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Comments to the editor:
Dear Editor,

We are pleased to submit our paper entitled 'Epistaxis and mortality' for consideration for publication in Rhinology. This paper represents further analysis of data from the National Epistaxis Audit 2016 conducted with ENT UK, The British Rhinological Society and INTEGRATE the National ENT Trainee Research Network. Given the size of the prospective audit, this study is uniquely placed to comment on mortality related to the most common emergency presentation in ENT. The higher than expected mortality that we observed at 3.4% could have implications for the future management of epistaxis referrals to ENT professionals worldwide. Epistaxis and mortality remains a poorly represented topic in the international literature but the present study hopes to address this deficit.

Thank you again for your consideration.

Yours Faithfully

John Hardman

SUMMARY

Background: Epistaxis is a common emergency presentation to ENT. The 'Epistaxis 2016: national audit of management' collected prospective data over a 30-day audit window in 113 centres. A 30-day all-cause mortality rate of 3.4% was identified. This study examines in more detail the subgroup of patients who died during the audit period.

Methodology: There were 985 eligible patients identified. Of these, 33 patients died within the audit period. WHO bleeding score, MEWS score, haemostasis time, source of referral, co-morbidities and cause of death were investigated from the dataset.

Results: Patients who died were more likely to come from a ward environment, have co-existing cardiovascular disease, diabetes, have a bleeding diathesis, be on antithrombotic medication or have received a blood transfusion. Patients did not die from exsanguination.

Conclusions: Epistaxis may be seen as a general marker of poor health and a poor prognostic sign.

Key words:
Epistaxis; mortality; audit; haemorrhage.

INTRODUCTION

Epistaxis continues to be a significant burden to patients and the wider healthcare economy.[1] It is the most common emergency presentations referred to ENT on call services.[2] The 'National Epistaxis Audit: 2016 ' (NEA2016) aimed to understand our current management of epistaxis in the UK and identify areas for improvement in care.[3] This study constituted the largest prospective cohort study of epistaxis and its outcomes to date and further demonstrated the power of trainee-led collaborative research in ENT. The INTEGRATE network, that facilitated the multicentre project, allowed the collection of high quality data for significantly lower costs than previous similar national studies.

The NEA2016 reinforced our understanding of epistaxis as a common condition that affects patients of all ages and backgrounds. Although outcomes were generally good, one of the more surprising outcomes from the audit was the higher than expected 30-day all-cause mortality rate of 3.4%. In orthopaedics, the high mortality associated with fractured neck of femur prompted the specialty to address this reality. In 2010 the National Hip Fracture Database was established with the aim of improving outcomes for affected patients.[4] Is it time for epistaxis to go the same way?

We have long known that some of the sickest patients seen in ENT are those presenting with epistaxis but this phenomenon has not been fully explored by the current literature. Are our patients ultimately dying as a direct result of epistaxis or should epistaxis be seen as more of a symptom of severe morbidity related to other causes? Moreover, are there any common characteristics of patients dying after an episode of epistaxis that we can learn from? This study aims to look in more detail at the subgroup of patients presenting with epistaxis who subsequently died within 30-days of their referral to ENT.

METHODS

This study used anonymised data from the 2016 National Audit of Epistaxis Management.[3] The following summarises the audit methodology. Further information can be found in the referenced publication.

The audit period was 30-days from 7th November 2016. Unscheduled presentations of epistaxis to acute ENT services aged 16 and over were eligible for inclusion. Patients given telephone advice only and those who were seen in scheduled outpatient appointments were not included.

Sites were recruited using open advertising via the Association of Otolaryngologists in Training (AOT) and through a national network of ENT trainees identified by INTEGRATE. Data were subsequently entered by 113 participating sites across the United Kingdom.

Data were entered prospectively through an online portal to the Data Safe Haven hosted by the University College London. Communication with the server was via a 256-bit Secure Hash Algorithm encryption and the server itself was certified to ISO27001 and conformed to the NHS Information Governance Toolkit standards.

Data were collected on patient demographics, co-morbidities, antithrombotic medications, bleeding severity, Modified Early Warning System (MEWS) scores, source of referral and cause of death. Data were also collected on the management on epistaxis including nasal packing, cautery, blood transfusions, surgery and use of interventional radiology.

Thirty-days following initial presentation, all patients' case notes were reviewed to identify adverse outcomes including re-presentation to hospital, myocardial infarction, pulmonary embolus, cerebrovascular accident, deep vein thrombosis and death. A clinical coding search was also conducted at 30-days following closure of the audit window in order to identify any missed presentations of epistaxis. Data from missed cases were entered retrospectively onto the database and highlighted as such.

The study steering committee had oversight of the data submissions in real time. This allowed timely support and feedback to the study leads during the audit and data collection window. Following the audit window, the data were scrutinised for duplications, errors and incomplete entries and site leads contacted to amend entries and improve data quality where possible.

Site leads were required to register the audit with their audit departments. They were also required to contact their local Caldicott Guardian to obtain approval for the data collection methodology. Formal ethical approval was not required, as per the NHS Research Ethics Committee guidance. Patient information posters were displayed at participating sites advising patients that data were being confidentially collected but that patients could have their information excluded if desired.

Formal statistical support was provided. Haemostasis time was defined as time from presentation to ENT until the time at which final haemostasis was achieved prior to discharge.

For comparison of means, an unpaired t-test was used with two-tailed p values. For comparison of categorical data, Fisher's exact test was used with two-tailed p values.

RESULTS

A total of 1,122 patients were included in the final analysis in the NEA2016 dataset. For the purposes of this study, a further 137 patients were excluded as their record did not specifically note an outcome for mortality at 30-days following initial referral to ENT services. Of the remaining 985 patients eligible for analysis, 33 were identified as having died within the 30-days and constituted our 'deceased' sub-group. The 952 patients who were alive at 30-days follow up were used as a comparator 'alive' sub-group.

Overall median age was 73 years but was higher in our deceased group than in our alive group (75 years (IQR 66,84) vs 73 years (IQR 62,82) respectively). The overall 30-day mortality rate for the NEA2016 cohort was 3.35% (n=33/985). The rate for males was 3.62% (n=20/552) and for females was 3.00% (n=13/433). The Office of National Statistics publish the cohort life expectancy each year, based on UK life tables.[5] The 2016 data give the comparative 30-day mortality rates for 73 year old males and females of 0.080% and 0.056% respectively. There was a male preponderance in both groups, representing 60.6% (n=20) of the deceased and 55.9% (n=532) of the alive group.

Table 1 demonstrates the prevalence of relevant comorbidities in both the deceased and alive sub-groups. Of note, 21.2% (n=7) of the deceased group were identified as having a bleeding diathesis compared to 3.7% (n=35) of the alive group (p=0.0003). Prevalence of diabetes, liver failure, thrombocytopaenia and heart disease were also significantly higher in the deceased group.

Overall levels of anticoagulation were high in both sub-groups [table 2]. Rates of treatment with heparin products was notably higher in the deceased group (15.2% vs 1.9%, p=0.001).

Location of referral differed between the two groups. In the deceased group, 45.5% (n=15) of patients were referred via the Emergency Department and 54.5% (n=18) were inpatients referred from a ward. Comparatively, in the alive group, 89.8% (n=848) of patients presented via the Emergency Department, 5.8% (n=55) were referred from a ward and 4.3% (n=41) presented via their GP.

Epistaxis and Mortality

Figure 1 shows the distribution of Modified Early Warning System (MEWS) scores at presentation for the two subgroups. The median MEWS score in both groups was 1. The mean MEWS value for the deceased group was not significantly different to the alive group (1.54 compared with 1.35, p=0.45). The data were more widely distributed about the mean in the deceased group with a standard deviation 1.62 compared with 1.28 for the alive group.

Figure 2 shows the distribution of World Health Organisation (WHO) bleeding scores at presentation for the two subgroups. The median WHO score in both groups was 2. The mean WHO score for the deceased group was not significantly different to the alive group (1.97 compared with 1.94, p=0.66). The data were more widely distributed about the mean in the deceased group with a standard deviation 0.68 compared with 0.37 for the alive group.

All patient had a recorded 'haemostasis time' at which point the epistaxis was deemed controlled. As such no patient in this audit died directly from exsanguination from epistaxis. The median haemostasis time was shorter for the deceased subgroup at 11.3 hours (IQR 0.6, 20.4), versus 18.3 hours (IQR 0.6,20.4) in the alive subgroup.

Ultimate cause of death was available for 23 of the 33 deceased patients (Table 3). Broadly grouped, 34.8% (n=8) patients died primarily due to an infective cause, 30.4% (n=7) from a primarily cardiovascular cause, 30.4% (n=7) directly secondary to malignancy and a single patient (4.3%) from liver failure. Cardiovascular disease was the most common cause of death reported overall, contributing to the deaths of nine of the 23 patients (39.1%). No deaths were attributed to haemodynamic shock.

	Deceased at 30 days (33/33)		Alive at 30 days (952/952)		Fisher's exact
Characteristic	**n**	**%**	**n**	**%**	**p**
History of Epistaxis	7	21.2	240	25.2	0.688
History of Hypertension	21	63.6	523	54.9	0.376
Bleeding Diathesis	7	21.2	35	3.7	0.0003*
Liver Failure	2	6.1	7	0.7	0.034*
Vitamin K Deficiency	0	0.0	0	0.0	1.000
Haemorrhagic Telangiectasia	0	0.0	6	0.6	1.000
Haemophilia	0	0.0	1	0.1	1.000
Willebrands	0	0.0	0	0.0	1.000
Thrombocytopaenia	3	9.1	12	1.3	0.012*
Diabetes	10	30.3	130	13.7	0.018*
Heart Disease	16	48.5	279	29.3	0.031*

Table 1: Comorbidities. *represents statistical significance

	Deceased at 30 days (33/33)		Alive at 30 days (952/952)		Fisher's exact
Anticoagulant	**%**	**n**	**%**	**n**	**p**
Blood Thinning	66.7	22	56.5	538	0.286
Aspirin	24.2	8	19.1	182	0.500
Clopidogrel	9.1	3	9.9	94	1.000
Heparins	15.2	5	1.9	18	0.001*
Warfarin	30.3	10	19.4	185	0.124
Oral Anticoagulants	15.2	5	13.4	128	0.794
Other	0.0	0	1.6	15	1.000

Table 2: Anticoagulant usage. *represents statistical significance

Cause of death	
Principle	**Contributing**
Brain metastasis	Melanoma
Lung cancer	-
Lung cancer	-
Lung cancer	Pulmonary embolus
Lung metastases	-
Metastatic cancer	-
Myelofibrosis	-
Heart failure	-
Heart failure	-
Heart failure	Ischaemic heart disease
Heart failure	-
Ischaemic bowel	-
Myocardial infarction	Renal failure
Post op 'cardiac surgery'	-
Liver failure	Alcoholic liver disease
Neutropenic sepsis	Acute myeloid leukaemia
Pneumonia	Alcoholic liver disease
Pneumonia	-
Pneumonia	COPD
Pneumonia	Heart Failure
Pneumonia	Myeloma
Pneumonia	Heart Failure
Cellulitis	Renal failure

Table 3: Cause of death

DISCUSSION

The overall 30-day mortality rate for our cohort was significantly higher than for the general age-matched UK population. Interestingly, the disparity was greater for men than women (3.35% and 0.080% vs 3.00% and 0.056%). Although patients presenting acutely to a hospital would be expected to have a higher mortality rate than the general population, the observed rate was felt to be higher than expected. As such, high mortality in any patient group should prompt extra scrutiny. In other conditions, a high mortality rate has prompted national strategies for standardising and improving care.[4] Alongside the 2016 national audit, the British Rhinological Society have produced consensus guidelines for the care of epistaxis patients.[6–11] It is hoped that these consensus guidelines will reduce variations in management that may be contributing towards suboptimal outcomes and may be used as the basis for future national audits to ensure improvement in our standards of care.

Significantly more patients were referred to ENT for epistaxis from wards in the deceased group than in the alive group (54.5% versus 5.8%). Patients referred to ENT from an inpatient setting will inevitably have a concurrent or pre-existing co-morbidity serious enough to have already required hospitalisation. These patients are therefore likely to represent an inherently more morbid group than those referred from ED or the community. Acute ENT services should, therefore, be mindful of this increased incidence of death in patients from an inpatient setting and should ensure close joint management with their referring teams.

Khan et al. 2014 looked at medicolegal claims related to epistaxis in patients treated in the USA. They identified four patients who brought legal claims related to nasal packing. In two of these cases the patient subsequently died after aspirating their nasal packs during extubation.[12] Our analysis did not highlighted any complications resulting directly from the mismanagement of epistaxis. However, it is worth noting this potential for harm from nasal packing, particularly in patients with altered consciousness.

There were no data to suggest that any of the patients in this study who died in the audit period had died as a direct result of the epistaxis itself [table 3]. Hypovolaemia, exsanguination and/or anaemia were not recorded as causes of death for any patient. It

appears instead that these patients had ultimately succumbed to underlying or pre-existing illnesses. However, it is possible that the blood loss associated with epistaxis may have contributed towards an increased morbid state.

Seven cases of death associated with epistaxis were described by Woolf and Jacobs in their 1961 publication relating to admissions to four hospitals over a 6 year period.[13] These case reports suggest that hypovolaemia and/or anaemia may have been a more significant contributory causes of death than seen in the present study. This change may reflect the improvement in acute care delivered in Emergency Departments, with the adoption of standardised management algorithms related to haemorrhage, such as described in the Advanced Trauma Life Support programme.[14]

Around a third of the patients in the deceased group died primarily as a result of cardiovascular causes, a third from infective diseases and a third from malignant causes [table 3]. Unfortunately, in the UK, the Office for National Statistics does not publish mortality data that specifies epistaxis as a specific cause. There are US data available that reported only four of the 2.4 million deaths recorded in 1999 were a result of epistaxis.[15] This very low rate should not diminish the significance of the higher than expected rate we have identified in this series. It is probable that rates of epistaxis related deaths are under reported.[13] It is hoped the NEA2016 will go some way towards increasing the awareness of epistaxis related deaths, both so this rate can be better appreciated and that care may be improved for this potentially very sick cohort of patients.

Woolf and Jacobs also questioned the role for blood transfusion in epistaxis and advocated early transfusion, especially in the presence of pre-existing cardiovascular disease.[13] Twenty one percent of our deceased group were WHO group 3 and so received a transfusion during their treatment episode. However, none of these patients had cardiovascular disease recorded as a contributing factor to their deaths. Conversely, in Woolf and Jacobs' case series, the majority of patients had notable drops in haemoglobin and ultimately died from either coronary thrombus, myocardial infarction, anaemia or directly from haemorrhage. Considering the high prevalence of cardiovascular disease in our cohort, and the high incidence of cardiovascular disease being recorded as an ultimate cause of death, particular attention should be paid to adequately managing conditions in epistaxis patients referred to

ENT on call services. Perhaps earlier reversal of antithrombotic agents or more aggressive transfusion policies should be considered and may help lower any associated mortality?

The WHO grading system of bleeding severity has limitations; the broad categories lack sensitivity and it is susceptible to variation in transfusion policies. Inevitably, the majority of epistaxis patients who are reviewed by ENT in the acute hospital setting will score 2 or more. To be classified as the highest category of '3' the patient must have received a blood transfusion. The average WHO haemorrhage scores did not vary significantly between the alive and deceased groups but a greater proportion of the deceased group were seen to have the highest WHO score of 3 (21.2% versus 3.9%). However, the proportion of patients with the lowest score of 1 was also higher in the deceased group, as the distribution of scores was generally wider (24.2% vs 10.4%, standard deviation 0.68 vs 0.37). Consequently, this study suggests that the WHO score is not a good predictor of patients at higher risk of death for epistaxis.

Interestingly, the haemostasis time was seen to be shorter in the deceased group than the alive group. There may be a number of explanations for this seemingly paradoxical finding. Anecdotally, shorter bleeding times are often seen to result from generalised nasal irritation or excoriation, rather than a defined bleeding point or identifiable fragile vessel. This type of more insidious epistaxis may be more commonly seen after medical interventions, such as administration of heparin products, and with certain patient factors, such as a bleeding diathesis or thrombocytopaenia. All these aspects were higher in the deceased subgroup. Additionally, the majority of the deceased subgroup were referred from a ward environment (54.5%). Consequently, there may have already been an increased tendency for their nasal mucosa to have been aggravated by the drying effects of non-humidified oxygen or from direct instrumentation by NG tube insertion or similar.

Limitations

The proportion of patients who died during the follow up period of the NEA2016 was small and so inevitably any deductive analysis will be limited. Regardless, it is felt that this small group is significant and worthy of further scrutiny. By utilising a prospective dataset, such as the NEA2016, we have hopefully minimised selection bias in identifying this unfortunate subgroup and so ensuring the real-world applicability of our conclusions.

Another limitation was the relatively short follow up period of 30-day utilised in the NEA2016. Ideally there would have been a longer observation period with more data available about time and cause of death. However, as this present study represents a retrospective analysis of a prospectively obtained dataset, obtaining further follow up data was unfortunately not possible. It could also be argued that any mortality directly related to epistaxis would have occurred in this 30-day window and so a longer period would have been superfluous.

CONCLUSIONS

The recent national audit of epistaxis management identified a higher than expected 30-day all-cause mortality rate. Those that died were more likely to be referred from a ward environment, have cardiovascular related diseases, diabetes and bleeding diatheses. Patients were not seen to die from exsanguination, rather, it appears that the epistaxis may be seen as a marker of general poor health and a poor prognostic sign in otherwise already morbid patients.

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The authorship committee comprised:	J. Hardman, M. E. Smith, M. Ellis, R. Williams, C. Hopkins.
The study steering committee comprised:	M. Ellis, A. Hall, J. Hardman, N. Mehta, M. E. Smith, R. J. Williams (chair).
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Statistical support was provided by:	J. Chynoweth, B. Jones, K Stevens.

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REFERENCES

1. NHS Digital. NHS Hospital Episode Statistics in England and Wales [Internet]. 2017 [cited 2017 Dec 31];Available from: http://content.digital.nhs.uk/hes
2. Lammens F, Lemkens N, Laureyns G, Lemmens W, Van Camp L, Lemkens P. Epidemiology of ENT emergencies. B-ENT 2014;10(2):87–92.
3. INTEGRATE (The National ENT Trainee Research Network). Epistaxis 2016: national audit of management. J Laryngol Otol 2017;131(12):1131–41.
4. Royal College of Physicians. The National Hip Fracture Database [Internet]. 2017 [cited 2017 Dec 31];Available from: https://www.nhfd.co.uk/
5. Office for National Statistics. Mortality rates (qx), principal projection, United Kingdom [Internet]. 2016 [cited 2018 Jan 1];Available from: https://www.ons.gov.uk/file?uri=/peoplepopulationandcommunity/birthsdeathsandmarriages/lifeexpectancies/datasets/mortalityratesqxprincipalprojectionunitedkingdom/2016based/wukprincipal16qx.xls
6. INTEGRATE (The National ENT Trainee Research Network). The British Rhinological Society multidisciplinary consensus recommendations on the hospital management of epistaxis. J Laryngol Otol 2017;131(12):1142–56.
7. Iqbal IZ, Jones GH, Dawe N, Mamais C, Smith ME, Williams RJ, et al. Intranasal packs and haemostatic agents for the management of adult epistaxis: systematic review. J Laryngol Otol 2017;131(12):1065–92.
8. Khan M, Conroy K, Ubayasiri K, Constable J, Smith ME, Williams RJ, et al. Initial assessment in the management of adult epistaxis: systematic review. J Laryngol Otol 2017;131(12):1035–55.
9. Mcleod RWJ, Price A, Williams RJ, Smith ME, Smith M, Owens D. Intranasal cautery for the management of adult epistaxis: systematic review. J Laryngol Otol 2017;131(12):1056–64.
10. Swords C, Patel A, Smith ME, Williams RJ, Kuhn I, Hopkins C. Surgical and interventional radiological management of adult epistaxis: systematic review. J Laryngol Otol 2017;131(12):1108–30.
11. Williams A, Biffen A, Pilkington N, Arrick L, Williams RJ, Smith ME, et al. Haematological factors in the management of adult epistaxis: systematic review. J Laryngol Otol 2017;131(12):1093–107.
12. Khan MN, Blake DM, Vazquez A, Setzen M, Baredes S, Eloy JA. Epistaxis: the factors involved in determining medicolegal liability. Int Forum Allergy Rhinol 2014;4(1):76–81.
13. Woolf CI, Jacobs A. Epistaxis Mortality: The Place of Blood Transfusion in its Prevention. J Laryngol Otol 1961;75(2):114–22.
14. American College of Surgeons, Committee on Trauma. Advanced trauma life support for doctors: ATLS student course manual. Chicago, IL: American College of Surgeons; 2008.
15. Centers for Disease Control. Work Table I. Deaths from each cause by 5-year age groups, race and sex: United States [Internet]. 2001 [cited 2017 Dec 31];Available from: https://www.cdc.gov/nchs/data/statab/vs00199wktbli.pdf

Figure 1: MEWS score at presentation

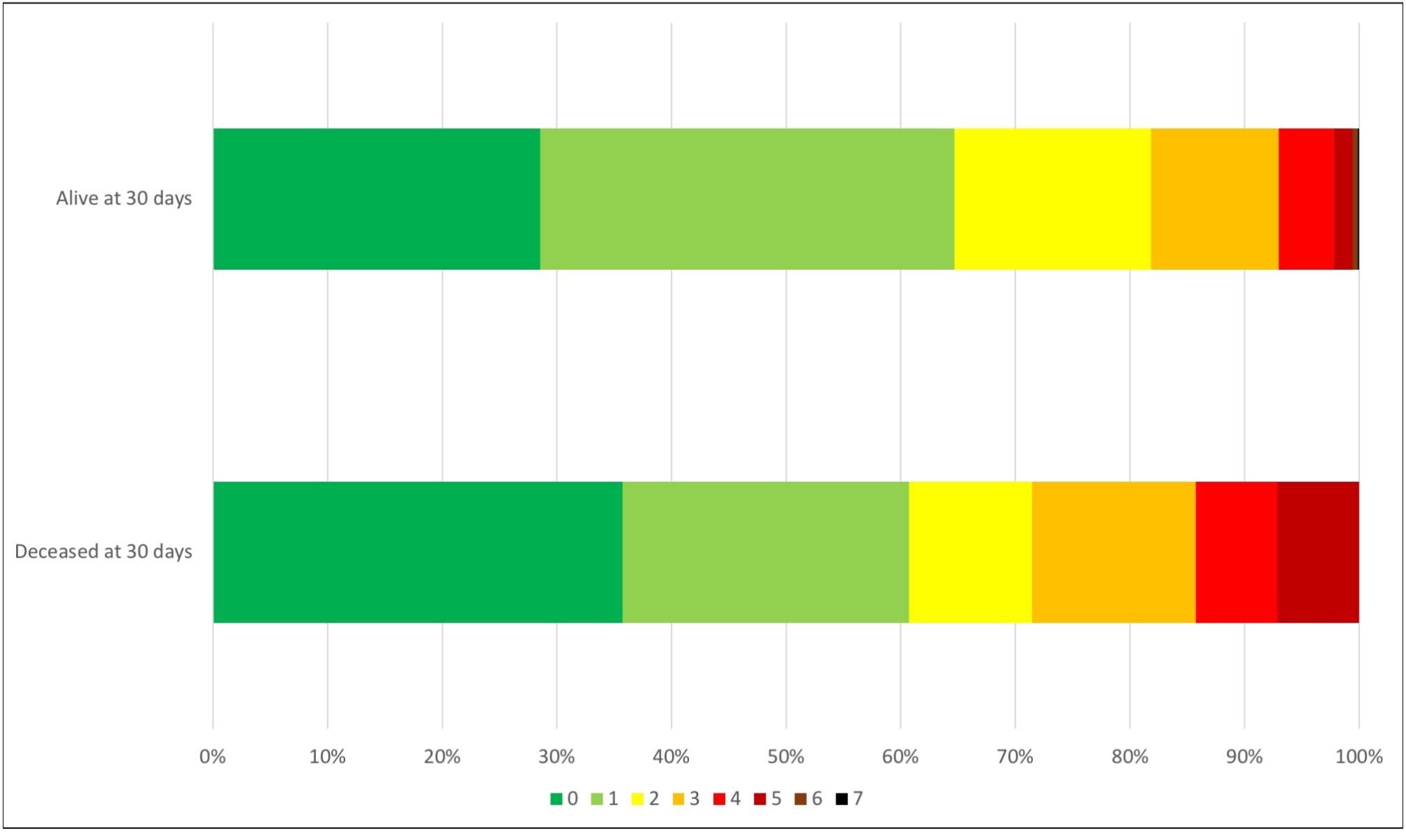

Figure 2: WHO bleeding score at presentation