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Impact of Implementation of the High-Sensitivity Cardiac Troponin T Assay in a University Hospital Setting

To the Editor:

The performance of the high-sensitivity cardiac troponin T assay (hs-cTnT)[1] has been evaluated in a multicenter study *(1)*. Effective July 2009, we replaced the fourth-generation troponin T assay (cTnT) with the hs-cTnT assay in clinical practice. This study audits the impact of this implementation.

The hs-cTnT, implemented on the cobas e 411 platform (Roche Diagnostics), fully replaced the cTnT performed on the Elecsys 2010 analyzer [cutoff, 30 pg/mL—based on actual assay performance (10% CV concentration)]. We obtained a detection limit of 5 pg/mL, a 99th percentile of 15 pg/mL, limited comparability with the cTnT at concentrations <100 pg/mL (on average, a 30-pg/mL cTnT concentration yielded a value of approximately 65 pg/mL with the hs-cTnT) and mean CVs of 9.1% for the cTnT (at 39 pg/mL) and 8.5% for the hs-cTnT (at 17 pg/mL). We retrieved hs-cTnT results for the first 3 months after implementation (July 16 to October 15, 2009) and cTnT results for the same period 1 year previously. Results were dichotomized as positive or negative with respect to cutoffs. Among troponin-positive patients with at least 2 results during their examination, we divided marker-release curves on the basis of typical or atypical kinetics. We defined as "typical" an increasing or decreasing pattern showing a troponin change between 2 consecutive samples exceeding +46% for increasing troponin results and −32% for decreasing results. Otherwise, the troponin pattern was considered "atypical." For definition of these percentage changes, we referred to the short-term biological variation for troponin I *(2)*. We are aware, however, that the 2 cardiac troponins may have different biological kinetics in blood, so their biological variation may be different.

In the evaluated period, 2287 hs-cTnT tests were performed during 1371 examinations of 1137 patients. Correspondingly, 2170 cTnT tests were performed during 1409 examinations of 1205 patients. After hs-cTnT implementation, a 5.4% increase in the hospital-wide test volume was recorded, despite a slight decrease in the number of admitted patients and examinations. The mean (SD) number of troponin tests per examination was 1.54 (1.0) before and 1.67 (1.1) after hs-cTnT implementation ($P < 0.0001$), with a single test ordered in 67.5% and 60.2% of examinations, respectively. The distribution of troponin orders and positive-test rates in different wards is shown in Table 1. A positive result was found in 31.7% of cTnT tests and in 58.7% of hs-cTnT tests (relative difference, +85%), corresponding to 25.3% and 51.6% positive examinations, respectively ($P < 0.0001$). Of all the hs-cTnT positive results, 64% fell in the 16–65 pg/mL interval, previously negative with the cTnT. In the emergency department after hs-cTnT implementation, the number of hospitalized patients with positive troponin results increased from 158 to 292 (+85%), but the rate of admission in intensive care and non–intensive care departments was unchanged ($P = 0.108$). In the same periods, 16 cTnT-positive patients (8.5%) and 109 hs-cTnT–positive patients (26.6%) were discharged. Of these discharged patients, 1 cTnT-positive patient and 13 hs-cTnT–positive patients were readmitted to the emergency department in the subsequent 2 months ($P = 0.804$, between the 2 assays).

We audited 458 cTnT and 546 hs-cTnT curves, of which 39.1% and 69.0%, respectively, had at least 1 positive result ($P < 0.0001$). The difference in the percentage of positive curves displaying a typical marker release was not significant (17.2% for the cTnT vs 20.5% for the hs-cTnT, $P = 0.32$). A higher absolute number of typical positive curves was observed after hs-cTnT implementation (from 79 to 112). This increased ability to detect events involving acute marker release was fully explained by the number of typically positive curves in which the hs-cTnT result never exceeded 65 pg/mL (n = 38).

The replacement of the cTnT with the hs-cTnT markedly increased the rate of positive tests. A similar outcome was previously described for a contemporary sensitive troponin I assay *(3)*. What is unique in our experience is the magnitude of the increase in positive results after hs-cTnT introduction, which was based on imple-

[1] Nonstandard abbreviations: hs-cTnT, high-sensitivity cardiac troponin T assay; cTnT, fourth-generation troponin T assay.

Table 1. Distribution of cardiac troponin orders and positive test rates in different hospital wards.

	Orders, n (% of total)			Positive tests, n (% of total ordered tests)			
	cTnT	hs-cTnT	hs-cTnT/cTnT difference	cTnT	hs-cTnT	hs-cTnT/cTnT difference	*P*
Emergency department	1472 (67.8%)	1465 (64.1%)	0%	272 (18.5%)	664 (45.3%)	145%	<0.0001
Internal medicine	184 (8.5%)	293 (12.8%)	59%	121 (65.8%)	263 (89.8%)	117%	
Intensive care unit	133 (6.1%)	104 (4.5%)	−22%	92 (69.2%)	88 (84.6%)	−4%	
Cardiology	99 (4.6%)	105 (4.6%)	6%	45 (45.5%)	78 (74.3%)	73%	
Pneumology	72 (3.3%)	45 (2.0%)	−38%	55 (76.4%)	41 (91.1%)	−25%	
Surgery	50 (2.3%)	40 (1.7%)	−20%	19 (38.0%)	29 (72.5%)	53%	
Others[a]	44 (2.0%)	64 (2.8%)	45%	13 (29.5%)	33 (51.6%)	200%	
Infectious disease	42 (1.9%)	70 (3.1%)	67%	22 (52.4%)	56 (80.0%)	155%	
Neurology	27 (1.2%)	19 (0.8%)	−30%	15 (55.6%)	19 (100.0%)	27%	
Orthopedics	23 (1.1%)	36 (1.6%)	57%	14 (60.9%)	27 (75.0%)	93%	
Nephrology	13 (0.6%)	45 (2.0%)	246%	12 (92.3%)	43 (95.6%)	207%	
Oncology	11 (0.5%)	1 (0.04%)	−91%	7 (63.6%)	1 (100.0%)	−86%	
All clinical wards	698 (32.2%)	822 (35.9%)	18%	415 (59.5%)	678 (82.5%)	63%	<0.0001
Total	2170 (100.0%)	2287 (100.0%)	5.4%	687 (31.7%)	1342 (58.7%)	95%	<0.0001

[a] All wards accounting for <10 tests were gathered into this group.

menting this assay in a routine protocol. The number of examinations with positive results increased from approximately 25% to >50%. Although the hs-cTnT could appear confounding as a test less specific for the diagnosis of myocardial infarction *(4)*, we were unable to demonstrate differences in the percentage of curves with a typical marker release when we compared the cTnT and the hs-cTnT. An interpretative approach based on the demonstration of a pathophysiology-defined release of troponin in the blood may allow the same specificity performance to be achieved when using different generations of troponin T assays, thus supporting the use of serial testing for clinical decisions *(5)*.

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