Active communication between laboratory physicians and clinicians: Need of the hour to improve patient outcomes

Sir,

Quality laboratory support has become an essential component for appropriate clinical diagnosis and management. Role of active communication between laboratory physicians and clinicians in this regard is often ignored. Here, we present two instances from a tertiary care teaching hospital which highlights the importance of active communication between the laboratory physicians and clinicians.

Case 1 – The routine biochemistry laboratory received an otherwise unremarkable, bar-coded blood sample for investigations from inpatient department. During validation of reports, the serum phosphate (S. Phos) level was observed to be 0.4 mg/dL which was way beyond the physiologically observable range for a 42-year-old male patient. The critical levels for S. Phos in the laboratory being <1.5 mg/dL, delta check was done as per protocol. He had a normal phosphate level (3.5 mg/dL) 1 day before. Since no accompanying clinical history was mentioned in the test requisition form and without any aberration in the quality control procedures for the day, this case of isolated hypophosphatemia was assumed to be a random error. As per protocol of our laboratory, all potential random errors are subjected to repeat testing before release of results as random errors are quite common in our setting. After confirmation, immediate communication was initiated by resident doctors with intimation to faculty-in-charge. However, as the whole process took about 2 h, it turned out to be too late, and the patient had already expired.

It was later found from the patient’s records that he was a chronic alcoholic admitted with a provisional diagnosis of pneumonia. He was being treated with intravenous fluids and empirical antibiotics as per protocol. The severe hypophosphatemia of 0.4 mg/dL was an incidental finding and was probably not anticipated by the resident clinical doctors. Since no suggestive clinical conditions were mentioned, the laboratory spent an extra 2 h in confirming and verifying the report rather than communicating it straightway. Had the brief clinical history of chronic alcoholism been mentioned, the laboratory physicians could have anticipated the isolated hypophosphatemia to be due to a condition called “Refeeding Syndrome (RFS)” which happens during active management of chronic alcoholics and thus could have released the life-threatening report at once and cautioned the treating clinicians.

RFS manifests as a severe electrolyte imbalance (principally low-serum levels of intracellular ions such as phosphate, magnesium, and potassium) and metabolic abnormalities in undernourished patients undergoing rapid replenishment enterally or parenterally. Chronic alcoholism, drug abuse, chronic infection (e.g., HIV), dysphagia and esophageal dysmotility, prolonged fasting (e.g., individuals on hunger strikes), bariatric surgery, low-birth-weight, and premature birth are some known risk factors.[1,2] Many of the reports of RFS in the literature may be more appropriately referred to as “refeeding hypophosphatemia” because these patients do not always display all clinical features of RFS and present as isolated hypophosphatemia.[3]

Case 2 – Consistently high hematocrit (72%–78%), prolonged prothrombin time (PT) (between 20.8s and 25.1s), activated partial thromboplastin time (APTT) (between 56.0s and 59.3s), and international normalized ratio (INR) (between 1.87 and 2.1) were observed consistently for 15 days in a 15-year-old patient with congenital heart disease. The deranged coagulation profile was assumed to be due to altered ratio of sodium citrate and plasma in the samples due to secondary polycythemia and was ignored. However, we failed to take note that initially his PT, APTT, and INR were high with normal platelet counts and D dimer levels. Later on, his platelets decreased, D-dimer got raised and PT, APTT, and INR were further raised, pointing toward disseminated intravascular coagulation (DIC) [Table 1]. This suspicion of DIC could have been verified by asking for samples in citrate vial appropriately titrating for hematocrit had there been good communication between laboratory physicians and clinicians.

The most commonly used anticoagulant for coagulation studies is trisodium citrate. A 32 g/L (0.109 M) solution is recommended in a ratio 9:1 (nine parts of the blood sample to one part of anticoagulant). In blood samples with increased hematocrit, the citrate concentration increases in the plasma within the collection tube and it remains in excess after binding the free-ionized calcium of blood.[4] When we add platelet-poor plasma to the PT and APTT reagents, the residual excess citrate binds a significant amount of calcium that is added to the clotting test reaction.[4] This causes an artefactual increase in PT and APTT. Since 1980, the Clinical and Laboratory Standards Institute has recommended a correction for

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Letter to the Editor

Proper communication between laboratory physicians and clinicians could have resolved the issue with a favorable outcome. Besides, proactive engagement, in this case, could have been to identify coagulation tests with high hematocrit and mandate that testing be done in the correct tube regardless of the initial or subsequent test results.

In both the above-mentioned cases, the outcome of the patients could have been better if laboratory personnel and clinicians communicated well mutually. Similar reports of such lapses are grossly under-reported in the literature due to obvious reasons. However, instances like these stress the need of healthy communication between laboratory physicians and treating physicians. It is time we recognize the need for it and try to bridge the gap. The concept of a diagnosis management team may work wonders in this regard.

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Table 1: Day-wise summary of hematology and coagulation study reports

|               | Day 1 | Day 5 | Day 9 | Day 18 | Day 19 | Day 22 | Day 24 |
|---------------|-------|-------|-------|--------|--------|--------|--------|
| TLC (/mm³)    | 6500  | 4600  | 4000  | Not done | Not done | 6600  | 8700  |
| DLC (%) (N/L/M/E/B) | 73/21/7/0/0 | 90/6/4/0/0 | 87/7/6/0/0 | Not done | Not done | 80/20/0/0/0 | 84/9/6/1/0 |
| RBC count (x10⁹/mm³) | 7.75  | 7.36  | 7.76  | 7.56   | Not done | 7.77  | 7.49  |
| Hb (g/dL)     | 23.0  | 22.1  | 23.0  | 22.4   | Not done | 23.3  | 22.7  |
| HCT (%)       | 76.3  | 72.1  | 75.7  | 73.7   | Not done | 78.3  | 74.4  |
| Platelet count (/mm³) | 41,000 | 80,000 | 54,000 | 14,000  | Not done | 16,000 | 20,000 |
| PT (s) (patient/control) | 20.8  | 21.0/13.0 | 23.4  | 36.5   | 25.0/13.0 | Not done | 50.5  |
| INR           | 1.87  | 1.62  | 2.10  | 3.36   | Not done | Not done | 4.45  |
| APTT (s) (patient/control) | 59.3  | 59.0/30.0 | Not done | Not done | 56.0/30.0 | Not done | 90.7  |
| TT (s) (patient/control) | Not done | 23.0/16.0 | Not done | Not done | 24.0/16.0 | Not done | Not done |
| Fibrinogen (mg/dL) (reference range 200-450 mg/dL) | Not done | 190 | Not done | 130 | Not done | Not done | Not done |
| D-dimer (mg/L) | 2.11  | >0.5   | Not done | Not done | >0.5    | Not done | Not done |

INR=International normalized ratio, APTT=Activated partial thromboplastin time, TT=Thrombin time, HCT=Hematocrit, RBC=Red blood cells, DLC=Differential Leukocyte count, TLC=Total leukocyte count, Hb=Hemoglobin, PT=Prothrombin time

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