DETECTING HYPERGLYCAEMIA IN DEXAMETHASONE-EXPOSED CANCER PATIENTS: AN EXPLORATORY STUDY

Gane, S.E. MSc, PhD, PGCE, RN[1]; Forbes, A. Prof MSc, PhD, RN[1]; Armes, Jo. Prof PhD, RN[2]

[1] *Kings College London, London, UK*
[2] *University of Surrey, Guildford, Surrey, UK*

Background
Dexamethasone is widely used during chemotherapy to reduce chemotherapy-induced nausea and vomiting, hypersensitivity reactions and delayed toxicity. Hyperglycaemia is a recognised adverse effect of glucocorticoid therapy and may result in steroid-induced diabetes (SID), which is associated with infection, chemotherapy toxicity, poorer outcomes and hospitalisation. Despite these risks, evidence-based guidance on screening for SID during cancer treatment remains limited.

Aim
To explore the utility and feasibility of screening methods and timings for detecting SID in dexamethasone-exposed chemotherapy patients.

Methods
This exploratory observational study recruited adults with breast, colorectal or prostate cancer receiving chemotherapy incorporating high-dose dexamethasone. Participants were observed before, during and after dexamethasone exposure over three chemotherapy cycles. Continuous glucose monitoring (CGM) was used to characterise glycaemic patterns and compare HbA1c, fructosamine, 1,5-anhydroglucitol (1,5-AG), RPG, CBG, urine glucose testing and symptom reporting. SID requiring treatment was identified using the King's College Hospital SID protocol.

Results
Fifteen participants were recruited before COVID-19-related study suspension. Six participants demonstrated higher glucose exposure (HGE), of whom three required diabetes treatment. Five further participants demonstrated higher fasting-only glucose exposure (HFGE). CBG monitoring emerged as the most practical screening method for identifying hyperglycaemia and treatment-requiring SID. HbA1c exceeded the diagnostic threshold in five of six HGE cases, although three results were potentially unreliable. Other screening methods showed inconsistent performance. A ≥10% reduction in 1,5-AG was observed in four HGE cases. Optimal CBG screening times were before breakfast and before the evening meal or bedtime. While glucose variability peaked approximately 24 hours following dexamethasone exposure, participants requiring treatment demonstrated delayed glycaemic recovery, with persistent glucose elevations on Days 4 and 5. HGE was more common in participants aged >60 years and those with impaired glucose tolerance or hypertension.

Conclusions

Twice-daily CBG monitoring following dexamethasone administration may provide a practical approach for detecting SID during chemotherapy. Delayed glycaemic recovery on Days 4 and 5, rather than peak glucose excursions alone, was associated with treatment-requiring SID and may help identify individuals requiring intervention. These findings support evaluation of risk-stratified SID screening approaches in larger oncology populations.