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Performance comparisons of three rapid screening methods for the G6PD deficiency test in newborns

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ABSTRACT

Background: Glucose-6-phosphate dehydrogenase (G6PD) deficiency is an X-linked recessive disorder that affects over 400 million people worldwide. The deficit causes individuals susceptible to hemolysis during oxidative stress. In newborns, G6PD deficiency can lead to hyperbilirubinemia, bilirubin-induced neurologic dysfunction, and kernicterus, making early detection and screening crucial.

Objectives: This study aimed to compare the diagnostic performance of three rapid screening tests for G6PD deficiency in newborns: the fluorescent spot test (FST), G6PD rapid test kit, and SD Biosensor, using spectrophotometry as the gold standard.

Materials and methods: Blood samples from 70 newborns were tested using these three methods. The diagnostic performances, including sensitivity, specificity, positive predictive value (PPV), negative predictive value (NPV), and efficiency of each method were analyzed.

Results: Both the FST and G6PD rapid test kit exhibited higher specificity, PPV, and efficiency compared to the SD Biosensor. Nonetheless, the SD Biosensor exhibited superior sensitivity and NPV, but it was unable to identify G6PD activity in 16.4% of instances due to elevated hemoglobin concentrations.

Conclusion: The FST and G6PD rapid test kit are reliable and suitable for G6PD deficiency screening in newborns, especially in settings with limited resources, due to their high efficiency, specificity, and rapid results. The SD Biosensor remains a valuable tool in clinical contexts requiring high sensitivity. For newborns with high hemoglobin levels, the FST or G6PD rapid test is recommended for accurate screening. Further studies with larger sample sizes are necessary to confirm the reliability of these tests in diverse populations.

Introduction

Glucose-6-phosphate dehydrogenase (G6PD) deficiency is the most prevalent human enzyme deficit, exhibiting an X-linked recessive inheritance pattern, affecting over 400 million individuals globally, particularly among those of African, Asian, and Mediterranean ancestry.[1] The global prevalence of G6PD-deficient variants mirrors the geographical distribution of malaria, supporting the hypothesis that G6PD deficiency provides some level of protection against malaria.[2,3] In Thailand, several studies indicated

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a significant prevalence of G6PD deficiency, impacting roughly 16.9% of the population.[4-7] The previous reports revealed that the prevalence of G6PD deficiency among neonates at Rajavithi Hospital Bangkok, Thailand from May 1995 to July 1998 was 5.1%[7] and at King Memorial Chulalongkorn Hospital, Bangkok, Thailand from February to August 2021 was 6.5%.[8]

G6PD plays a crucial role in the first step of the hexose monophosphate pathway, also known as the pentose phosphate pathway (PPP). In this process, G6PD converts glucose-6-phosphate (G-6-P) into 6-phosphogluconolactone (6-PG), while also reducing nicotinamide adenine dinucleotide phosphate (NADP) to NADPH. In red blood cells, the PPP is the only source of NADPH, which is vital for protecting the cells from oxidative damage. This protection is mainly carried out by glutathione in its reduced form (GSH). The NADPH is essential for the regeneration of GSH by the enzyme glutathione reductase. Therefore, the primary function of NADPH in red blood cells is to maintain the reduced state of glutathione, which helps prevent oxidative damage to the cells. Individuals with deficiency are particularly susceptible to hemolytic events during infections or after exposure to fava beans and certain medications.[9] The G6PD deficiency in newborns presents an increased risk of hyperbilirubinemia, which may rapidly escalate and lead to bilirubin-induced neurologic dysfunction (BIND), potentially resulting in kernicterus, a permanent and devastating neurological damage. Therefore, screening for G6PD deficiency and close monitoring of affected infants are important.[10]

Bilirubin is primarily produced from the breakdown of hemoglobin in red blood cells. While small amounts of bilirubin serve as antioxidants, excessive accumulation can be cytotoxic, necessitating timely excretion from the body.[11] In neonates, bilirubin production is significantly higher than in adults due to the higher turnover and shorter lifespan of their erythrocytes.[12] However, their ability to eliminate bilirubin is less efficient than that of adults.[13] The combination of high bilirubin production and limited elimination capacity puts newborns at risk of developing neonatal hyperbilirubinemia, which if severe and untreated, can lead to kernicterus.[14] Fortunately, kernicterus is almost always preventable, but it requires timely and accurate detection of hyperbilirubinemia in newborns.[15] One of the most common causes of neonatal hyperbilirubinemia is G6PD deficiency.[16]

In hospital laboratories, the measurement of G6PD enzyme levels in blood for diagnosing G6PD deficiency can be performed using several methods. The gold standard for quantifying G6PD activity in red blood cells is the spectrophotometric assay, which measures G6PD activity by detecting the formation of NADPH, based on the difference in absorbance of the sample at 340 nm over time.[17,18] However, due to various limitations, such as budget constraints, many hospitals are unable to perform enzyme activity tests for G6PD deficiency using standard methods. In several hospitals, the fluorescent spot test (FST), which is endorsed by the International Committee of Standardization in Hematology (ICSH)[19], is still commonly used as a reliable screening method for G6PD deficiency. Additionally, many hospitals are increasingly adopting rapid test and biosensor devices for G6PD screening. In particular, the biosensor, which allows for quantitative measurement of G6PD enzyme levels, has gained prominence in screening practices. The SD Biosensor (STANDARD G6PD test, Suwon, Korea) is a new point-of-care device that provides a quantitative G6PD measurement, adjusted for hemoglobin levels, making it accessible for use in smaller clinics and laboratories.[20,21] Nevertheless, there is still limited research on the effectiveness of biosensor and rapid test methods. Further studies are needed to evaluate the accuracy and efficiency of these methods for G6PD deficiency screening, to establish their reliability as standard diagnostic tools. The aim of this study is to evaluate the efficacy of three rapid test kits for screening G6PD deficiency in neonates: FST, G6PD rapid test kit, and SD Biosensor, utilizing data from the spectrophotometry method as the reference.

Materials and methods

Blood samples

At Nakornping Hospital, 0.5 mL whole blood samples of newborns were collected in ethylenediaminetetraacetic acid (EDTA) anticoagulated tubes. The G6PD activity was measured using the G6PD kit (GPD0204, Mindray, Shenzhen, China) and an automated UV enzymatic analyzer (BS-360E, Mindray). The principle of the test kit is mainly based on light absorption. The NADP is reduced to NADPH by G6PD with the presence of its specific substrate, G-6-P. The enzyme activity can be determined by measuring the changes in absorbance rate at 340 nm due to the reduction of NADP. Based on the manufacturer's instructions, the blood samples which had G6PD enzymatic activity <3.8 U/gm Hb were diagnosed as G6PD deficiency. This technique was used as a reference method. The over-left blood samples were aliquoted into microcentrifuge tubes and sent to the Associated Medical Sciences-Clinical Service Center (AMS-CSC), Chiang Mai University, Chiang Mai, Thailand. The whole blood samples were stored in a refrigerator at 4 °C until used. The samples size was calculated using the Yamane's formula[22] as follow:

$$n = N / (1 + Ne^2)$$

n: represents the required sample size.

N: represents the total population size of neonates requiring G6PD activity assessment at Nakornping Hospital each month (83 samples).

e: represents the desired margin of error (0.05).

The required sample size (n) was 69 samples. As a result, whole blood samples from 70 babies were taken for this study and the flowchart illustrating the experimental process is shown in Figure 1.

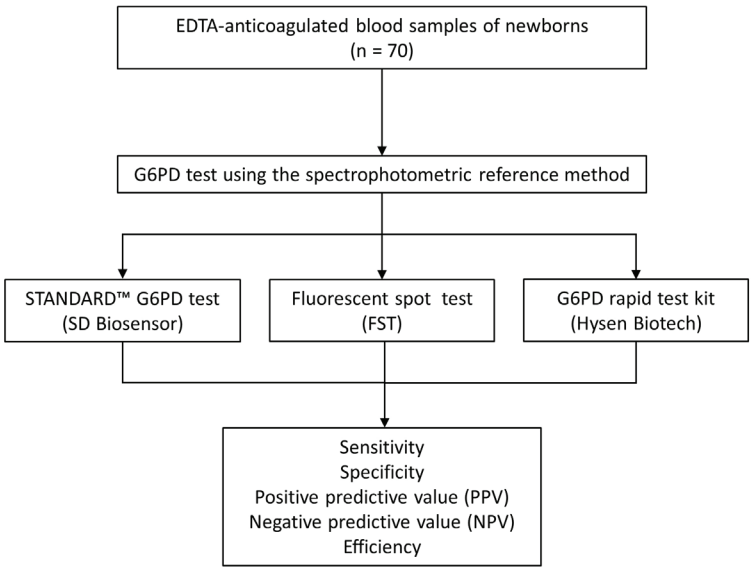

Figure 1. *The flowchart illustrating the experimental process.*

G6PD activity measurement using the SD Biosensor

The principle of SD Biosensor (STANDARD G6PD test, Suwon, Korea) is based on a colorimetric detection system for the automatic calculation of G6PD activity on the code chip for each test device. G6PD catalyzes the first step in the PPP, oxidizing G-6-P to 6-PG and reducing NADP to NADPH. When NADPH is generated by G6PD, the 5-bromo-4-3-indolyl-phosphate (BCIP) and nitro blue tetrazolium (NBT) are reduced by the diaphorase reaction to yield a violet color. The rate of the color production is directly proportional to the concentration of G6PD present in the specimen. The color intensity can be measured through reflectance photometry of the reduced BCIP and NBT. For the test, begin by removing the chip and inserting it into the device. Pipette 10 µL of whole blood using a STANDARD Ezi tube+ into the buffer tube that contains the extraction buffer. Mix with the STANDARD Ezi tube+ 8-10 times. Then, apply 10 µL of the mixed specimen using the STANDARD Ezi tube+ to the application hole of the test device. The result will appear on the screen after 2 minutes. The measuring ranges of the tests were as follows: total Hb 4-25 gm/dL and G6PD 0-20 U/g Hb. Interpretation of the test results was done according to the manufacturers' reference ranges: G6PD activity ≤4.0 U/g Hb was interpreted as G6PD deficiency for both males and females, G6PD activity ≥4.1 U/gm Hb was interpreted as normal for males while G6PD activity between 4.1 and 6.0, and ≥6.1 U/gm Hb was interpreted as G6PD intermediate and normal, respectively for females.

Detection of G6PD deficiency using the fluorescent spot test (FST)

The principle of FST is based on the G6PD enzyme's ability to oxidize G-6-P into 6-PG and reduce NADP to NADPH. The amount of NADPH generated is directly proportional to G6PD activity, observable through its fluorescence when excited by UV light at 340 nm.[23] For the test, 5 µL of whole blood was pipetted into test tube that contained 100 µL of G6PD screening reagent (Sigma-Aldrich, MO, USA) and positive control tube that contained 100 µL of NADP free reagent. The reagents were incubated at room temperature, and 10 µL of the mixture was spotted onto Whatman filter paper after incubation for 5 and 15 minutes. The spots were air-dried and then examined under ultraviolet light with a wavelength range of 340 nm. The blood samples with control reagent showed no fluorescence. Positive results, indicating G6PD deficiency, occur when the spot shows no fluorescence at both 5 and 15 minutes. Negative results, indicating normal G6PD activity, are determined when the spot fluoresces under UV light at both 5 and 15 minutes. In addition, the intermediate G6PD activity can be interpreted when the spot shows no fluorescence at 5 minutes but fluorescence at 15 minutes

Detection of G6PD deficiency using G6PD rapid test kit

The principle of G6PD rapid test kit (Hysen Biotech, Seoul, South Korea) is based on the G-6-P substrate colorimetric method. In the presence of NADP, G-6-P is oxidized by G6PD enzyme in the sample, resulting in the production of 6-PG and NADPH. The light yellow NBT is reduced to insoluble blue-purple crystalline formazan in the reaction between NADPH and phenazine methosulfate (PMS). The experiment was conducted in accordance with the manufacturer's instructions. Specifically, 300 µL of buffer was introduced into the tube containing the reagent pad, which was subsequently shaken to dissolve the colored substance from the pad into the buffer. Following this, 10 µL of whole blood samples were added to the tube. The sample-reagent mixture was incubated for 10 minutes. The card was then taken out and the reacted buffer was deposited into the designated hole of the card. After approximately 1 minute, the liquid was fully absorbed, allowing for

result interpretation. Positive results, indicative of G6PD deficiency, manifest as pink, white, or yellow coloration in the center of the card. Conversely, negative results, signifying normal G6PD activity, present as blue, blue-purple, or black coloration in the center of the detection card.

Statistical analysis

Statistical analyses were performed using Microsoft Excel version 2021. The sensitivity, specificity, positive predictive value (PPV), negative predictive value (NPV), and efficiency of each approach were estimated based on the definitions of positive and negative results, which correspond to G6PD deficient and non-deficient outcomes, respectively. The formulas were as follows:

Sensitivity = true positive/(true positive + false negative) × 100%

Specificity = true negative/(true negative + false positive) × 100%

Positive predictive value (PPV) = true positive/(true positives + false positive) × 100%

Negative predictive value (NPV) = true negative/(true negative + false negative) × 100%

Efficiency = (true positive + true negative)/(true positive + true negative + false positive + false negative) × 100%

Results

Venous blood samples in EDTA anticoagulated tubes were collected from 70 newborns for this study. Their characteristics and hematological data, including total Hb and reticulocyte counts and G6PD enzymatic activity assessed by gold standard method are shown in Table 1. Based on the ultraviolet (UV) spectrophotometric method, a gold standard method, the G6PD deficiency was found in 22 (31.4%) newborns, while 48 (68.6%) newborns had normal G6PD enzyme activity.

The screening results for G6PD deficiency using the FST and G6PD rapid test kit of 70 newborns showed identical outcomes, detecting G6PD deficiency in 22 newborns, while 48 newborns had normal G6PD enzyme activity. Among those, there was one false positive and one false negative case. Thus, the sensitivity, specificity, PPV, NPV, and efficiency were calculated, with the results shown in Table 2.

Among the 70 samples, the SD Biosensor was conducted on 67 neonates due to insufficient blood samples from three individuals for testing. Moreover,

Table 1. Characteristics and hematological data of samples.

Characteristics and hematological data (N=70)		Reference ranges
Age (days)	3.5±2.9 (1-17)	1-28
Gender: Male/Female	45 / 25	
Total Hb (g/dL)	17.3±2.3 (11.5-21.9)	17.0-20.0
Reticulocyte counts (%)	5.0±1.9 (1.7-8.9)	2.0-6.0
G6PD activity measured by gold standard method (U/gm Hb)	18.6±12.9 (1.3-45.5)	≥3.8

Note: *The values are presented as mean±SD (ranges).*

Table 2. Performances of three methods.

	Reference Method		Total	Sensitivity (%)	Specificity (%)	PPV (%)	NPV (%)	Efficiency
	Deficiency	Normal						
FST (N=70)								
Deficiency	21	1	22					
Normal	1	47	48	95.4	97.9	95.4	97.9	97.1
Total	22	48	70					
G6PD rapid test kit (N=70)								
Deficiency	21	1	22					
Normal	1	47	48	95.4	97.9	95.4	97.9	97.1
Total	22	48	70					
SD Biosensor (N=56)								
Deficiency	20	4	24					
Normal	0	32	32	100.0	88.9	83.3	100.0	92.8
Total	20	36	56					

results were indeterminate for 11 of 67 (16.4%) individuals because their total Hb levels exceeded the upper detectable limits (>25 gm/dL) of the SD Biosensor. Based on the gold standard method, 36 of 56 remaining samples had normal G6PD enzyme activity and 20 were G6PD deficiency.

The G6PD deficiency analysis conducted by SD Biosensor revealed that 32 samples had normal G6PD enzyme activity, while 20 samples demonstrated G6PD deficiency. Furthermore, one female identified by SD Biosensor demonstrated intermediate enzyme activity (4.7 u/gm Hb). However, the reference method lacks a defined threshold for intermediate activity, and the G6PD enzyme activity of this sample, analyzed by the gold standard method at 30.3 U/g Hb, was within normal values. Consequently, this case was categorized into the G6PD false positive group for performance evaluations. Additionally, three other false positive cases were identified by SD Biosensor. Thus, the sensitivity, specificity, PPV, NPV, and efficiency were calculated, with the results presented in Table 2.

The performances of each test were analyzed by using the diagnostic results obtained from the gold standard method as references. The results showed that the FST and G6PD rapid test kit had a higher specificity, PPV, and efficiency than the SD Biosensor. However, the former methods had a lower sensitivity and NPV than the later method (Table 2).

Discussion

In this study, we evaluated the performance of FST (Sigma-Aldrich), G6PD rapid test kit (Hysen Biotech), and SD Biosensor (STANDARD™ G6PD Test) for screening G6PD deficiency in newborns by comparing their diagnostic results with those of enzymatic assay, which is considered the gold standard method. The results indicated that both FST and the G6PD rapid test kit exhibited higher specificity, PPV and efficiency compared to the SD Biosensor (Table 2). In contrast, the SD Biosensor demonstrated the highest sensitivity and NPV (Table 2), consistent with previous studies on biosensor-based methods. Pal et al found that the SD Biosensor performed similarly to a reference assay, with a sensitivity of 95.5-100% and specificity of 97%, making it a reliable option for diagnosing G6PD deficiency in both males and females across diverse clinical settings, including resource-limited areas.[20] Similarly, the study by Adu-Gyasi *et al*. reported that the CareStart G6PD deficiency RDT, a biosensor-based method, demonstrated a sensitivity of 100% and a specificity of 72.1%.[24] In the present study, the overall detection rate of the SD Biosensor was 83.6% for all neonates, with 11 tests (16.4%) failing to detect G6PD activity due to high Hb levels. This failure is particularly relevant in newborns, who tend to have higher Hb levels that exceed the detectable range of the SD Biosensor (Hb 4-25 gm/dL). An elevated Hb level (>25 gm/dL) not only affects the absorbance of the colorimetric reaction but can also retard the rate of G6PD enzymatic activity, potentially leading to false positive results as found in four cases (1 intermediate and 3 deficiencies) (Table 2). The G6PD activity in these four cases, as evaluated by the reference method, exceeded the cutoff value (3.8 U/g Hb) for G6PD deficiency, ranging from 19.1 to 30.3 U/g Hb. Consequently, to prevent uncorrected outcomes, the device displayed unanalyzed results in samples with elevated Hb levels, as observed in 11 samples. This makes it less optimal for use as a screening tool in newborns, as accurate screening in this population is crucial. Despite these limitations, the SD Biosensor remains a viable option for detecting G6PD deficiency in other contexts where high sensitivity is essential. However, additional confirmation may be required to avoid false positives.

Both the FST and G6PD rapid test kit are highly suitable for clinical use due to their excellent specificity, PPV, and efficiency; ease of use; and quick turnaround times, approximately 20 minutes and 15 minutes, respectively, for each sample. These methods are particularly advantageous in settings where rapid screening and diagnosis are required, such as newborn care or regions with limited healthcare access.

Several studies have evaluated the performance of the FST in screening for G6PD deficiency. Jiang et al confirmed that the FST is a reliable and convenient screening method, demonstrating high sensitivity (92-100%) and specificity (98%).[25] Similarly, Keihanian *et al*. reported that the FST performed well in clinical settings, with a sensitivity of 91.4%, specificity of 99.9%, NPV of 99.4%, and PPV of 97.7%.[26] Overall, these studies indicate that the FST provides acceptable sensitivity and specificity for detecting G6PD activity in newborns. However, there are certain challenges with this approach. For example, the process requires scientific expertise and UV light.

A study by Tinley *et al*. evaluated the BinaxNOW® G6PD test, a rapid qualitative enzyme chromatographic test (ECT) that detects G6PD activity by reducing nitro blue tetrazolium dye to a blue formazan product, like the G6PD rapid test kit. The test demonstrated high sensitivity (98%) and specificity (97-98%).[27]

In clinical settings, the enzymatic assay remains the gold standard for confirming G6PD deficiency.[26] However, for hospitals with limited equipment or resources, both FST and the G6PD rapid test kit are reliable alternatives. They provide high sensitivity and specificity, and their quick results make them an excellent choice for rapid screening in newborn care settings. On the other hand, the G6PD rapid test kit offers a faster turnaround time compared to FST, making it an attractive option for hospitals with budgetary constraints. Furthermore, even though FST is a qualitative method, it can potentially identify samples with G6PD intermediate activity. Consequently, in clinical practice, the G6PD test should be initially conducted using the FST or G6PD rapid test kit, followed by

quantification of G6PD enzymatic activity in deficient samples using the SD Biosensor or an automated UV enzymatic analyzer at the central laboratory.

Limitations

The limitation of this study is a small sample size (70 newborns), which may limit the generalizability of the results to larger or more diverse populations. Therefore, to verify the validity of these tests across a range of demographics, more research with larger sample sizes and/or a multi-center study is required.

Conclusion

The FST and G6PD rapid test kit are reliable and suitable for G6PD deficiency screening in newborns, especially in settings with limited resources, due to their high efficiency, specificity, and rapid results. The SD Biosensor remains a valuable tool in clinical contexts requiring high sensitivity. For newborns with high hemoglobin levels, FST and G6PD rapid test kit are recommended for accurate screening. Further studies with larger sample sizes are necessary to confirm the reliability of these tests in diverse populations.

Ethical approval

This study was approved by the Ethics Committee of the Faculty of Associated Medical Sciences at Chiang Mai University (approval No. AMSEC67EM-035). In addition, it was also subsequently submitted to the Nakornping Hospital Ethics Committee for approval regarding research involving human subjects (approval No. NKP165/67).

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Conflict of interest

The authors state no conflict of interests.

CRediT authorship contribution statement

Jiranan Neamyanon: conceptualization, investigation, methodology, writing original draft; **Aungkana Saejeng:** conceptualization, resources, review and editing; **Phaithoon Wongwian:** blood and data collections, investigation, review and editing; **Suparporn Kiti:** blood and data collections, investigation, review and editing; **Satitpong Nanjai:** blood and data collections, investigation, review and editing; **Thaworn Jaiyasen:** blood and data collections, investigation, review and editing; **Sakorn Pornprasert:** conceptualization, project administration, validation, writing, reviewing and editing.

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