


# BMJ Open Prevalence of and mothers' knowledge, attitude and practice towards glucose-6-phosphate dehydrogenase deficiency among neonates with jaundice: a cross-sectional study

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## ABSTRACT

**Background** Neonatal jaundice (NNJ) is a frequent complication of glucose-6-phosphate dehydrogenase (G6PD) deficiency.

**Objectives** To estimate the prevalence of G6PD deficiency among neonates with jaundice and to assess mothers' perception towards G6PD and NNJ.

**Methods** A cross-sectional study was carried out on 487 ethnic Egyptian neonates with indirect hyperbilirubinaemia from June 2018 to July 2019. The collected data included maternal and neonatal characteristics. Laboratory investigations included serum bilirubin, reticulocyte count, ABO grouping, Rh typing and neonatal serum G6PD test. Mothers were interviewed individually using a structured, researcher-administered questionnaire to assess their perceptions of G6PD deficiency and NNJ.

**Results** The prevalence of G6PD deficiency was 10.10%. Neonates with G6PD deficiency showed higher levels of serum bilirubin ( $p < 0.001$ ). Male gender, family history of G6PD deficiency and consanguinity were risk factors for G6PD deficiency (OR=4.27, 95% CI 1.66 - 10.99; OR=9.54, 95% CI 4.80- 18.95; OR=10.219, 95% CI 5.39 - 19.33, respectively). Mothers' perceptions of NNJ and G6PD were low, with only 30% having good knowledge on NNJ and 17.10% on G6PD deficiency, 46.8% with positive attitude towards NNJ and 45.0% towards G6PD deficiency, and 29.9% with good practice towards NNJ and 19.9% towards G6PD deficiency.

**Conclusion** G6PD deficiency seems to be an important cause of NNJ. Mothers' perceptions of both NNJ and G6PD deficiency were low. A mass health education programme on both of these diseases is needed to ensure better and early detection, good timing of treatment, and better prevention of the triggering factors to ensure better health for children.

## INTRODUCTION

The term 'jaundice' is used to describe the yellow-orange discolouration of the skin and conjunctiva due to excessive bilirubin in the skin and mucous membranes.<sup>1 2</sup> It is not a disease, but a symptom or sign of a disease.<sup>3</sup> Jaundice (hyperbilirubinaemia), although a

## Strengths and limitations of this study

- The study collectively assessed the prevalence and risk factors of glucose-6-phosphate dehydrogenase (G6PD) deficiency, aside from assessing mothers' level of knowledge, attitude and practice (KAP) towards both G6PD deficiency and neonatal jaundice (NNJ).
- The study clarified the extent of change in NNJ based on previous levels published in some research articles in the same region, and also drew on how G6PD deficiency is a poorly known disease despite it being serious.
- A suitable sample size was studied in a short period and allowed us to reach a large number of mothers and families.
- There was a need to carry out more investigations to mothers, such as a direct Coombs test.
- A post-test to assess the extent of understanding and KAP among mothers was needed; however, it was difficult to collect the same number of study participants one more time.
- KAP towards these diseases needs to be assessed in the general population to ensure risk factors are identified and addressed, and we also tried to help mothers deliver and share with their families the knowledge they gained from the health education session that was conducted.

common benign occurrence in the first week of life, can sometimes progress to critical levels.<sup>4</sup> Neonatal jaundice (NNJ) is a frequent complication of glucose-6-phosphate dehydrogenase (G6PD) deficiency, which is a genetic disease more often observed in male patients as this is an X linked enzymatic deficiency. However, in female patients, it might present deficient activity levels severe enough to induce haemolysis even if they are heterozygous.<sup>5 6</sup> African, Asian, Mediterranean and Middle-Eastern descents are most commonly affected by this disorder.<sup>7 8</sup> The prevalence

of G6PD deficiency among Egyptian neonates is 8.9%.<sup>9</sup> G6PD enzymatic deficiency was first reported in India, and its prevalence varies a lot from place to place.<sup>9</sup> Haemolysis in patients with G6PD deficiency can be triggered by specific agents, including certain foods, drugs and infections.<sup>6</sup> Avoiding the triggers and starting treatment early, including intensive phototherapy, to decrease the incidence of severe haemolysis are simple and inexpensive and can be started before the symptoms appear.<sup>8–10</sup> Counselling with regard to avoiding the risks of jaundice and triggering agents should be directed to parents of deficient newborns.<sup>10</sup> G6PD deficiency is commonly known in Egypt as fava bean anaemia or favism (after intake of fava bean), as this enzyme deficiency increases the susceptibility of red blood cells to agents such as oxidants present in raw beans, some medications and oxidative stress caused by infections.<sup>11</sup> There are few studies conducted on the prevalence of G6PD deficiency among Egyptian neonates with jaundice,<sup>9</sup> as well as on the knowledge, attitude and practice (KAP) towards NNJ or G6PD deficiency.<sup>11,12</sup> However, data on the prevalence and measurement of KAP in both NNJ and G6PD deficiency from a single study are not available. This study aimed to estimate the prevalence of G6PD deficiency among Egyptian neonates with jaundice and assess mothers' perceptions by studying their level of KAP towards G6PD deficiency and NNJ to ensure avoidance of triggering factors.

## SUBJECTS AND METHODS

A cross-sectional study was carried out in Egypt on 487 neonates with indirect hyperbilirubinaemia from June 2018 to June 2019 at three Egyptian neonatal and paediatric centres, medically serving their population and also receiving patients from the surrounding areas. Mothers with their neonates visiting these paediatric and neonatal centres to seek medical advice for treatment of jaundice were from some Egyptian governorates, mainly Menoufia Governorate (the place of the study) and the surrounding governorates. At 0–10 days of age, admitted term and preterm neonates with clinically evident jaundice were included in the study. The exclusion criteria were neonates with direct hyperbilirubinaemia >20% (conjugated hyperbilirubinaemia exists when more than 20% of the total bilirubin or more than 2 mg/dL is conjugated; if neither criterion is met, hyperbilirubinaemia is classified as unconjugated), metabolism errors, congenital anomalies and sepsis. All these exclusion criteria were set to be more focused on G6PD deficiency as a single cause of NNJ in this study. The collected data included maternal and neonatal characteristics such as gestational age (on admission), parity, gravidity, neonatal gender, weight and age of onset of jaundice (maternal recall). The neonates had been previously subjected to laboratory investigations, including serum bilirubin (total, direct, indirect), reticulocyte count, ABO grouping and Rh typing of the mother and the baby, Coombs test (for the baby only), and C reactive protein (as routine and to

exclude sepsis). Ultraviolet-kinetic method using cellular enzyme determination reagents by spectrophotometry was used to obtain a quantitative estimation of serum G6PD from 1 mL of whole blood collected in an EDTA tube. G6PD deficiency was defined as <4.6 g/L Hb.<sup>10</sup> The neonates were subjected to phototherapy according to phototherapy guidelines for hospitalised infants from ≥35 weeks of gestation.<sup>13</sup> To assess perception of G6PD deficiency and NNJ, mothers of neonates with jaundice (N=487) and mothers of neonates currently with no jaundice (n=3) (excluded from the study due to very small number) were interviewed individually during admission by a trained team of data collectors. Mothers of neonates receiving medical examination received researcher-administered questionnaire. We interviewed mothers of neonates with and without jaundice while seeking medical advice. The mothers of neonates with jaundice were interviewed before informing them about having a G6PD deficient baby. The questionnaire was designed by experts in paediatrics and public health specialties, and was based on their experience in paediatrics and public involvement, as well as on published reviews of literature. A pilot study was conducted on about 30 mothers (about 6% of the calculated sample) to test the adequacy of the questionnaire with regard to content, language and time consumed to answer, and to explore the potential obstacles and difficulties with regard to execution of the study. It was also used as a tool to train the team of data collectors (five nurses and three junior staff) to avoid interobserver and intraobserver bias. Training was conducted with the working team for 2 days, followed by assessment of the degree of response to training and the quality of asking questions and of reporting data. For NNJ, the questionnaire (online supplementary questionnaire) included questions such as the mother's knowledge on diagnosis, causes, complications and treatment. With regard to the attitude of mothers towards NNJ and its treatment, the questions included whether the mother thinks that NNJ is a worrisome condition and so on. With regard to practice, the questionnaire asked whether the mother would seek medical advice and so on. For G6PD deficiency, the questionnaire (online supplementary questionnaire) included questions such as whether the mother has ever heard about G6PD deficiency or its common term fava bean anaemia. At this point, we continued our questions using the common term. However, during data analysis, we used the scientific term to avoid misunderstanding among the readers. The questionnaire included questions such as whether G6PD deficiency is a blood disease, whether both parents have to be carriers of G6PD deficiency, whether the inheritance of G6PD deficiency is related to the baby's gender, agents that can trigger G6PD deficiency such as fava beans and medications, pallor, shortness of breath, whether G6PD deficiency is a cause of jaundice, and whether gastrointestinal tract (GIT) symptoms such as nausea and vomiting are symptoms of G6PD deficiency. With regard to attitude, the mothers were asked whether they see this as a serious problem, whether

**Table 1** General characteristics of mothers and neonates with jaundice

General characteristics	Study group (N=487)	
<b>Mothers' characteristics</b>		
Age (years), mean±SD (range)	31.45±4.77	(39–22)
<b>Gestational age (weeks)</b>		
Mean±SD (range)	37.71±1.05	(41–37)
Median (IQR)	37	37–38
BMI (kg/m <sup>2</sup> ), mean±SD (range)	22.02±2.37	(27.10–18.30)
<b>Gravidity, n (%)</b>		
≤3	292	(60.0)
>3	195	(40.0)
<b>Parity, n (%)</b>		
≤2	252	(51.7)
>2	235	(48.3)
<b>Neonatal characteristics</b>		
<b>Gender, n (%)</b>		
Male	339	(69.6)
Female	148	(30.4)
Birth weight (kg), mean±SD (range)	2.60±0.29	(3.50–2.30)
Age of neonate (days), mean±SD (range)	4.45±0.86	(8–3)
<b>Bilirubin (mg/dL), mean±SD (range)</b>		
Total	15.17±5.14	(25.50–7.30)
Direct	1.08±0.38	(0.50–1.50)
Indirect	13.17±3.74	(23.15–6.40)
Hb (g/dL), mean±SD (range)	12.18±1.75	(14.50–9.50)
Reticulocyte count (%), mean±SD (range)	3.38±1.30	(6.0–1.40)
Age of onset of jaundice (maternal recall), mean±SD (range)	3.45±0.85	(7–2)
<b>Need for phototherapy on admission, n (%)</b>		
G6PD deficiency	5	(0.20)
All causes	23	(4.7)
Family history of G6PD deficiency, n (%)	144	(29.6)
Consanguinity, n (%)	103	(21.1)
ABO incompatibility, n (%)	63	(12.9)
Rh incompatibility, n (%)	47	(9.7)

BMI, body mass index; G6PD, glucose-6-phosphate dehydrogenase.

marriage between contagious couples consanguineous couples is a cause and so on. With regard to practice, the questionnaire included questions on seeking medical advice (a general question not specifying the current condition), premarital counselling and so on. Answers were scored as 1 (for correct) and 0 (for incorrect),

and participants with at least 60% correct answers were considered to have good knowledge. The correct answer was determined for any single or multiple right answers in order to help estimate the final score. Participants with at least 60% positive answers were considered to have positive attitude and practice. A health education talk, with adequate clarification of the diseases, was given to mothers by the researcher through an organised special day at a conference room.

### Sample size calculation

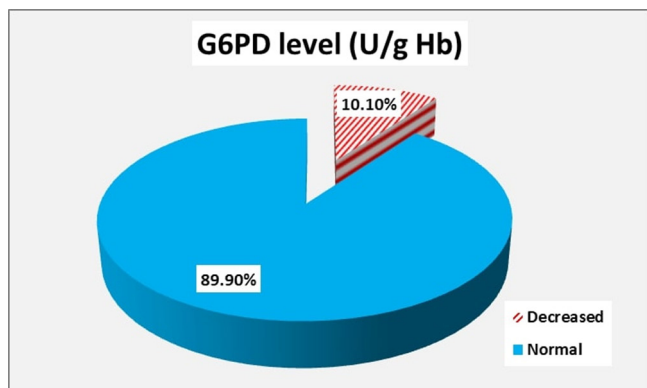
Based on a previous review of literature<sup>10</sup> which reported an 8.9% prevalence of G6PD among newborns with jaundice with nearly the same inclusion and exclusion criteria as our study, the sample size was calculated using the following equation:  $n=(z^2 \times p \times q)/D^2$ , at 95% CI. The sample size was estimated to be 487 neonates with jaundice.

### Statistical analysis

Data were analysed using SPSS V.22. Descriptive statistics in the form of percentage (%), mean±SD and range were performed. An independent t-test and analysis of variance were used for normally distributed quantitative variables, while  $\chi^2$  was used for qualitative variables. OR was used to assess the risk of exposure: OR=1: exposure does not affect the odds of outcome; OR >1: exposure is associated with higher odds of outcome; and OR <1: exposure is associated with lower odds of outcome. Pearson's correlation was used to assess the direction and strength of association between the variables. A p value less than 0.05 was set as statistically significant.

### Patient and public involvement

This work aimed to study the prevalence of G6PD deficiency among Egyptian neonates with jaundice and mothers' perception of these diseases. To improve the relevance of research, research oriented to patients and the public is vital. A paper-based survey asked some mothers seeking medical advice in the three neonatal and paediatric centres to submit unanswered questions regarding G6PD deficiency and NNJ. The final top 4 research priorities derived from an inperson meeting were ranked. Thirty respondents submitted 40 questions. The respondents were from urban and rural areas, and their age ranged from 20 to 40 years. The 40 questions were refined to 17 unique questions, and from this list the top 4 research questions of priority were derived, namely whether these diseases are infectious, whether they can be transmitted to the next generation, whether they are a lifelong disease and whether there is complete cure. The observers administered questionnaires to the respondents to assess their degree of response and reactivity. The interviewed mothers recommended generalising the screening over a large population and conducting it in the most crowded districts. Hence we asked them to tell every pregnant woman they suspect to have a yellow baby to seek medical advice for free in the neonatal centres



**Figure 1** Prevalence of glucose-6-phosphate dehydrogenase (G6PD) deficiency among the group of neonates with jaundice. Hb, Hemoglobin.

where we are working and encourage them to participate in the study. The 30 women helped us recruit approximately 92 women. The rest of the sample was based on our advertisement, plus the usual patients coming on their own to the study centres. We organised a special day at a conference room to thank all participants, disseminate the results and provide an indepth group health education session about the two diseases, with special focus directed to mothers of G6PD-deficient babies on avoiding the triggering factors and seeking prompt medical advice. For other mothers, the main aim of the health education session was to correct wrong information and to build knowledge base for new mothers so they know well of these diseases and share this knowledge to their families and to others in their community.

## RESULTS

The study was conducted on 478 neonates with jaundice. Mothers' age ranged from 22 to 39 years ( $31.45 \pm 4.77$ ). Neonates were aged 0–10 days, with 69.6% male and 30.4% female. Their birth weight ranged from 2.30 to 3.50 kg. Bilirubin levels (mg/dL) included total bilirubin of  $15.17 \pm 5.14$ , direct bilirubin of  $1.08 \pm 0.38$  and indirect bilirubin of  $13.17 \pm 3.74$ . Mean Hb (g/dL) showed a good level at about  $12.18 \pm 1.75$  despite the low and high range (9.50–14.50). The total percentage of neonates with jaundice who needed phototherapy on admission was 4.7%. G6PD-deficient cases who needed phototherapy on admission represented about 0.20% of the total neonates with jaundice. Family history of G6PD deficiency and consanguinity were reported among 29.6% and 21.1%, respectively. ABO incompatibility and Rh incompatibility were detected in 12.9% and 9.7%, respectively (table 1). The prevalence of G6PD deficiency was 10.10% ( $< 4.6$  u/g Hb for 42 male neonates ( $2.88 \pm 0.95$ ) and for 7 female neonates ( $4.0 \pm 0.57$ )) (figure 1). Neonates with G6PD deficiency showed higher levels of bilirubin (total, direct and indirect) ( $p < 0.001$ ). In this population of neonates with jaundice, G6PD-deficient neonates were more likely to be of male gender (OR=4.27, 95% CI 1.66–10.99), to be born to consanguineous parents (OR=10.21, 95% CI 5.39–19.33) and to have family history of G6PD deficiency (OR=9.54, 95% CI 4.80–18.95) (table 2). A positive correlation was observed between G6PD level among G6PD-deficient neonates and time of onset of jaundice based on maternal recall ( $r=0.436$ ,  $p=0.002$ ). One of the interesting findings was that total bilirubin was higher in G6PD-deficient cases ( $23.03 \pm 2.94$ , CI 22.18–23.87,

**Table 2** Distribution of the G6PD groups according to bilirubin, neonate gender, family history and consanguinity

	G6PD				Test of significance	P value	OR (95% CI)
	Deficient (n=49)		Normal (n=438)				
	Mean±SD		Mean±SD				
<b>Bilirubin (mg/dL)</b>							
Total	23.03±2.94		14.30±4.55		t=18.40	<0.001*	–
Direct	1.38±0.14		1.02±0.41		t=12.47	<0.001*	
Indirect	17.02±3.45		12.74±3.52		t=8.21	<0.001	
<b>Neonate gender</b>							
	n	%	n	%	$\chi^2=10.49$	0.001*	4.27 (1.66 to 10.99)
Male	42	85.7	297	45.0			1.0
Female	7	14.3	141	55.0			
<b>Family history of G6PD deficiency</b>							
Positive	37	75.5	107	24.4	$\chi^2=55.21$	<0.001*	9.54 (4.80 to 18.95)
Negative	12	24.5	331	75.6			1.0
<b>Consanguinity</b>							
Positive	33	67.3	70	16.0	$\chi^2=69.72$	<0.001*	10.21 (5.39 to 19.33)
Negative	16	32.7	368	84.0			1.0

\*Significant family history: not in the immediate family but in their relatives.  
G6PD, glucose-6-phosphate dehydrogenase.

**Table 3** Knowledge, attitude and practice of mothers towards G6PD deficiency (N=487)

Knowledge	n	%
Hearing about G6PD deficiency (per say).		
Yes	2	4.1
No	485	96
Hearing about fava bean anaemia (G6PD deficiency).		
Yes	112	23
No	375	76
G6PD deficiency is a blood disease.		
Yes	100	89
No	12	11
I don't know	0	0
Is it a hereditary disease?		
Yes	105	94
No	5	4.5
I don't know	2	1.8
Parents (both or just the mother) have to be carriers of G6PD deficiency to have an affected child?		
Yes	5	4.9
No	10	8.9
I don't know	101	90
The inheritance of G6PD deficiency related to the baby's gender?		
Yes	10	8.9
No	3	2.7
I don't know	99	88
Knowing whether personally you may have a child with G6PD deficiency.		
Yes	8	7.1
No	85	76
I don't know	29	26
There should be a family history of G6PD deficiency to result.		
Yes	75	67
No	15	13
I don't know	62	55
Some medications can trigger G6PD deficiency.		
Yes	44	39
No	10	8.9
I don't know	58	52
Symptoms of G6PD		
Pallor		
Yes	10	8.9
No	52	46
I don't know	60	54
Nausea, vomiting, anorexia and diarrhoea		
Yes	15	13
No	45	40

Continued

**Table 3** Continued

Knowledge	n	%
I don't know	52	46
Dizziness		
Yes	30	27
No	5	4.5
I don't know	77	69
Shortness of breath		
Yes	35	31
No	5	4.5
I don't know	72	66
G6PD is a cause of jaundice.		
Yes	105	94
No	2	1.8
I don't know	5	4.5
<b>Attitude</b>		
Is this a serious problem?		
Yes	100	89
No	5	4.5
I don't know	7	6.3
Consanguinity is the cause of the disease.		
Yes	40	36
No	20	18
I don't know	52	4.3
Next pregnancy should be prevented.		
Yes	3	2.7
No	50	45
I don't know	59	53
Follow-up of the diseased child should continue for life.		
Yes	100	89
No	2	1.8
I don't know	10	8.9
<b>Practice</b>		
Have you been subjected to premarital counselling?		
Yes	112	100
No	0	0
Have you been subjected to genetic screening?		
Yes	2	1.8
No	110	98
I don't know	0	0
Seeking medical advice after delivery to be assured.		
Yes	0	0
No	112	100

G6PD, glucose-6-phosphate dehydrogenase.

**Table 4** Knowledge, attitude and practice of mothers towards NNJ (N=487)

Knowledge	n	%
<b>Hearing about NNJ</b>		
Yes	487	100
No	0	0
<b>Site to detect NNJ</b>		
Skin	334	68.6
Eye	123	25.2
Tongue	30	6.2
<b>Causes</b>		
Prematurity	341	70
ABO disparity between mother and baby	73	15
Breast feeding	146	30
Infection	139	33
Haemolysis	194	39.8
Dehydration	170	34.9
Increased ultrasound examination during pregnancy	292	60
Mothers with diabetes	73	15
Others	141	28.9
<b>Complications</b>		
Death	146	30
Cerebral palsy	112	23
Mental retardation	170	34.9
Handicapping	112	23
Hearing loss	30	6.2
<b>Methods of treatment</b>		
Phototherapy	461	94.7
Blood exchange transfusion	146	30
Drugs	399	82
Neon lamp	364	74.7
Increase breast feeding	238	48.9
<b>Attitude</b>		
<b>NNJ is a worrisome condition?</b>		
Yes	248	50.9
No	200	41.1
I don't know	39	8
<b>Phototherapy is the best method for treatment.</b>		
Yes	409	84
No	8	1.6
I don't know	70	14.4
<b>Blood exchange transfusion is the best method for management.</b>		
Yes	107	22
No	50	10.3
I don't know	330	67.7
<b>Is it important to seek medical advice?</b>		

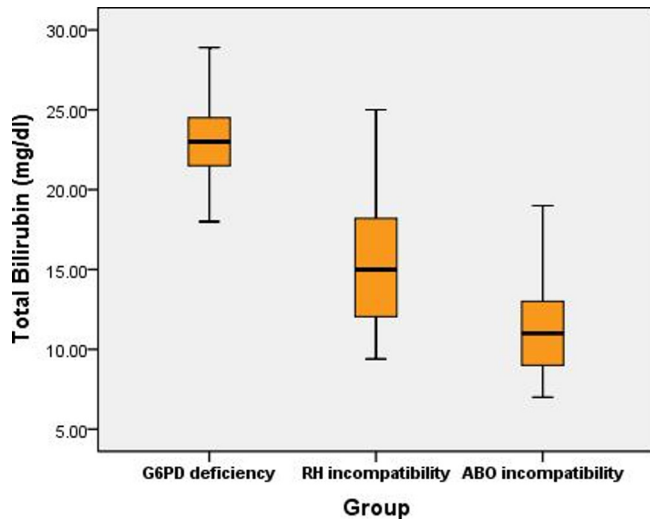
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**Table 4** Continued

Knowledge	n	%
Yes	450	92.4
No	20	4.1
I don't know	18	13.5
<b>Practice</b>		
<b>Seeking medical advice quickly if baby has NNJ.</b>		
Yes	477	95.9
No	10	4.1
I don't know	0	0
<b>Reasons for denial of medical care</b>		
n=10		
Afraid of hospitalisation	6	60
Admission/investigation not required	1	10
High cost of medical care	2	20
Lack of transportation	0	0
Long hours to reach hospital	0	0
<b>Time of seeking medical advice</b>		
Within 24–48 hours	136	28.5
>48 hours	341	71.5
<b>Continuation of breast feeding</b>		
Yes	448	92
No	39	8

NNJ, neonatal jaundice.

M=23, IQR: 21.3–25) than those with Rh incompatibility (15.7±4.75, CI 14.33–17.12, M=15, IQR: 11.6–18.2) or ABO incompatibility (11.0±2.59, CI 10.49 to 11.79, M=11, IQR: 9–13) (figure 2). With regard to KAP towards NNJ and G6PD deficiency, it seems that mothers showed a somehow better perception towards jaundice in comparison with G6PD deficiency. Unfortunately KAP towards both diseases was low, as majority of mothers (95.9%) did not know the term G6PD deficiency while about 24% of them had heard about fava bean anaemia. Also 90% of them did not know that parents (both or just the mother) have to be carriers of G6PD deficiency to have an affected child. All mothers knew fava beans can trigger G6PD deficiency, while 39.3% knew that it is triggered by drugs (table 3). Almost all mothers knew about NNJ. About 70% of them thought that prematurity is a cause of NNJ, 68.6% knew that they can detect jaundice through the skin of their newborn, while 25% reported that jaundice can be detected through the sclera of their newborn. About 95% of the mothers knew that phototherapy is a method of treatment for NNJ (table 4). Good knowledge on NNJ was reported in 30% vs 17.10% on G6PD deficiency, positive attitude towards NNJ was reported in 46.8% vs 45.0% towards G6PD deficiency, and finally good practice towards NNJ was reported in 29.9% vs 19.9% towards G6PD deficiency (figure 3).

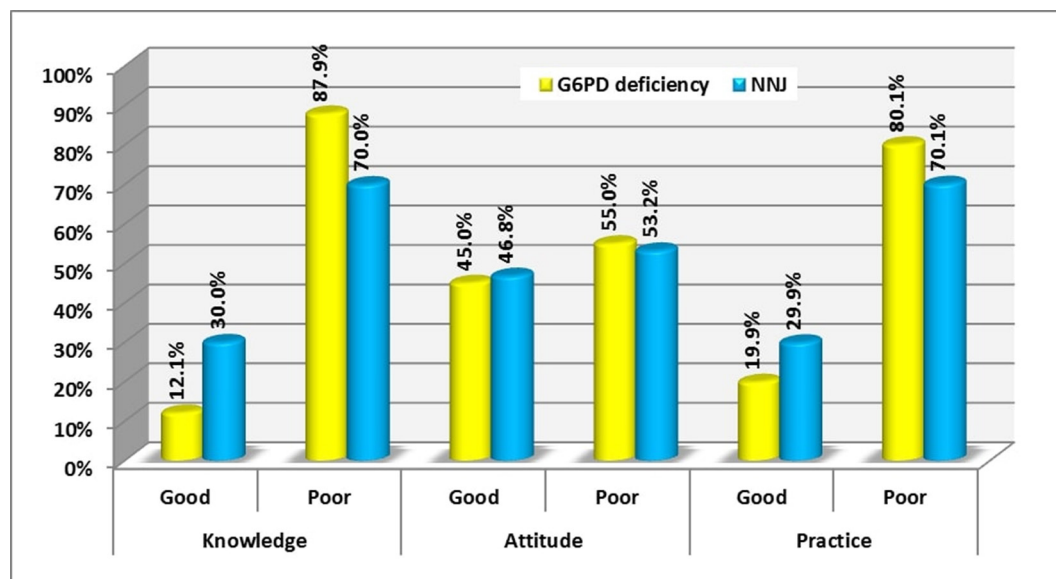


**Figure 2** Distribution of total bilirubin among patients with glucose-6-phosphate dehydrogenase (G6PD) deficiency, Rh incompatibility and ABO incompatibility.

## DISCUSSION

The prevalence of G6PD deficiency in neonates with jaundice was reported at 10.1%, which is within the range of the prevalence revealed in some Egyptian studies (8.9%–30.2%).<sup>9–12</sup> The higher prevalence of G6PD deficiency among neonates with jaundice (30.2%) in El-Menshay *et al*<sup>12</sup> may be due to the small-sized purposive sample chosen to conduct the study. The wide range of G6PD deficiency prevalence in Egypt could be explained by the country's special geographical position, being situated between three continents with different ethnic groups. The case is the same at the global scale. In Iraq, the prevalence was 10.65%,<sup>13</sup> while in Iran it was around 9%<sup>14</sup> and in South Brazil 7.9%.<sup>15</sup> Neonates with G6PD deficiency showed higher levels of bilirubin, and this result is in parallel to

that of Bahraini and Nigerian studies conducted by Isa *et al*<sup>16</sup> and Badejoko *et al*,<sup>17</sup> respectively. Male gender is associated with a higher risk for G6PD deficiency, which is similar to the findings reported in Egypt by Abo El Fotoh and Rizk,<sup>9</sup> Elella *et al*,<sup>10</sup> and El-Menshay *et al*,<sup>12</sup> in India by Sinha *et al*,<sup>18</sup> and in Iran by Eghbalian and Monsef.<sup>19</sup> Family history of G6PD deficiency and consanguinity are risk factors for G6PD deficiency, which coincide with an Egyptian study conducted by Abo El Fotoh and Rizk<sup>10</sup> and Garg and Joag.<sup>20</sup> On the contrary, a study conducted in Japan stated that only one case of G6PD deficiency was born to non-consanguineous Japanese parents without any family history.<sup>21</sup> A positive correlation between G6PD deficiency and time of onset of jaundice (based on maternal recall) was found. As known, there are two peak time points for patients with jaundice to be admitted: the first is on the third day and the second is on the seventh day of life. Bimodal peaks of maximum serum bilirubin concentrations are known to occur among G6PD-deficient infants, and when the haemolytic episode starts early the elevation of serum bilirubin is anticipated to be clear and hence a course of hyperbilirubinaemia may be predicted.<sup>22</sup> This finding is in accordance with Abo El Fotoh and Rizk,<sup>10</sup> but in disagreement with a Turkish study conducted by Atay *et al*.<sup>23</sup> Mean Hb (g/dL) showed a good level at about 12.18±1.75 despite the low and high range (9.50–14.50), and this is normal for Egyptians as the prevalence of anaemia among Egyptian pregnant women is approximately 52.5%.<sup>24</sup> The total percentage of neonates with jaundice who needed phototherapy on admission was 4.7%. G6PD-deficient cases who needed phototherapy on admission represented about 0.20% of the total neonates with jaundice. The neonates were subjected to phototherapy according to phototherapy guidelines for hospitalised infants from ≥35 weeks of gestation.<sup>25</sup> In G6PD deficiency, hyperbilirubinaemia



**Figure 3** Perception of glucose-6-phosphate dehydrogenase (G6PD) deficiency and neonatal jaundice (NNJ) among mothers.

is thought to be secondary to reduced hepatic conjugation and excretion of bilirubin, rather than increased bilirubin production resulting from haemolysis.<sup>18</sup> Total bilirubin was obviously higher among G6PD-deficient cases than those with Rh or ABO incompatibility, and this finding agrees with that concluded by Das and Singh<sup>26</sup> in India and Hussain *et al.*<sup>27</sup> in Pakistan, but also in contrast to the findings obtained by Shah and Yeo<sup>28</sup> in Singapore and Aletayeb *et al.*<sup>29</sup> in Iran. KAP towards G6PD deficiency showed that majority of the mothers (95.9%) did not know the term G6PD deficiency, which does not agree with Al-Joborae,<sup>30</sup> who found that about 91% of mothers in Iraq had heard about G6PD deficiency. In Egypt, the most commonly used term is 'Fava bean anemia', so in our study 23% of mothers had heard about fava bean anaemia and only 4.1% knew the term 'G6PD deficiency anemia'. All mothers knew that fava beans can trigger an attack of haemolysis due to G6PD deficiency, hence the term, and this is in agreement with a study carried out in Bahrain by Al Arrayed and Al Hajeri.<sup>31</sup> In this study, about 40% of mothers thought that haemolysis can be triggered by drugs, and this result is inconsistent with that obtained by Almuahini *et al.*<sup>32</sup> The current study revealed that all mothers have heard about NNJ and about 70% of them reported that prematurity causes its occurrence. This result is consistent with Magfour *et al.*<sup>33</sup> in Saudi Arabia. Jaundice can be detected in infants' skin and sclera by 68.6% and 25% of mothers, respectively. These findings agree with that obtained by Aggarwal *et al.*<sup>34</sup> in India. The study was performed on a carefully selected group of mothers aiming to evaluate their KAP. However, the study also benefited the group as most of them were first-time mothers of neonates with jaundice, so it seemed for us as the case of studying of a group of general population, besides this group will share the health education message efficiently as it is based on self-experience. Mothers showed a somehow better perception towards jaundice in comparison with G6PD deficiency. This is in agreement with the results reported by Boo *et al.*<sup>35</sup> in Malaysia. Our results however still show poor KAP towards these diseases, which is in agreement with Goodman *et al.*<sup>36</sup> in Nigeria and Alfouwais *et al.*<sup>37</sup> in Saudi Arabia. In contrast to this study's results, Al-Joborae<sup>30</sup> in Iraq and Al Arrayed *et al.*<sup>32</sup> reported that mothers had a fairly good level of awareness of G6PD deficiency. The results of this study showed some improvement in the level of knowledge in comparison with Allahony *et al.*<sup>38</sup> who reported that only 18.9% of mothers had good knowledge, 48.0% had good attitude and only 25.3% had good practice towards NNJ, which reflect the effect of health education carried out to the mothers in their study and which also show that we are still in dire need of more extensive and focused health education. The results of this study showed that the risk factors for hyperbilirubinaemia were prematurity, ABO incompatibility and infection, which are in agreement with Saadat *et al.*<sup>39</sup> and Rabiepoor *et al.*<sup>40</sup> in Iran.

## Strengths

The study collectively assessed the prevalence and risk factors of G6PD deficiency, aside from assessing the level of KAP towards G6PD deficiency and NNJ. The study clarified the extent of change in NNJ based on previous levels published in some research articles in the same region, and also drew on how G6PD deficiency is a poorly known disease despite it being serious. It is recommended that special health education sessions be provided to mothers in health centres from day 1. A suitable sample size was studied in a short period and allowed us to reach a large number of mothers and families.

## Limitations

There was a need to carry out more investigations to mothers, such as a direct Coombs test. A post-test to assess the extent of understanding and KAP among the mothers was also needed; however, it was difficult to collect the same number of study participants one more time. We reached only the mothers who sought medical advice for their neonates. KAP towards these diseases needs to be assessed in the general population to ensure risk factors are identified and addressed, and we tried to help mothers deliver and share with their families the knowledge they gained from the health education session that was conducted.

## CONCLUSION

G6PD deficiency seems to be an important cause of NNJ. Cord blood for complete blood count, direct Coombs test, blood grouping, bilirubin and G6PD screening in high-risk populations are recommended to determine earlier whether a prolonged hospital stay is needed. G6PD deficiency and NNJ are serious conditions. The study revealed that mothers' KAP towards NNJ, despite being low, shows promising improvement. In contrast, mothers' KAP towards G6PD deficiency is very poor. It is important to provide mass health education programmes on G6PD deficiency and NNJ to ensure better and early detection, good timing of treatment, and better prevention of the triggering factors to ensure better health for children.

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