Injury AKI reported in 2009 that only 50% of patients who died with a diagnosis in the UK received good care [1]. This was a damning report that kick started a programme of work in the UK to better understand the syndrome of AKI, its epidemiology and impact. The journey has been an interesting one which has now been championed by the National Health Service (NHS) England as a major patient safety issue. However it is important not to lose sight of the complexity of the syndrome and continue to develop the research programme to understand the pathophysiology which will in turn drive forward the development of new therapies. The National Institute of Health and Care Excellence (NICE) AKI clinical practice guideline published in 2013 [2] will soon be followed by a quality standard to drive a quality improvement programme.

The initiatives in the UK have been driven forward by the recognition of the cost of AKI to the NHS and the potential for prevention and associated cost savings [3]. From a health economics perspective the paper published in this month’s edition of Nephrology Dialysis Transplantation by Marion Kerr et al. is an important piece of work which will support the much needed global drive to look for ways to reduce the burden of acute kidney injury (AKI). It has been made possible by the availability of national costing data in the UK which has been collected over the last 10 years in support of the current commissioning structure based around Hospital Episode Statistics (HES). It should also promote more use of this data in other disease areas.

It is now important to put the data under more detailed scrutiny by unpinning which subgroups are contributing most to the burden of illness and cost and in particular which are potentially reversible. If this could be achieved there would be an opportunity to potentially reduce the economic consequences substantially. This can only be achieved by combining the national datasets with more granular clinical data, typically collected at local level without national standardization. It is therefore imperative that there is more investment in performing more detailed retrospective audit and the prospective collection of data by national registries. This would enable healthcare professionals to start to attribute the causality of AKI and suggest optimal management/prevention strategies. Central to this process will be mapping out the process of care in adequate detail. It must be emphasised that AKI is a syndrome with many causes and must not be all lumped together as one entity if we are to ultimately make sense of it and enable a transformational change in its management and outcomes [4].

suggested area to focus on would be with more tightly defined patient groups such as patients developing AKI post cardiac surgery or vascular surgery and then progress to more heterogeneous populations like acute medical or surgical admissions.

Further work now needs to be carried out to investigate the magnitude and cost of AKI diagnosed in the primary care. The authors refer to the ‘NHS Costs of Care’ when what they report is the NHS secondary care inpatient costs of care which excludes the NHS primary care, community and secondary care outpatient costs. With the inclusion of general practice (GP) records within data accessible through the Health and Social Care Information Centre (HSCIC) it should be possible to do this in the near future [5]. There is a chance for the Department of Health (DoH) to permit access to this data by researchers in addition to the commissioners to whom it is currently limited.

This paper demonstrates the ability to diagnose the incidence and prevalence of AKI using laboratory databases. Recently in the UK there has been national agreement to adopt a uniform AKI algorithm based on a modified form of the Kidney Disease Improving Global Outcomes (KDIGO) AKI definition [6]. With the advent of the National Reference Laboratory, the time has come when we should be able to gather all the serum creatinine levels across all laboratories at a national level. However for this data to be used in a uniform manner across the UK for comparisons to be valid on the epidemiology of AKI the method of measurement of serum creatinine must be standardized. All biochemistry laboratories should measure serum creatinine using an enzymatic method as recommended by The Renal Association AKI guidelines [7]. NHS England has identified AKI as a priority patient safety area with workstreams dedicated to the biochemical detection of AKI. There is much work going on at local level to establish AKI patient pathways. This will provide the opportunity to capture more detail on episodes of AKI.

There is a clear message that the use of data from the HSCIC (i.e. HES) under-estimates the incidence of AKI in hospital admissions by KDIGO AKI criteria. The analysis of HES only included ICD10 N17 and N280. (‘Acute renal failure’ and ‘Ischaemia and Infarction of the kidney’). There are many other diagnoses that were not included which fall within the syndrome of AKI and probably explains the under-representation in the HES extract compared with the NHS laboratory-based figures.

The current coding of AKI in the UK is not fit for purpose. The HRG code LA07 is used to assign a cost to a patient admission dominated by an AKI diagnosis. The LA07 code is broad and does not map well with the KDIGO AKI criteria [e.g. nephrotic syndrome with normal creatinine is included, whilst AKI secondary to a myocardial infarction (MI) would not be included]. It can include various levels of concomitant comorbidity, different durations of inpatient stay, outpatient management, elective and non-elective management. HRG-based costing is therefore limited as the HRG system is designed to support commissioning rather than accurately reflect the costs associated with a specific diagnosis. The only solution to this really is to conduct expensive and time-consuming ‘micro-costing’ whereby data are collected directly from the clinical notes/record, supplemented by observer studies (e.g. an observer sitting on an acute medical work and documenting activities conducted in the management of a patient with a new diagnosis of AKI). However there are new initiatives in setup/pilot phase across the UK referred to as Patient Level Costing and Information Systems. These are locally implemented costing platforms implemented in alignment with national methods recommendations which will provide much more granular and clinically valid cost estimates.

Following the publication of the NICE AKI guideline, a national headline reported that ~12 000 deaths annually in the UK may be associated with AKI. It is important to note that causality (proportion of deaths directly attributable to AKI) remains unproven and more research necessary to support such bold statements. Similarly, the annual AKI-related cost is estimated as £1.02 billion per year. However the AKI-related cost is not AKI-attributable cost, and more research is needed to unpick how much of these costs would have occurred in the same ill patients if they had not had AKI along with accompanying illnesses. The same principle applies for the lifetime cost of post-discharge care. Attributing causality both for mortality and excess costs is absolutely essential if healthcare is going to correctly target policy or interventions to reduce the burden of AKI.

As with all research, it is important to consider the source of the funding. The research was commissioned by NHS Kidney Care which could be considered to be an organization with aspirations to increase the current level of investment in the research and treatment of AKI. That being said this article should now provide strong support for further independent research into a syndrome that has long been overlooked on a global scale and is still poorly understood [8]. The International Society of Nephrology (ISN) has launched the 0 by 25 initiative with the aim of preventing the unnecessary deaths of patients in the poorest parts of the low income countries by 2025. A first step will be to describe the epidemiology of AKI in these countries and then understand its economic impact. The American Society of Nephrology has recently attempted to identify the epidemiology with interesting results [9]. The paper by Kerr et al. demonstrates that this initial goal is attainable and represents a paradigm to be replicated elsewhere.


In Focus

References


Almost all of the papers dealing with chronic kidney disease (CKD) invariably start underlining that morbidity and mortality rate of this patient population is unacceptably high. Conversely, very few papers have considered quality of life and none selected quality of life as the primary end point. This reflects a clear bias for doctors and investigators, possibly because the treatment of CKD, especially in stage 5 on dialysis or with transplantation, is life-saving. Thus, the efforts of nephrologists were and are still concentrated in reducing the mortality and the morbidity of patients, without carefully considering quality of life. This attitude has been amplified by the influence of evidence-based medicine and regulatory authorities: the only end points that really matter are the hard-ones. Surrogate end points, especially when they are not easily quantifiable, are considered often insufficient to prove the efficacy of a given treatment. This process has markedly improved the scientific quality of clinical trials. However, we are reaching the paradox that no clinical trial is capable of proving efficacy on hard end points. Recently, almost all large, well-designed clinical trials testing a variety of treatments have given negative results. This has led physicians back to the importance of individualizing the cure of the single patient according to his characteristics and it is revaluating a comprehensive view of the possible associated benefits of a given treatment.

Apart from treating hypertension and dyslipidaemia, anaemia correction is an important potential area for reducing cardiovascular morbidity and mortality in CKD. Before the availability of erythropoiesis stimulating agents (ESA), CKD patients, especially those on dialysis, were heavily anaemic and symptomatic. Blood transfusions partially and temporary alleviated symptoms, but at the price of the risk of viral infections, iron overload and sensitization, which could jeopardize the possibility of receiving a transplant.

Since the early 1990s, the introduction of ESA has revolutionized the quality of life of CKD patients, who previously had very limited possibility not only to maintain a job, but also to move: they could barely survive and did not enjoy life. Doctors very soon realized the potential of ESAs. These agents were very effective in increasing haemoglobin (Hb) levels even to normal values. A striking improvement in quality of life was the second most evident effect. However, despite this marked improvement in patient well-being, in 1989 the Food and Drug Administration (FDA) approved Epoetin alfa to elevate or maintain the red blood cell level and to decrease the need for transfusions, without even mentioning quality of life.

Observational data from large databases have invariably shown a clear relationship between anaemia and outcomes: the worse the anaemic status, the worse the patient mortality and morbidity [1, 2]. The possibility of improving survival and reducing comorbidities through anaemia correction became the aim of several clinical trials. Perhaps forgetting that mild anaemia may have a compensatory role in CKD patients, trials were designed to compare near-to-normal Hb target ranges with partial anaemia correction.