Clinical Medicine 2019 Vol 19, No 1: 86–7

Greater illness severity characterises steroid diabetes following acute hospitalisation

Over 10% of hospital inpatients receive treatment with glucocorticoids[1] and yet the incidence of steroid diabetes (SD), and therefore monitoring requirements, in these patients is unknown. Acute illness can also induce hyperglycaemia through neuroendocrine and inflammatory responses,[2] which may be exacerbated by use of glucocorticoids.

We determined the incidence of SD in acute medical admissions and whether SD corresponds to illness severity rather than to the dose of glucocorticoid administered.

This was a retrospective case-note review of inpatients who received treatment with glucocorticoids (dose ≥10 mg prednisolone once daily, or equivalent)[3] for ≥24 hours in an acute medical admissions unit. We excluded patients using glucocorticoid therapy immediately prior to hospitalisation (other than inhaler or topical therapy); those with accident & emergency (A&E) department triage glucose ≥11.1 mmol/L, length of stay <24 hours, and diabetes mellitus (any of medical history, glycated haemoglobin ≥6.5% before or up to 3 months after admission, any glucose lowering therapy). A consecutive series was sought until n=100 in the study cohort.

New hyperglycaemia (capillary glucose ≥11 mmol/L after initiation of glucocorticoid therapy) was considered SD.[2,4] Glucose values were evaluated for up to 7 days after admission. The National Early Warning Score (NEWS) was used to determine illness severity. Glucocorticoids were converted to equivalent potencies to allow dose comparison; 1 steroid unit ≡ 5 mg prednisolone.[3] Data are mean (standard deviation) unless described. Local Research & Innovation approval was sought and we were advised that ethical approval was not required.

Between July 2015 and April 2017, 498 patients with an acute medical admission received glucocorticoids of ≥10 mg prednisolone per day (or equivalent) for at least 24 hours. We excluded n=268 without glucose monitoring; a further n=130 with pre-existing diabetes were also excluded, leaving 100 patients for analysis. Exacerbation of pulmonary disease constituted the reason for admission for over half the cohort. The incidence of SD was 14%. Median duration of admission before SD was identified was 2.5 days (interquartile range [IQR] 1–4). Patients with SD had higher NEWS at admission and higher peak NEWS during their hospitalisation (Table 1) but no difference in type of steroid, nor mean steroid dose. Glucose concentration from the A&E department did not predict likelihood of SD (Table 1).

Table 1. Characteristics of steroid diabetes and normal glucose group

Parameter		Steroid diabetes n=14	Normal glucose n=86	*p* value
Age, years[#]		64.9 (16.0)	61.3 (18.0)	0.498
Gender	Male	6 (43%)	40 (47%)	
	Female	8 (57%)	46 (53%)	0.799
Type of steroid	Prednisolone	11 (79%)	51 (59%)	
	Hydrocortisone	1 (7%)	26 (30%)	0.257
	Dexamethasone	1 (7%)	7 (8%)	
	Methylprednisolone	1 (7%)	2 (2%)	
Relative daily steroid dose, steroid unit*		6.0 (6.0–10.9)	7.3 (6.0–8.7)	0.303
Admission NEWS*		6 (3–9)	4 (2–6)	0.041
Maximum NEWS*		8 (5–9)	6 (3–8)	0.048
A&E capillary glucose, mmol/L*		6.1 (5.3–10.0)	6.0 (5.3–7.0)	0.195
Length of stay, days*		7.0 (4.8–16.5)	5.0 (3.0–8.0)	0.113
Inpatient mortality		2 (14%)	5 (6%)	0.249

[#] = mean (standard deviation); * = median (interquartile range); NEWS = National Early Warning Score

Patients developing SD following acute hospitalisation had greater illness severity than those maintaining normoglycaemia, whereas the steroid dose (equivalent to 35 mg prednisolone daily) was no different. The incidence of SD that we found (14%) is similar to Umpierrez *et al*[5] who reported hyperglycaemia in 12% of a general medical inpatient population (with no history of diabetes), although in that series the use of glucocorticoids was not reported. We hypothesise that exogenous glucocorticoid therapy contributes little to the pathogenesis of hyperglycaemia with acute hospitalisation. Use of resource intensive glucose monitoring may be better directed to those of greater illness severity than to all hospitalised patients treated with glucocorticoids. This requires evaluation. ■

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