standard deviations of the scores of the first follow-up assessment of participants without dementia for the first three follow-ups (n = 573). The resulting composite score was validated against the CDR score by calculating the correlation between the composite score and CDR of the samples of the first (total n = 1,099, with dementia n = 235) and second (total n = 683, with dementia n = 190) follow-ups and by calculating the correlation of the changes in both scores between the first and second follow-up. Participants without demenita were assigned a CDR score of 0.

The Pearson correlation coefficient between the composite score and CDR was 0.88 at the first follow-up, 0.83 at the second follow-up, and 0.62 for in scores between the first and second follow-ups. All correlation coefficients were significant at $P<.001$. The composite score interquartile range (IQR) was (-0.66–0.32) for participants with a CDR of 0, (-2.58 to -0.90) for a CDR of 1, (-5.50 to -2.56) for a CDR of 2, and (-9.61 to -5.23) for a CDR of 3.

The results indicate high correlations between the Composite Cognitive and ADL Functioning Score and the CDR score at one point in time. Changes over time in the composite score also correlated highly with change in CDR. These observations support the validity of the composite score that was developed. The fact that the IQRs of the composite scores per CDR score overlapped little shows that the composite score could accurately separate CDR categories. This supports the clinical relevance and provides information for the clinical interpretation of the Composite Cognitive and ADL Functioning Score. Based on the validation by CDR scores in this study population, the Composite Cognitive and ADL Functioning Score is easy to use and interpret and clinically relevant, and because it reduces the need for clinical judgment of each individual required in comparable composite measures, it is highly useful for large population-based studies.

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AGE AND ACUTE-SEVERITY ILLNESS PORTEND DAILY ACTIVITY DYSFUNCTION 6 MONTHS AFTER HOSPITAL DISCHARGE

To the Editor: Intensive care unit (ICU) patients can experience significant morbidity, mainly after prolonged hospitalization.[1] Nevertheless, there is no evidence of evaluation of functional capacity after hospital discharge, although it can theoretically be severely affected. The goal of the current study was to evaluate performance in activities of daily living (ADLs) of critically ill patients according to the Barthel index 6 months after hospital discharge.

All consecutive patients admitted to the ICU from September to December 2008 were eligible. Only patients with an ICU stay longer than 3 days were included, to avoid admissions only for hemodynamic or respiratory monitoring. Demographic data were collected, along with the evaluation of acute severity illness according to the Simplified Acute Physiology Score (SAPS) II and Sequential Organ Failure Assessment (SOFA) scores and comorbidities (Charlson index). The Barthel index was calculated after interviews with two physiotherapists soon after ICU admission, on the day of hospital discharge, and by telephone interview 6 months after hospital discharge. Briefly, the Barthel index measures ability to totally or partially perform ADLs (feeding, grooming, toilet use, dressing, bathing, fecal and urinary continence, transfer from bed to a chair, walking a short distance, and climbing stairs),[2] and ranges from 0 to 100 points. Numerical data were presented as medians and interquartile ranges (IQRs; 25–75%). A

Table 1. Functional Dependence and Barthel Index at Hospital Admission, Discharge, and 6 Months After Discharge of 86 Critically Ill Patients with an Intensive Care Unit Stay of Longer than 3 Days

Activity	Dependence at Admission	Dependence at Discharge	Dependence 6 Months After Discharge
Feeding, %	7	8	10
Bathing, %	13	15	16
Self-care, %	12	10	15
Dressing, %	21	21	20
Urinary continence, %	6	6	14
Fecal continence, %	7	12	12
Bathroom use, %	21	26	20
Bed-to-chair transfer, %	30	19	14*
Short walk, %	19	19	13
Climbing stairs, %	51	44	26*
Barthel index, points (interquartile range)	87.5 (75–100)	100 (71–100)	100 (86–100)*

*$P<.05$—chi-square test for each evaluated activity and Mann-Whitney test for Barthel index.

multivariate analysis was performed to find factors associated with degree of functional inability after 6 months.

One hundred sixteen patients stayed in the ICU for longer than 3 days. There were 26 (22%) deaths within 6 months of hospital discharge. Eighty six patients were discharged who had a median age of 68 (range 57–77). There were more women (57%); 21 (24%) were admitted for medical reasons; median SAPS II score was 23.5 (IQR 18–30) points; the median SOFA score on Day 1 of ICU admission was 1 (IQR 0–2) point and the median hospital length of stay was 9 (IQR 7–14) days. The main causes of admission were cardiovascular (n = 27), gastrointestinal (n = 22), respiratory insufficiency (n = 13), and neurological decompensation (n = 11). The Barthel index was 90 (IQR 75–100) points on ICU admission; 100 (IQR 71–100) points on the day of hospital discharge; and 100 (IQR 86–100) points after 6 months.

There was some degree of functional incapacity (Barthel index <100 points) in 50 (58%) patients on ICU admission, in 42 (49%) on the day of hospital discharge, and in 28 (33%) after 6 months. Specific incapacities and the Barthel index at hospital admission and discharge and after 6 months are shown in Table 1. The widest variations in all of the evaluated activities were for motor function, namely bed-to-chair transfer and climbing stairs. Age (median 76 (IQR 68–81) vs 62 (IQR 53–74), $P<.001$) and SAPS II score (31 (IQR 24–39) vs 21 (IQR 17–28) points, $P<.001$) were significantly higher for patients with a lower Barthel index. Age (odds ratio (OR) = 1.05, 95% confidence interval (CI) = 1.01–1.10; $P=.01$) and SAPS II score (OR = 1.06, 95% CI = 1.01–1.11; $P=.02$) were independent associated factors for a low Barthel index 6 months after discharge.

There is limited information available on the functional capacity of discharged critically ill patients The Barthel index was evaluated in clusters of ICU patients—older patients and those with neurological (stroke) and orthopedic conditions.[3–5] Age and severity of acute illness were independent predictors of quality of life of ICU survivors 6 months after hospital discharge, as shown according to the Medical Outcomes Study 36-item Short Form Questionnaire for evaluation of ADLs;[6] but the authors did not retrieve information on functional capacity from patient interviews. Although the prevalence of some degree of incapacity is high at the moment of hospital discharge, the Barthel index tends to normalize after some months, especially if the main disease is reversible. The results of the current findings for the Barthel index are similar to those of other studies with patients discharged after critical illness.[7–9] A significant proportion of patients do not fully recover functional capacity 6 months after hospitalization. The current study contributes data regarding the postdischarge period, during which many critically ill patients continue to need functional assistance. It is expected that initiatives for long-term care of critically ill patients, such as early exercise training for critically ill survivors, which can enhance recovery of functional exercise capacity at hospital discharge, will be added.[10] Regular visits by physical therapists can reduce the patients' time to return to normal daily activities.

In conclusion, 30% of critically ill patients, with an ICU stay longer than 3 days, present significant dysfunction in ADLs 6 months after hospital discharge. Older age and greater acute severity of illness scores can predict which patients will have higher degrees of dependence after hospital discharge.

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COMMENTS/RESPONSES

HEPARIN-INDUCED THROMBOCYTOPENIA: AN INCREASINGLY COMMON CAUSE OF BILATERAL ADRENAL HEMORRHAGE

To the Editor: Barrou and colleagues suitably highlighted the entity of bilateral adrenal hemorrhage (BAH) and subsequent adrenal insufficiency as a consequence of knee arthroplasty.[1] We write to highlight an important mechanism of BAH that was not mentioned in this report: heparin-induced thrombocytopenia (HIT). HIT is an increasingly well-documented cause of BAH, and Findling and colleagues first formally described this association in 1987.[2] We have recently reviewed the available literature and found 17 reported cases of BAH in the setting of HIT in addition to a case that we are reporting from our own institution.[3]

HIT is a common, well-documented adverse effect of treatment with heparin resulting in paradoxical arterial and venous thromboses. HIT occurs in up to 1% of patients receiving unfractionated heparin for postoperative antithrombotic prophylaxis, more frequently in orthopedic patients (4.8%), such as the patient described.[4,5] The process is antibody mediated and results in activation, consumption, and thrombocytopenia in the clinical syndrome of HIT.[4] In contradistinction to other drug-induced thrombocytopenias, HIT leads to acquired hypercoagulability rather than hemorrhage. These prothrombotic complications of HIT necessitate immediate discontinuation of heparin and initiation of a thrombin inhibitor to prevent vascular thromboses.[5,6] In the setting of HIT, the adrenal gland has a vascular construction that lends itself to venous thrombosis, subsequent arterial hemorrhage, and resulting adrenal insufficiency.[4]

BAH is a serious condition because the resulting adrenal insufficiency leads to life-threatening hemodynamic collapse if unrecognized.[7] This diagnosis must be considered and treated appropriately because the complications related to the prothrombotic state are severe. Shock associated with HIT must raise the suspicion of adrenal insufficiency and BAH. This insidious disorder, if left untreated, is fatal.[8–10]

In the presented case, was there any abnormality in the patient's platelet count or any indication for an evaluation of HIT? It is likely that the patient described had prior exposure to heparin with a previous myocardial infarction and coronary stent placement. He then subsequently received heparin (Enoxaparin) postoperatively after total knee arthroplasty (a high-risk procedure associated with heparin). His symptoms began on postoperative Day 6, consistent with the timing of HIT, occurring 5 to 10 days following heparin initiation.[5] In light of the acquired hypercoagulability syndrome of HIT secondary to platelet activation and thrombin generation, we strongly encourage an evaluation and examination for HIT. The BAH is probably secondary to adrenal vein thrombosis and should also prompt this hypercoagulable examination.

Isolated HIT is well documented and widely recognized as a complication of heparin use, but the secondary complication of adrenal vein thrombosis leading to BAH remains insufficiently recognized and undertreated. We agree with the authors regarding the nonspecific presentation of adrenal insufficiency after hemorrhage. As a complication of HIT, BAH coupled with the catastrophic clinical course of untreated adrenal collapse requires a high index of suspicion to achieve rapid diagnosis, prevent further arterial and venous thrombosis, and allow the provision of life-saving therapy.