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    - Colson JH, Armour WJ. Sport injuries and their treatment. 2nd rev eds. London: S. Paul, 1986.

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Surat kabar: nama pengarang. Judul, Kompas 2007; April 10:2 (koll), 5 (kol2)

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Situs web/internet:

- Artikel/jurnal dalam format elektronik:
  McCook A. Pre-diabetic condition linked to memory loss. Diunduh dari http://www.nlm.nih.gov/medlineplus/news_11531.html 3 Februari 2007.

Disertasi:

Wila Wirya IGN: Penelitian beberapa aspek klinik dan patologi anatomis sindrom nefrotik idiopatik pada anak di Indonesia. Jakarta: FKUI, 1992. Disertasi

Sumber dari jurnal tanpa Pengarang:

Anonim: Coffee drinking and cancer of the pancreas (Editorial). Br Med J. 1981; 283: 628.

Prosiding pertemuan ilmiah:

Vidianty J, Pardede SO, Trihono PP, Hidayati EL, Alatas H, Tambunan T. Gambaran antropometri pada anak dengan sindrom nefrotik. Prosiding pertemuan ilmiah tahunan Ilmu Kesehatan Anak (PIT IKA) III Ikatan Dokter Anak Indonesia (IDAI), Yogyakarta, 2007: 75-8.

Tabel: ketik atau cetak setiap tabel dengan dua spasi pada lembar terpisah. Setiap tabel diberi judul singkat dan nomor berurut sesuai dengan urutan pengutipannya yang pertama kali di dalam teks.

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Afiliasi: apakah sudah lengkap	ya □	tidak □
Abstrak:		
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2. Bahasa Indonesia?	ya □	tidak □
3. Bahasa Inggris?	ya □	tidak □
4. Terdiri atas paling banyak 250 kata	ya □	tidak □
5. Kata kunci?	ada □	tidak ada □
Pendahuluan		
1. Ada/tidak ada	ada □	tidak □
2. Tujuan penelitian?	ada □	tidak □
Metode		
1. Ada	ada □	tidak □
2. Tidak ada	ada □	tidak □
3. Memuat semua metode yang akan dikerjakan?	ada □	tidak □
Hasil		
1. Memuat hasil penelitian	ya □	tidak □
2. Terpisah dari diskusi	ya □	tidak □
Diskusi		
Terpisah dari Hasil	ya □	tidak □
Daftar Pustaka		
1. Disusun menurut cara Vancouver?	ya □	tidak □
2. Sebagian besar pustaka 10 tahun terakhir?	ya □	tidak □

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No	Nama	Penulis	Tanda Tangan	Email
1.		Koresponden	……	……..
2.		Pertama	……...	……..
3.		Pendamping	……..	……..
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Abstrak:

1. Satu paragraf ?	ya □	tidak □
2. Bahasa Indonesia?	ya □	tidak □
3. Bahasa Inggris?	ya □	tidak □
4. Terdiri atas paling banyak 250 kata	ya □	tidak □
5. Kata kunci?	ada □	tidak ada □

Pendahuluan

1. Ada/tidak ada	ada □	tidak □

Pelaporan Kasus

1. Apakah metode diagnostik terapeutik dan alat yang digunakan dicantumkan dengan jelas (merk, tahun dll)	ya □	tidak □
2. Apakah identifikasi subjek ditutupi (anonimitas)	ya □	tidak □

Diskusi terpisah dari hasil	ya □	tidak □

Daftar Pustaka

1. Disusun menurut cara Vancouver?	ya □	tidak □
2. Sebagian besar pustaka 10 tahun terakhir?	ya □	tidak □

Persetujuan penulis

No	Nama	Penulis	Tanda Tangan	Email
1.		Koresponden	……	……..
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Afiliasi: Apakah sudah lengkap	ya □	tidak □
Abstrak:		
1. Satu paragraf ?	ya □	tidak □
2. Bahasa Indonesia?	ya □	tidak □
3. Bahasa Inggris?	ya □	tidak □
4. Terdiri atas paling banyak 250 kata	ya □	tidak □
5. Kata kunci?	ada □	tidak ada □
Pendahuluan	ada □	tidak ada □
Isi sesuai judul?	ya □	tidak □
Daftar Pustaka		
1. Disusun menurut cara Vancouver?	ya □	tidak □
2. Sebagian besar 10 tahun terakhir?	ya □	tidak □

Persetujuan penulis

No	Nama	Penulis	Tanda Tangan	Email
1.		Koresponden	……	……..
2.		Pertama	……...	……..
3.		Pendamping	…….	……..
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5.		Pendamping	…….	……..
6.		Pendamping	…….	……..
7.		Pendamping	…….	……..

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Editorial

Pengaruh Pekerjaan sebagai Pengelas Terhadap Kesehatan

Retno Wahyuningsih

Majalah Kedokteran UKI

Proses pengelasan merupakan proses yang sangat penting dalam industri yang menggunakan logam sebagai bahan produksi. Proses pengelasan (*welding*) dalam industri, misalnya mobil, berguna untuk menggabungkan komponen logam sehingga menjadi hasil akhir yang dapat digunakan. Dalam proses tersebut digunakan panas sangat tinggi yang dapat mencapai 4000ºC. Tingginya panas yang dihasilkan dapat mengakibatkan terlepasnya berbagai zat berbahaya seperti logam dan gas ke udara.[1] Sharifian *et al.*[2] melakukan studi kohort di Iran terhadap pekerja pengelasan dan menemukan bahwa pekerjaan pengelasan yang berlangsung cukup lama dapat mempengaruhi kesehatan terutama terhadap fungsi paru. Beberapa peneliti lain menyatakan bahwa toksin maupun gas yang dihasilkan pada proses pengelasan berpengaruh terhadap kesehatan paru.[3,4] Pada terbitan kali ini Achore *et al*., menulis hasil studi kohort tentang pengaruh proses pengelasan pada pekerja yang telah bekerja sebagai tukang las (*welders*) selama beberapa tahun. Hasilnya akan melengkapi pengetahuan kita tentang pengaruh proses pengelasan terhadap kesehatan.

Stunting yang saat ini merupakan "*hot issue*" di Indonesia,[5] juga tidak terlepas dari perhatian para peneliti. Dalam nomor ini juga diterbitkan artikel hasil penelitian tentang *stunting* di Sumedang, Jawa Barat oleh Reviani *et al*. yang membahas berbagai faktor yang berhubungan dengan *stunting*.

Selain itu Utomo *et al.,* menulis tentang rinitis alergika, sementara Wulandari *et al*., menyampaikan hasil penelitian tentang lalat yang ditangkap di berbagai warung di wilayah Jakarta Barat ternyata dapat bertindak sebagai sumber penularan parasit usus. Masih tentang parasit usus Ronny menyampaikan telaahnya tentang *Blastocystis hominis* di kalangan wistawan yang memanng sedamg *booming* saat ini. Wisatawan tentu perlu memperhatikan kesehatannya agar dapat menikmati perjalananya. Akhirnya tidak lengkap terbitan ini bila tidak disertai laporan kasus yang kali ini ditulis oleh Suling tentang Sindrom Wellens Tipe A, bentuk kelainan jantung yang jarang ditemukan.

Selamat membaca.

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Artikel Asli

Evaluation of Long-Term Respiratory Effects of Exposure to Welding Fumes

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Abstract

Welding fumes are known to cause respiratory health problems. We aimed to evaluate the long-term respiratory effects of exposure to welding fumes. Inception cohorts of welding, plumbing, and heating apprentices were prospectively contacted 7-17 years post-apprenticeship. Questionnaires, as well as spirometry and non-specific bronchial hyperresponsiveness (NSBHR) tests were repeatedly administered. A long-term evaluation was done in 71 former apprentices at the Hôpital du Sacré-Cœur de Montréal between 2013 and 2017. Post-apprenticeship exposure to welding fumes and gasses was evaluated using three methods: self-report, expert assessment and the asthma-specific job exposure matrix. The incidence of wheezing and excessive lung function decline, given continued post-apprenticeship exposure to welding fumes, was estimated using Cox regression. Incident wheezing was found in 18.8% of subjects, and excessive lung function decline was observed in 12.7% of subjects. All three exposure assessment methods consistently showed that subjects with continued, post-apprenticeship exposure to welding fumes or gasses had a lower risk of developing wheezing symptoms or excessive decline in lung function, although none of the associations were significant. In conclusion, continued post-apprenticeship exposure to welding fumes does not seem to increase the risk of developing long-term respiratory outcomes.

Keywords: apprentices, lung function, occupational, welding

Evaluasi Dampak Jangka Panjang Pajanan Asap dan Gas Pengelasan Terhadap Kesehatan Pernapasan

Abstrak

Asap dan gas pengelasan *(welding fumes and gasses)* diketahui dapat menyebabkan masalah pernapasan. Penelitian ini dilakukan untuk mengevaluasi efek pernapasan jangka panjang dari pajanan asap dan gas pengelasan. Mantan peserta sekolah kejuruan pengelasan secara prospektif dihubungi 7-17 tahun pasca-pendidikan. Subjek penelitian melengkapi kuesioner, uji spirometri dan uji bronkus non-spesifik. Evaluasi jangka panjang dilakukan pada 71 subjek di Hôpital du Sacré-Cœur de Montréal antara tahun 2013 dan 2017. Pajanan asap dan gas pengelasan pasca-pendidikan dievaluasi menggunakan tiga metode: laporan oleh pekerja, penilaian oleh ahli dan penggunaan matriks pajanan di tempat kerja pekerjaan khusus. Hubungan antara pajanan asap dan gas pengelasan dengan insidens mengi dan penurunan fungsi paru-paru berlebihan dievaluasi menggunakan regresi Cox. Insidens mengi ditemukan pada 18,8% subjek, dan penurunan fungsi paru berlebihan diamati pada 12,7% subjek. Ketiga metode penilaian pajanan secara konsisten menunjukkan bahwa subjek dengan pajanan asap dan gas pengelasan pasca-pendidikan memiliki risiko lebih rendah terkena gejala mengi atau penurunan fungsi paru-paru yang berlebihan, meskipun tidak ada hubungan yang bermakna. Sebagai kesimpulan, pajanan asap dan gas pengelasan jangka panjang tampaknya tidak meningkatkan risiko penurunan fungsi pernapasan.

Kata kunci: fungsi paru, pekerjaan, asap dan gas pengelasan

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Introduction

Work-related asthma includes work-exacerbated asthma (pre-existing asthma or concurrent asthma that is worsened by workplace exposures) and OA (asthma induced by agents in the workplace). On the basis of general population-based studies, 17.6% of adult-onset asthma is attributable to occupational exposure.[1] More than 400 different agents in the workplace have been identified as causing OA,[2] including high-molecular-weight, HMW, (mostly proteins) and low-molecular-weight, LMW, (mostly chemical) agents. For the former, the most common causal agents include flour and laboratory animal allergens, to which bakers and laboratory animal workers are respectively exposed to. For the latter, the most common etiologic agents include cleaning products, isocyanates and wood dust, to which health care workers, cleaners, car painters, and carpenters might be exposed to.[3] OA is associated with work impairment [4,5] impaired quality of life, and psychological distress.[6,7]

The international multicenter population-based Respiratory Health in Northern Europe study showed that adult-onset rhinitis and asthma were consistently higher among welders across population samples from Northern Europe.[8] Our previous study, comprising an apprentice cohort of welders in the province of Quebec that were followed up for 18 months, demonstrated exposure to welding fumes to be associated with respiratory symptoms and pulmonary function changes.[9] A substantially increased number of subjects with percent predicted forced expiratory volume in one second (%pFEV1) <80% and non-specific bronchial hyperresponsiveness (NSBHR) were observed.[9]

The current study aimed to: (1) estimate post-apprenticeship incidence of wheezing and excessive decline in lung function; (2) assess the association between post-apprenticeship, continued exposure to welding fumes and gasses and these outcomes; and (3) assess the association between work productivity and activity impairment with lower respiratory symptoms suggestive of OA.

Methods

Design and Sample

To answer our study objectives, we re-contacted the inception cohort of 286 welders and 44 plumbing and heating apprentices, 7-17 years post-apprenticeship. Between September 2013 and April 2017, 90 of 330 (27.2%) eligible subjects participated in a long-term follow-up.

Standardized respiratory, work history, as well as work impairment and activity impairment (WPAI) for specific health problems questionnaires, were administered at follow-up. In addition, participants underwent a lung function test using spirometry and a methacholine challenge test to assess NSBHR.[9,10] The study was approved by the Hôpital du Sacré-Cœur de Montréal research ethics committee (CER 2012-801).

Measurements

Two primary outcomes were evaluated. First, the incidence of wheezing (absent at the end of apprenticeship) was considered present if subjects reported wheezing in the past 12 months. Second, an excessive decline in lung function from the end of apprenticeship to the end of follow-up was defined as a decrease in $\%pFEV_1 \geq 15\%$ based on Knudson equation.[11] Our secondary outcome was WPAI assessed using questionnaires.[12] Absenteeism (work time missed), presenteeism (reduced on-the-job effectiveness), work productivity loss

(overall work impairment: absenteeism plus presenteeism), and activity impairment in the past seven days were calculated.

Demographic and clinical determinants of incident wheezing and excessive respiratory decline at follow up were explored. NSBHR was defined as the provocative concentration of methacholine causing a 20% decrease in FEV1 (PC20) ≤8 mg/ml. We also evaluated changes in lung function and bronchial responsiveness to methacholine during apprenticeship and post-apprenticeship. An increase in bronchial hyper-responsiveness during apprenticeship was defined as (1) having a 3.2-fold decrease in PC_{20} from baseline to the end of apprenticeship, or (2) a change at the end of apprenticeship to a PC_{20} of 16 mg/ml or less in subjects with an initial PC_{20} of greater than 32 mg/ml.[13]

Exposure Assessment

Three methods were used to assess continued, post-apprenticeship exposure to welding fumes and gasses: self-report, expert assessment, and asthma-specific job exposure matrix (JEM). Subjects were asked to report the type of jobs, tasks assigned, and any welding activities performed at their workplace. Since self-reports are prone to recall bias, an occupational hygienist used information reported by participants on their job(s), task(s) and the company(ies) in which they worked to classify their exposures. For each job reported, a three-point score (0, 1 and 2) was used by the hygienist to assess exposure. A zero score indicated no exposure to welding fumes; 1 indicated possible exposure (i.e., working in a welding environment); and 2 implied probable exposure (i.e., having a job with welding task). Subjects were categorized as being continuously exposed if any of the jobs were coded as one or two and held for one year or longer. Finally, the International Standard Classification of Occupations (ISCO-88) codes assigned by the hygienist to post-apprenticeship held-jobs were subsequently linked with the JEM. Subjects were considered to have had "current exposure" if they were in occupations involving exposure to welding fumes (i.e., welders) or irritant gases or fumes (i.e., plumbers) for at least one year. Unexposed subjects or those who had been exposed for less than a year post-apprenticeship in their current job, composed the reference group. A similar approach was previously used by our group to evaluate post-apprenticeship outcomes in other cohorts.[13,14]

We included 71 subjects who had questionnaire responses and respiratory function test results at the end of the apprenticeship. Kappa coefficients and percentage of the agreement were computed to evaluate agreement between self-report, expert assessment and JEM exposure categories on estimates of continued exposure to welding fumes and gasses post-apprenticeship.[15] For estimating the incidence of wheezing, we excluded subjects with missing wheezing information (n=8) or those who reported wheezing at the end of apprenticeship (n=6). Cox regression was used to estimate hazards ratios (HRs) and corresponding 95% confidence intervals (CIs) for the associations between each of characteristics at the end of apprenticeship and continued exposure to welding fumes with the incidence of primary outcomes. WPAI outcomes were expressed as impairment percentages, with higher numbers indicating greater impairment and less productivity, i.e., worse outcome.[12] Data analyses were performed using IBM SPSS for Windows version 24.0 (SPSS, Inc., Chicago, IL).

Results

Participants who did not participate were similar to those who participated regarding

their characteristics (age, smoking status, BMI, wheezing and NSBHR) at the end of apprenticeship (results not shown).

The incidence of wheezing was 21% (12 of 57 subjects). An excessive decline in $\%pFEV_1$ was found in 9 out of 71 (12.7%) subjects with respiratory function test results.

Subjects reported having held up to a maximum of 12 jobs when they were contacted after 17 years. Based on expert assessment and JEM, almost all (69 of 71 subjects) have ever had a job with exposure to welding fumes or gasses. Based on self-report, 45 (78.9%) subjects have ever been exposed. As for the current job, according to expert-assessment, 37 of 71 (52.1%) subjects have ever been exposed to welding fumes for at least one year. The number exposed to welding fumes were slightly lower (49.2%) based on asthma-specific JEM, and much lower (38.0%) based on self-report. The percent agreement was 86.0% between expert assessment and JEM (kappa coefficient (k) 0.719, $p<0.001$), and 85.9% ($k=0.721$, $p<0.001$) between expert assessment and self-report and 74.7% ($k=0.491$, $p<0.001$) between self-report and JEM.

Table 1 shows the distribution of demographic, clinical, and exposure characteristics by the incidence of wheezing during the long-term follow-up. As

Table 1. Distribution of Demographic, Clinical, and Exposure Characteristics By the Incidence Of Wheezing During Long-Term Follow-Up

	Incidence of Wheezing		HR (95% CI)
	No N=47	Yes N=13	
Characteristics at the end of apprenticeship			
Mean age (SD), years	26.1 (8.1)	23.9 (6.5)	0.98 (0.88-1.08)
Male sex	38 (84.4)	9 (75.0)	0.74 (0.19-2.76)
Current smoking	16 (35.6)	5 (41.7)	1.31 (0.41-4.13)
Body mass index (mean, SD)	25.2 (3.8)	22.6 (3.1)	0.85 (0.71-1.01)
Overweight (body mass index ≥ 25 kg/m^2)	20 (44.4)	3 (25.0)	0.59 (0.16-2.18)
Predicted FEV1, mean (SD)	97.7 (14.6)	98.4 (22.5)	1.01 (0.98-1.04)
Non-specific bronchial hyperresponsiveness ($PC_{20} \leq 8$ mg/mL)	5 (11.1)	5 (41.7)	2.22 (0.69-7.04)
Tightness	5 (11.1)	3 (25.0)	1.63 (0.44-6.09)
Cough	1 (2.2)	1 (8.3)	2.17 (0.27-17.24)
Changes during apprenticeship			
Excessive decline in percentage of FEV1	6 (13.3)	1 (8.3)	0.59 (0.08-4.63)
Increase in bronchial responsiveness	6 (13.3)	3 (25.0)	1.16 (0.31-4.33)
Post-apprenticeship exposure to welding fumes			
Self-reports	17 (37.8)	4 (33.3)	0.52 (0.16-1.73)
Expert assessment	24 (53.3)	6 (50.0)	0.95 (0.31-2.97)
Asthma-specific job exposure matrix	23 (51.1)	7 (58.3)	1.48 (0.47-4.68)
Work duration (years)			
Self-reports (median, IQR)	1.7 (0, 8.2)	1.4 (0, 8.7)	0.97 (0.87-1.08)
Expert assessment (median, IQR)	0 (0, 7.5)	0 (0, 8.0)	0.94 (0.83-1.06)
Asthma-specific job exposure matrix (median, IQR)	1.2 (0, 7.5)	4.6 (0, 8.8)	0.94 (0.83-1.06)

Data are in n (%) unless otherwise noted.
CI: confidence interval; FEV1: forced expiratory volume in 1 second; HR: hazard ratio; IQR: interquartile range; PC20: methacholine concentration that causes a 20% fall in FEV1; SD: standard deviation.

displayed in Table 1, NSBHR, cough or chest tightness, or being a smoker at the end of apprenticeship were associated with a higher risk of developing wheezing (HRs ranged between 1.3 and 2.2). However, no statistically significant associations were observed. The observed associations between current welding exposure and wheezing were inconsistent across the exposure assessment type (HRs ranged from 0.5 to 1.5), with none reaching statistical significance.

Table 2 shows a non-significant increased risk of excessive lung function decline in subjects who were overweight and in those who reported wheezing, chest-tightness or cough at the end of the apprenticeship. All three exposure assessment methods consistently showed that subjects who were currently exposed to welding fumes had a lower incidence of excessive lung function decline. The univariate HRs ranged from 0.4 to 0.8.

Only two subjects reported a WPAI due to respiratory symptoms one week prior to the interview. One of them is an overweight male welder aged 40-years old, who also reported to be a smoker. The other is a male welder with persistent wheezing since the end of apprenticeship but had normal lung function. He was overweight at follow-up.

Table 2. Distribution of Demographic, Clinical, and Exposure Characteristics By Excessive Respiratory Decline During Long-Term Follow-Up

	Excessive respiratory decline		HR
	No	Yes	95% CI
	N=62	N=9	
Characteristics at the end of apprenticeship			
Mean age (SD), years	25.3 (7.8)	23.9 (6.5)	1.01 (0.89-1.14)
Male sex	52 (83.9)	7 (77.8)	0.54 (0.10-2.78)
Current smoking	27 (45.8)	3 (33.3)	0.65 (0.16-2.63)
Body mass index (mean, SD)	24.4 (3.9)	24.4 (3.7)	1.04 (0.82-1.30)
Overweight (body mass index $\geq$ 25 kg/m^2)	18 (33.3)	4 (57.1)	3.09 (0.67-14.24)
Predicted FEV1, mean (SD)	96.5 (15.6)	113.1 (14.3)	1.04 (0.99-1.09
Non-specific bronchial hyperresponsiveness ($PC_{20} \leq 8$ mg/mL)	12 (19.4)	1 (11.1)	0.59 (0.07-4.98)
Wheezing	6 (10.9)	1 (11.1)	1.28 (0.15-10.68)
Tightness	3 (8.3)	1 (20.0)	1.54 (0.16-14.97)
Cough	2 (3.6)	2 (22.2)	3.08 (0.61-15.65
Changes during apprenticeship			
Excessive decline in pFEV1	9 (14.5)	1 (11.1)	0.75 (0.09-6.49)
Increase in bronchial responsiveness	9 (14.8)	1 (11.1)	0.49 (0.06-4.02)
Current exposure to welding fumes			
Self-reported	24 (38.7)	3 (33.3)	0.42 (0.11-1.70)
Expert-assessment	33 (53.2)	4 (44.4)	0.65 (0.17-2.43)
Asthma-specific JEM	31 (50.0)	4 (44.4)	0.83 (0.22-3.11)
Work duration (years)			
Self-reported (median, IQR)	0 (0, 6.2)	0 (0, 8.8)	0.94 (0.82-1.08)
Expert-assessment (median, IQR)	1.4 (0, 7.0)	0 (0, 8.8)	0.97 (0.85-1.09)
Asthma-specific JEM (median, IQR)	1.0 (0, 6.9)	0 (0, 8.8)	0.94 (0.82-1.08)

Data are in n (%) unless otherwise noted.
CI: confidence interval; FEV1: forced expiratory volume in 1 second; HR: hazard ratio; IQR: interquartile range; PC20: methacholine concentration that causes a 20% fall in FEV1; SD: standard deviation

Discussion

Our findings suggest that subjects with continued, post-apprenticeship exposure to welding fumes or gasses had a lower risk of developing wheezing or an excessive decline in lung function. The observed associations were consistent across all exposure assessment methods. A healthy worker effect (HWE) might explain our findings. This is in accordance with a study in a New Zealand urban population where cumulative exposure to gases/fumes was associated with a higher %pFEV$_1$, suggesting a likely consequence of an HWE.[16]

We observed a higher incidence of wheezing in subjects who were smokers, reported chest tightness or a cough or had NSBHR at the end of the apprenticeship. We also found a higher incidence of excessive lung function decline in subjects who were overweight or reported lower respiratory symptoms at the end of the apprenticeship. Although associations did not reach statistical significance, this is concomitant with a previous study by Wang and colleagues who found that being overweight was related to a lower level and steeper slope of decline in pulmonary function.[17]

Two symptomatic welders reported mild to moderate WPAI in the past week. None reported missing work or reducing hours of work due to respiratory symptoms, but reported work impairment by 10-20% and activity impairment by 20-40%. The WPAI questionnaires are designed to inquire about impairment in work productivity and activities up to 7 days.[12] Therefore, our findings might not reflect the potential long-term impact of occupational exposure on work productivity and activity.

There are potential limitations to this study. First, the sample size was small. As such, for current exposure-outcome associations, our reference group consisted of unexposed subjects or those who were exposed for less than a year post-apprenticeship. This could have resulted in underestimating the observed associations. Second, we had a low participation rate (27.2%). Non-response in observational epidemiologic studies may bias association estimates because of potential selection bias.[18] In our study, however, the characteristics at the end of the apprenticeship of subjects who participated in our long-term follow-up did not significantly differ from those who did not participate.

Conclusion

Post-apprenticeship exposure to welding fumes and gases was not associated with an increased risk of developing long-term respiratory outcomes. An inverse association was found between post-apprenticeship exposure to welding fumes and long-term respiratory disease development.

Conflict of Interest

The authors have no conflicts of interest associated with the material presented in this paper.

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Artikel Asli

Profile of Allergic Rhinitis Based on Nasal Eosinophil Count, Total Nasal Symptoms Score and Peak Nasal Inspiratory Flow

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Abstract

Allergic rhinitis (AR) is a symptomatic disorder of the nose induced after allergen exposure by an immunoglobulin E (IgE)-mediated inflammation of the membranes lining the nose. The manifestation of AR can affect patients' quality of life. The objective of this study was to evaluate the profile of allergic rhinitis patients in term of nasal eosinophil count, total nasal symptom score (TNSS) and peak nasal inspiratory flow (PNIF). Fourteen allergic rhinitis patients were evaluated using the nasal eosinophil count with Wright-Giemsa staining, total nasal symptom score, and PNIF. The study comprised of 6 (42.9%) men, and 8 (57.1%) women with a mean of age 21.15±3.78 years. Participants' symptoms and characteristics included sneezing (42%), nasal blockage (21.4%), itchy nose (21.4%), and rinorrhea (14.3%) with a mean TNSS of 8.2±1.8. Intermittent AR was found in 14.3% subjects, persistent AR 78.6%, mild AR 21.4%, and moderate-severe AR 78.6%. The mean PNIF was 80±27.46 L/min, and mean eosinophil count of 2.5±1.74. In conclusion, allergic rhinitis patients demonstrated positive nasal eosinophil count with Wright-Giemsa staining, with the majority of them having persistent and moderate-severe RA. They also had nasal airflow impairment, which could affect quality of life.

Keywords: allergic rhinitis, eosinophil, nasal airflow, quality of life.

Profil Rinitis Alergika Berdasarkan Hitung Eosinofil, Total *Nasal Symptoms Score* dan *Peak Nasal Inspiratory Flow*

Abstrak

Rhinitas alergika (RA) merupakan kelainan pada hidung yang diinduksi oleh pajanan alergen yang berhubungan dengan reaksi inflamasi yang diperantarai IgE pada mokusa hidung. Manifestasi RA dapat mempengaruhi kualitas hidup pasien. Tujuan penelitian ini adalah simptomatik evaluasi profil pasien RA dari segi hitung eosinofil nasal, skor total sindrom nasal (TNSS) dan aliran inspirasi nasal (PNIF). Empat belas pasien RA dievaluasi jumlah eosinofil nasal dengan pewarnaan Wright-Giemsa, skor total simtom nasal dan PNIF. Terdapat 6 (42,9%) laki –laki dan 8 (57,1%) perempuan dengan umur rata-rata usia 21,15±3.78 tahun. Simtom dan karakteristik pasien yaitu bersin (42%), sumbatan hidung (21,4%), hidung gatal (21,4%) dan rinorea (14,3%), dengan rata-rata TNSS 8.2±1.8. Rinitas alergika intermiten ditemukan pada 14,3% subyek, yang persisten 78,6%, sedang 21,4% serta sedang-berat 78,6%. Rata-rata PNIF 80±27.46 L/menit, serta rata-rata jumlah eosinofil 2.5±1.74. Dapat disimpulkan pasien RA menunjukkan jumlah eosinofil nasal positif dengan pewarnaan Wright- Giemsa dan kebanyakan memiliki RA yang persisten dengan tingkat sedang-berat. Terdapat juga gangguan aliran udara hidung yang mungkin mempengaruhi kualitas hidup mereka.

Kata Kunci: rinitis alergika, eosinofil, aliran udara hidung, kualitas hidung

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Introduction

Allergic rhinitis is a symptomatic disorder of the nose induced after allergen exposure by an immunoglobulin E (IgE)-mediated inflammation of the membranes lining the nose.[1,2] The symptoms of nasal reactions occurring in allergies are sneezing, nasal obstruction, mucous discharge (rhinorrhea) and /or itching of the nose.[2] These symptoms occur during two or more consecutive days for more than 1 hour on most days.[3]

The prevalence of allergic disease in the United States (US) is 20%, and is still increasing.[4] A survey published in 2006 recorded that 54.6% of individuals in the US test positively for at least 1 allergen.[5] The prevalence of AR in the Asia-Pacific countries such as Australia, China, Hong Kong, Malaysia, Philippines, Taiwan and Vietnam, ranges on average from 4.2-13.2%.[6] Allergic rhinitis is rarely found in people under the age of 5 years old, and it peaks between the ages of 17 and 22 years old.[5] With regard to the relationship between gender and AR, cases are found in women more often than man.[7, 8]

Allergic rhinitis develops from environmental, immunologic and genetic factors.[9] Environmental factors include aeroallergens, such as mites and cockroaches, and are significantly related to the persistence of RA. The degree of AR is significantly related the allergen concentration in the environment.[10] Indonesia is a country without a spring season, which can lead to lighter symptoms because of the allergen exposure and low concentration over a period of years (perennial).[11] Other environment factors such as temperature, humidity, pollution and lifestyle, significantly influence the manifestation of AR. The immunologic factor is related to the role of Th1 and Th2. The latest concept of allergic disease is the role of regulatory T cells (Treg), and the atopic diseases are caused by a deficiency of Treg. The Treg play an important role in the regulation of Th1 and Th2.[12] Genetic factors such as the atopic history of the family, is the strongest factor in the development of allergic symptoms.[13]

Allergic rhinitis is not a dangerous disease, but the symptoms can affect patient's quality of life.[14] The cost of this disease can make an impact on a nation's economy, and half of the cost is used for prescription medication.[1] A decrease in productivity and missed work can cause economic problems, and AR can impact, health insurance due to exacerbations and complications of allergic disease.[15]

Allergic rhinitis can be diagnosed by anamnesis, physical examination and additional examination by using allergic tests, whether it is in vivo or in vitro, but on allergic tests, that many more people have positive result than have AR.[1] Patients with nasal symptoms who had a family atopic history can have a stronger AR diagnosis. Nasal eosinophil count can be used as an additional examination in the health facilities that have limited skin prick tests.[16] The degree of AR symptoms can be measured by using a scoring system of symptom that is easily understood by the patient, which will be related to daily activity. The total nasal symptoms score (TNSS) is the overall symptom score of AR, and is often used to evaluate the AR treatment.[17,18] Higher values on the TNSS show more severe AR symptoms. Allergic rhinitis manifestation based on the duration of the symptoms can be classified as intermittent and persistent. The degree of severity can be classified as mild, if there are no problems in daily activity, and moderate-severe if there are problems in daily activity.[2] Nasal airflow and patency can be objectively examined by using peak nasal inspiratory flow (PNIF).[19] Low value for PNIF shows that there is reduced nasal airflow and patency.

The aim of this study was to determine the profile of allergic rhinitis patients in terms of nasal eosinophil count, total nasal symptom score and peak nasal inspiratory flow.

Material and Methods

Patients

Fourteen patents with allergic rhinitis symptoms, six men and eight women had a detailed clinical history and a complete physical examination. The diagnosis of persistent, intermittent, mild and moderate-severe allergic rhinitis was made according to ARIA guidelines and on the basis of one's history of allergy to house dust.

Study Design

This research was a cross-sectional study of patients aged 15-55 years old diagnosed with AR based on ARIA-WHO 2008 criteria who came to the Ear Nose and Throat Department at Rumah Sakit Umum UKI, Jakarta in 2016. The participants were recruited with consecutive sampling technique. All patients were asked for their informed consent to be participants in this study. Patients with acute and chronic upper respiratory infections within 30 days before the study, anatomic nasal disorders (i.e., septum deviation), nasal polyps, nasal or oral corticosteroids within the previous 4 weeks, and use antihistamines within the previous week were excluded from the study.

Symptom assessment, nasal eosinophil count and peak nasal inspiratory flow were performed in all patients. This study obtained ethical approval from Rumah Sakit Umum UKI Jakarta.

Nasal Symptoms

The following symptoms were assessed with clinical interview: nasal obstruction, sneezing, rhinorrhea, and itchy nose. Each symptom was evaluated on the following scale: 0 = absent, 1 = mild (symptom was present but was not annoying or troublesome), 2 = moderate (symptom was frequently troublesome but did not interfere with either normal daily activity or sleep), and 3 = severe (symptom was sufficiently troublesome to have interfered with normal daily activity or sleep). Total nasal symptom score was the sum of each individual symptom and was considered as described in previous reports.[20]

Nasal Eosinophil Count

The nasal eosinophil count was determined by scraping the head of the inferior turbinate with a cytobrush (usually used for cervix cytology examination, made in Indonesia with Swedish license), rotating it three times, creating a smear with the Wright-Giemsa stain and analyzed by optic microscope (Olympus CX21LED, Olympus Corporation, Tokyo, Japan). The number of eosinophils was expressed as a mean of 10 optical fields at a magnification of 100.

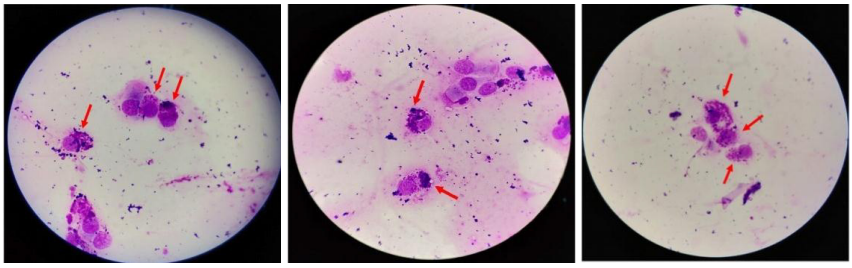

Figure 1. Displays three images showing nasal mucosal eosinophils (red arrows) in Wright-Giemsa staining.

Peak Nasal Inspiratory Flow

Nasal airflow and patency were measured by peak nasal inspiratory flow with In-Check Nasal, Clement Clarke International Limited, as described in previous reports,[20] in the following way: . Hold the In-Check horizontally, and ensure the face mask forms an air tight seal around the nose. The patients were asked to exhale fully, close their mouth, and inhale forcefully through their noses (sniff). The examination was repeated to obtain three readings, with the highest reading recorded in the patient's notes, (L/Min).

Figure 2. Peak Nasal Inspiration Flow (PNIF).

Statistical Analysis

The research data were collected, tabulated and processed. The data then were analyzed descriptively in term of percentages, means and standard deviations (SDs), and the data presented in tables.

Results

Fourteen patients with AR, 6 (42%) men and 8 (57.1%) women with a mean of age 21.15±3.78 years were included in the study and had the following nasal symptoms; sneezing (42%), itching (21.4%), nasal blockage (21.4%) and rhinorrhea (14.3%), with a mean TNSS of 8.2±1.8 (Table 1). Intermittent AR was found in 14.3% patients, persistent AR 78.6%, mild AR 21.4%, and moderate-severe AR 78.6%. The mean PNIF was 80±27.46 L/Min, while the mean nasal eosinophil count was 2.5±1.74 cells.

Discussion

This study examined patients with AR with a mean of age 21 years old. Similarly, other study showed the peak incidence of AR occurred between 17 to 22 years of age.[5] The number of AR cases among women was higher then men. Other studies also found significant differences between men and women, such that AR was found more often in women than in men.[8, 21]

The common clinical symptoms of AR are itchy nose followed by recurrent sneezing, rhinorrhea and nasal blockage.[2] In the other cases, these symptom were followed by itchy eyes, itchy ear, and itchy palatum molle. In this study, sneezing was the most common symptom, which is consistent with other study where 90% of the participants complaining of severe sneezing.[22]

The mean TNSS in this study was 7.92±1.9, which is comparable to other study that found the TNSS in patients with AR was 7.1±2 and without AR 1.9±1.[23] A mean TNSS of 7.9 shows that the AR symptoms were severe. This study found that most cases of AR, based on the time period of symptoms, were persistent AR. The persistence of AR correlates significantly with the presence of aeroallergens, such as mites and cockroaches.[24] This study also

Table 1.The Characteristics of Allergic Rhinitis Patients

Variable	(%) / Mean (Standard Deviation) n:14
Sex	
Men	6 (42.9%)
Women	8 (57.1%)
Age in years	21.15±3.78 (min=15, max=31)
Symptom of AR	
Itching	3 (21.4%)
Sneezing	6 (42.9%)
Rhinorrhea	2 (14.3%)
Nasal blockage	3 (21.4%)
Total nasal symptom score	7.92±1.9 (min=4, max=12)
Duration of symptom	
Intermittent	2 (14.3%)
Persistent	12 (85.7%)
Degree of severity	
Mild	3 (21.4%)
Moderate-severe	11(78.6%)
Nasal eosinophil count	2.5±1.74 cells (min=0.9, max=6.9)
Peak nasal inspiratory flow	80.77±26.91 L/Min (min=45, max=130)

found that most of AR, based on the degree of severity, were moderate-severe, occurring in 78.6% of the sample. The severity of AR correlated with the allergen concentration in the environment.[10]

The mean PNIF in this study was 80 L/Min was lower than normal (120 L/Min).[25] The low value for PNIF indicates the presence of barriers to air flow in the nose. Airway blockage of the nose can effect the quality of life. The nasal blockage is mostly caused by the obstruction of nasal mucous and is rarely caused by fluid (rhinore). Histamines are not the main factors that cause blockages in the nose; rather, nasal blockages are usually caused by other factors, such as cysleukotriene (cysLT1) and thromboxane A_2 (TXA_2). Processes involved in nasal blockage include the loss of sympathetic tonus caused by nervous irritation in the mucous of the nose, vasodilatation of the blood vessels in the nose, and contraction of the vena cushion and venule compression because of the dilated artery in the intraosseous canal from periosteal cavity.[26]

Conclusion

Allergic rhinitis patients had an increased nasal eosinophil count. The most frequent AR cases were persistent and moderate-severe based on the total nasal symptom score. Additionally, there was nasal airflow impairment, which could affect quality of life.

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Factors Associated with the Incidence of Stunting in 18 - 24 Months Old Children in Malaka Village, Sumedang District, West Java, Indonesia

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Abstract

Stunting has been one of major concerns for the Indonesian government. In 2013, Indonesia reached the number of 37.2% for national stunting incidence, constantly increasing from year 2010 (35.6%) and 2007 (36.8%) according to Indonesian Basic Health Research (Riset Kesehatan Dasar). The National Nutrition Status Monitoring (Pemantauan Status Gizi) in 2017 recorded that 17.8% children of Indonesia weremalnourished and 12.1% of them were stunted. Whereas data from the Ministry of Health of the Republic of Indonesia in 2017 showed that the prevalence of under-fives nationally with stunting was 29.6%. At provincial level, West Java ranked 26th with 35.3% as the number of stunting incidence. Sumedang District is one of 100 districts assigned as the main focus for stunting intervention program. In addition, Malaka Village in Sumedang District is one of 1,000 villages in Indonesia designed to be intervened as it held a high incident of children suffering from stunting. This study was aimed to find the association between birth length, birth weight, family economic status, exclusive breastfeeding, mother's knowledge with the incidence of stunting in children aged 24-48 months old. The total sample of the study was 62 children, determined through accidental (convenience) sampling. Malaka Village had a prevalence of stunted children at a percentage of 27.4%. From univariate analysis with Spearman's Rho correlation test, birth length and exclusive breastfeeding showed significant associations with stunting while other factors failed to demonstrate such associations.

Keywords: malnutrition, birth length, birth weight, exclusive breastfeeding

Faktor yang Berhubungan dengan Stunting pada Anak Usia 18-24 Bulan di Desa Malaka, Sumedang, Jawa Barat, Indonesia

Abstrak

Stunting merupakan salah satu permasalahan utama bagi pemerintahan Indonesia. Pada tahun 2013, insiden stunting secara nasional di Indonesia mencapai 37.2%. Angka ini meningkat terus-menerus sejak tahun 2010 (35.6%) dan 2007 (36.8%) berdasarkan data dari Riskesdas. Pemantauan Status Gizi tahun 2017 mencatat bahwa 17.8% anak-anak Indonesia mengalami malnutrisi dan 12.1% di antaranya juga mengalami stunting. Data dari Menteri Kesehatan Republik Indonesia pada tahun 2017 menunjukkan bahwa prevalensi balita stunting sebesar 29.6%. Pada tingkat provinsi, Jawa Barat berada pada peringkat 26 dengan insiden stunting sebesar 35.3%. Kabupaten Sumedang merupakan salah satu dari 100 kabupaten yang ditetapkan sebagai focus intervensi stunting. Desa Malaka pada KabupatenSumedang juga merupakan salah satu dari 1,000 desa di Indonesia yang akan dilakukan intervensi sunting karena angka kejadian stunting yang cukup tinggi. Penelitian ini bertujuan untuk mencari hubungan antara panjang badan lahir, berat badan lahir, status ekonomi keluarga, ASI eksklusif, pengetahuan gizi ibu terhadap kejadian stunting pada anakusia 24-48 bulan. Total sampel dari penelitian ini sejumlah 62, yang ditentukan dengan *accidental (convenience) sampling*. Prevalensi anak stunting di Desa Malaka ditemukan sebesar 27.4%. Dengan analisis univariat menggunakan uji korelasi Spearman Rho, didapatkan bahwa panjang badan lahir dan ASI eksklusif memiliki hubungan yang signifikan terhadap kejadian stunting, sedangkan faktor lainnya tidak menunjukkan hubungan yang signifikan.

Kata kunci: malnutrisi, panjang badan lahir, berat badan lahir, ASI eksklusif

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Introduction

Nutritional status is a condition caused by a balance between the number of nutrient intakes and the amount needed by the body for various biological functions including physical growth, development, activity, and health care. An unbalanced nutritional consumption to one's body will cause malnutrition, whether it is excessive or deficient nutritional intakes. Nutritional status of children under five is a reliable indicator of community nutrition and has been developed as an indicator of community health and well-being because, at such age, children are vulnerable to a variety of diseases. The process of rapid growth and development of children under five requires a balanced nutritional intake, thus an unmet need of nutritional intakes will disrupt the process.[1] This explains as one of the most reliable indicators to see the nutritional status of under-fives is growth.

In 2012, 165 million children under 5 years were in stunting condition and 90% were in Africa and Asia.[2] Based on data from UNICEF, an estimated three million children die each year due to malnutrition. In 2016 out of 667 million under-fives throughout the world 159 million under-fives suffered stunting and 50 million under-fives suffered wasting.[3] In the 2013 Indonesian Basic Health Research (Riskesdas) data, the prevalence of national stunting was 37.2%, where there was an increase compared to 2010 (35.6%) and 2007 (36.8%).[4] According to data from the Ministry of Health of the Republic of Indonesia in 2017, the prevalence of under-fives nationally with malnutrition was 17.8%, stunting was 29.6%, and wasting was 9.5%. When compared with the prevalence rate from 2016, stunted under-fives experienced an increase from the previous figure of 27.5%, while the rate of wasted under-fives underwent a decline from the previous figure of 11.1%.[5] Based on the results of the national Nutrition Status Monitoring in 2017, 17.8% of children suffering from malnutrition were found and 12.1% of them were stunted.[6]

Based on national stunting prevalence, from 34 provinces in Indonesia, West Java ranked 26th (35.3%).[4] The stunting prevalence in Sumedang district in 2013 was 41.08% with the number of stunting under-fives at 37,970.[7] Sumedang district was included in the 100 districts that have the highest stunting rate in Indonesia, which has been the focus of the government. Ten villages in the district, including Malaka Village, were additionally included in 1,000 villages, which represent the focus of the stunting intervention program in Indonesia.

Stunting is a condition of failure to thrive in children under five due to chronic malnutrition so children are too short for their age. Malnutrition occurs since in-utero until in the first 2 years after the baby is born, known as the first 1000 days of life (270 days in the womb and 730 days after birth). However, stunting conditions generally can only be seen after the age of two years. Stunting affects the level of intelligence, vulnerability to disease, decreases productivity and then inhibits nation's economic growth which can ultimately increase poverty and inequality. Stunting is caused by many factors related to one another.

As follows, the primary aim of this study was to find factors associated with the incidence of stunting. Factors that influence the occurrence of stunting in 18 - 24 months old children, specifically the mother's knowledge regarding nutrition for children, family economic status, exclusive breastfeeding, birth length, and birth weight were observed in this study.

Materials and Methods

This study used a cross-sectional design to determine factors associated with stunting in children aged 24-48 months. The study

was conducted in November 2018 in Malaka Village, Sumedang District, West Java. To meet the number of samples needed, non-probability sampling method (convinience sampling) was used in the form of accidental sampling. The tools used in this study were questionnaires and anthropometry kit. Inclusion criteria for respondents were those who were willing to participate in the study, had children aged 24-48 months old, and domiciling in Malaka Village, Sumedang Districts, West Java. Whereas the exclusion criteria include those who were not willing to participate in the study, were not aged 24-48 months old, and were not domiciled in Malaka Village, Sumedang.Prior to the initiation of the study, the prospective respondents were asked informed consent.

The type of data used were primary data in the form of interviews and anthropometric measurements. Data were compiled and presented in the form of narration, text, tables, and graphs. Collected data were then analysed through univariate analysis method using IBM SPSS® to determine the association between the dependent and independent variables. The independent variables were characteristics of children (birth length and birth weight), family economic status, exclusive breastfeeding and mother's knowledge of nutrition. Whereas

Table 1. Operational Definition of Key Variables Used in the Study

Variable	Definition	Measurement Tool	Scale	Measurement Results
Age	The life span of a child, calculated since birth date until the time of research.	Questionnaire	Ratio	1 = Under-fives (< 5 years old) 2 = Children (5 – 11 years old)
Stunting	A combination between short-statured and very short-statured under-fives in accordance with the height-for-age graph from WHO	Anthropometry	Nominal	1 = Z-score <-2 SD until ≤ -3 SD (short stature) 2 = Z-score >-3 SD (very short stature)
Economic status	The economic status of a person or family based on monthly income based on regional minimum wages in Sumedang District.	Questionnaire	Ratio	1 = > Rp.2.600.000 2 = Rp.1.000.000 until Rp. 2.600.000 3 = < Rp.1.000.000
Birth length	Birth length in infants is measured in conjunction with the baby's birth weight. < 48 cm = short 48 - 52 cm = normal > 52 cm = tall	Questionnaire	Nominal	1 = Short 2 = Normal 3 = Tall
Birth weight	Body weight in infants measured or weighed within the first 1 hour after birth.	Questionnaire	Ratio	1 = < 2500 gr 2 = > 2500-4000 gr 3 = > 4000 gr
Exclusive breastfeeding	Breast milk given to babies since birth until the age of six months, without adding or replacing with other foods or drinks.	Questionnaire	Nominal	1 = exclusive breastfeeding is not given 2 = exclusive breastfeeding is given
Mother's knowledge regarding nutrition		Questionnaire	Ratio	1 = Insufficient 2 = Sufficient 3 = Good

the dependent variable was the incidence of stunting in children aged 24-48 months old. Stunting was defined as a short or very short stature based on body length or body height with height-for-age z scores less than –2 (see Table 1).

Results

This research was conducted in 62 children aged 24-48 months in Malaka Village.

Table 2. Distribution of Characteristics of the Mothers and Children By the Presence of Stunting

Characteristics	Normal		Stunting	
	f	%	f	%
Children				
Sex				
Male	19	42.22	7	41.18
Female	26	57.78	10	58.82
Birth length				
Short stature (<48 cm)	8	17.78	9	52.94
Normal (48-52 cm)	36	80.00	8	47.06
Tall stature (>52 cm)	1	2.22	0	0
Birth weight				
Low (<2500 g)	2	4.45	2	11.76
Normal (2500-4000 g)	42	93.33	14	82.36
High (>4000 g)	1	2.22	1	5.88
Exclusive breastfeeding				
Given	26	57.78	4	23.53
Not given	19	42.22	13	76.47
Mothers				
Highest education level				
Elementary school	8	17.78	2	11.76
Middle school	17	37.78	11	64.71
High school	13	28.89	4	23.53
Diploma/bachelor	7	15.55	0	0
Family economic status				
Insufficient	9	20.00	2	11.76
Sufficient	18	40.00	5	29.42
Good	18	40.00	10	58.82
Knowledge regarding nutrition				
Insufficient	0	0	1	5.88
Sufficient	2	4.45	1	5.88
Good	43	95.55	15	88.24

Based on the children's characteristics, it is known that in this study there were more stunting girls than boys (58.82 vs. 41.18). More than half (52.94%) stunting children vs. 17.78% non-stunting children had short birth length (Table 2). However, the majority (>75%) children in both groups had normal weight at birth. The number of stunting vs. non-stunting children who were not given exclusive breastfeeding 76.47 vs 42.22%, respectively.

As for the mothers' characteristics, in stunting children, most (64.71%) mothers' highest education was the middle school level, while more than half of the mothers of children without stunting were high school or university graduates (Table 2). Surprisingly, 58% stunting children came from family with good economic status and 88% of their mothers had good knowledge about nutrition.

Table3. Variable Correlation Test Using Spearman's Rho Test

Variable	Correlation coefficient	Significancy
Birth length	.355**	.005
Birth weight	.055	.670
Family economic status	-.167	.193
Eclusive breastfeeding	-.306*	.016
Mother's knowledge regarding nutrition	.138	.286

*. Correlation is significant at the 0,05 level (2-tailed).
**. Correlation is significant at the 0,01 level (2-tailed).

As shown in Table 3, short birth length had positive association (r = 0.355 p = 0.005) while having exclusive breastfeeding had a negative association (r = -0.306 p = 0.016) with the incidence of stunting in children aged 24 - 48 months old in Malaka Village, Sumedang District, West Java, Indonesia. Other factors, namely birth weight, family economic status, and mother's knowledge regarding nutrition did not show significant associations with the incidence of stunting.

Discussion

Based on the characteristics of birth length, more stunting respondents had shorter birth lengths (<48 cm) than those who did not suffer from stunting condition. This is in line with the theory of the Ministry of Health of the Republic of Indonesia published in the Infodatin Situasi Balita Pendek Year 2016, which said that short birth length is one of the risk factors for stunting in children. Short birth length can be caused by various factors, including parents' genetic and lack of nutrition during pregnancy.[8]

While based on birth weight characteristic, stunting was more common in respondents who had normal birth weight (2500-4000 g). This is not in line with the theory of the Indonesian Ministry of Health,which said that the high number of low birth weights(LBW) is estimated to be the cause of the high incidence of stunting in Indonesia. LBW is the most dominant factor causing stunting.[8] Small sample size and non-random sampling methods might explain the discrepancy, that the samples may not be good representation of the Indonesian stunting children.

Based on family economic status, children with good family economic status experience more stunting than those without. Moreover, stunting children in this study was more prevalent from mothers who had good knowledge of nutrition. This is not in accordance with the theory of the Indonesian Ministry of Health, Info dan Situasi Balita Pendek Year 2016, which stated that low family economic status is one of the causes of stunting, which is influenced by several factors, including parents' work, education level of parents, and number of family members. The economic status of the family will affect the ability to fulfill family nutrition and the ability to obtain health services. Children in low economic families are more at risk of stunting because of the inability to fulfill adequate nutrition, increasing the risk of malnutrition.[8] It is important to note that the author assessed the family economic status of family income based on Sumedang District's minimum regional wage for fulfilling daily needs in which each regions are different.

Birth length and exclusive breastfeeding are other factors that can cause stunting. Short birth length in children shows inadequate nutrients consumed by mothers during pregnancy. Exclusive breastfeeding equally carries out an important role in fulfilling infants' nutritional needs. Insufficient nutrition consumption will lead to growth restriction especially during the first 1000 days of life, increasing the risk of stunting.[8] Table 3 shows positive relationship between short birth length and the incidence of stunting: the shorter the birth length, the higher the incidence of stunting. In contrast,

exclusive breastfeeding had a negative association with the incidence of stunting: the more children were given exclusive breastfeeding, the stunting incidence decreased. This is in line with the research conducted by Kusuma[9] which showed short birth length and non-exclusive breastfeeding were risk factors for stunting in infants.

A larger study with random sampling technique is required to obtain more accurate and representative data in Sumedang District. Further research development regarding stunting related factors should be done, including environmental factors, family genetics, maternal reproductive health and gestational history.

Conclusion

The incidence of stunting in children aged 24-48 months old in Malaka Village, Sumedang District, West Java in 2018 was 27.5%. Birth length and exclusive breastfeeding were significantly associated with stunting condition while birth weight, family economic status, and mother's knowledge regarding nutrition did not show significant association.

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Artikel Asli

Identifikasi Telur Cacing Usus dan Kista Protozoa Usus pada Tubuh Lalat dari Warung Makan di Tanjung Duren Timur Jakarta Barat

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Abstrak

Penularan penyakit pada manusia dapat terjadi melalui banyak cara antara lain melalui lalat sebagai vektor mekanik. Lalat dapat membawa bakteri patogen, protozoa, larva serta telur cacing yang menempel pada tubuhnya dan dapat mencemari bahan makanan. Tujuan penelitian ini untuk mengidentifikasi telur cacing usus dan protozoa usus pada tubuh lalat di warung makan kelurahan Tanjung Duren Timur, Jakarta Barat. Desain penelitian yang digunakan dalam penelitian ini adalah studi deskriptif dengan pendekatan poton lintang. Penelitian dilakukan di Tanjung Duren Timur, Jakarta Barat. Hasilnya, 38 warung tempat penangkapan lalat seluruhnya positif mengandung parasit. Parasit yang ditemukan adalah telur cacing tambang, *Ascaris lumbricoides* dan kista *Entamoeba histolytica* pada 17 (44,8%) warung makan, kista *Giardia lamblia* pada 12 (31,6%) warung makan, dan kista *Entamoeba coli* pada 32 (84,2%) warung makan.

Kata kunci: telur cacing usus, protozoa usus, vektor mekanik, lalat.

Identification of Eggs of Intestinal Worms and Intestinal Protozoa Cysts Attached to the Bodies of the Flies at Street Food Stalls in Tanjung Duren Timur, West Jakarta

Abstract

Transmission of diseases can occur in many ways, including through flies as mechanical vectors. Flies can carry pathogenic bacteria, protozoa, larvae and worm eggs that attached to their bodies and can contaminate food. The purpose of this cross-sectional study was to identify eggs of the intestinal worm and intestinal protozoa attached to the body of flies at food stalls in Tanjung Duren Timur, West Jakarta. As a result, all (n=38) stalls where flies were caught were entirely positive for parasites. Hookworm eggs, *Ascaris lumbricoides* and *Entamoeba histolytica* cysts were found in 17 (44.8%) food stalls; *Giardia lamblia* cysts in 12 (31.6%); and *Entamoeba coli* cysts in 32 (84.2%).

Keywords: *eggs of intestinal worms, intestinal protozoa, mechanical vectors, flies.*

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Pendahuluan

Penularan dan penyebaran penyakit pada manusia bisa terjadi melalui berbagai cara, salah satunya yaitu penyebaran melalui vektor.[1] Lalat yang merupakan vektor mekanik sering di jumpai dalam keseharian kita dan pada hampir semua jenis lingkungan. Lalat dapat membawa bakteri patogen, protozoa, larva serta telur cacing yang menempel pada tubuhnya,[2] contohnya telur cacing (*Oxyrus vermicularis, Trichuris trichiura,* cacing tambang*,* dan *Ascaris lumbricoides),* selain itu kista protozoa *(Entamoeba hystolitica* dan *Giardia lamblia)*.[3,4] Lalat dapat menularkan penyakit melalui bahan makanan atau minuman yang terkontaminasi oleh bibit penyakit yang menempel pada tubuh, kaki, tarsi, dan probosis, bulu badan, bulu anggota gerak, muntahan serta tinjanya.[5] Lalat tersebar di berbagai belahan dunia, dan kebanyakan ditemukan di sekitar kehidupan manusia, antara lain *Musca domestica*, dan lalat hijau.[1]

Di Jakarta Barat, warung makan merupakan tempat yang banyak dikunjungi masyarakat setiap hari. Masyarakat lebih memilih membeli makanan siap saji yang dinilai lebih praktis daripada mengolahnya sendiri, namun tidak semua warung makan memperhatikan kebersihan yang antara lain terlihat dari keberadaan lalat di warungnya.

Penelitian ini bertujuan untuk mengidentifikasi parasit usus (cacing dan protozoa) yang terdapat pada tubuh lalat di sekitar warung makan tersebut.

Bahan dan Cara Kerja

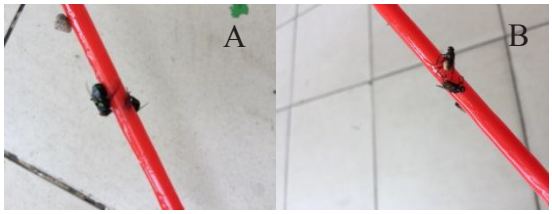
Penelitian ini merupakan penelitian deskriptif-potong lintang. Lalat yang diperiksa berasal dari warung makan di wilayah studi dan berdasarkan rumus Slovin,[6] jumlah warung makan yang diperlukan adalah 38 (CI 95%).

Cara Pengumpulan Sampel

Lalat dikumpulkan dengan cara membagikan perangkap lalat ke warung makan dikelurahan Tanjung Duren Timur Jakarta Barat. Perangkap dibuat dari sedotan yang diolesi lem lalat. Pada hari ke kedua semua lalat yang didapat dari warung makan dikumpulkan untuk diperiksa dilaboratorium. Selanjutnya dari setiap warung diambil 10 ekor lalat per warung makan secara acak. Lalat yang masih hidup dan menempel di sedotan, dilepaskan satu persatu dengan pinset lalu dimasukan ke dalam gelas plastik dan dimatikan dengan pemberian kloroform dalam kapas. Kemudian lalat yang sudah mati dimasukan ke dalam tabung yang telah diberi kode, dan diisi dengan larutan formalin 10% sebanyak 10ml, dan selanjutnya diaduk dengan lidi selama 10 menit untuk fiksasi. Kemudian ditambah larutan eter sebanyak 3 ml, ditutup dengan prop karet, dikocok selama 30 detik, dan didiamkan selama kurang lebih 1 jam. Setelah kotoran yang melekat pada lalat terlepas, tabung berisis lalat disentrifugasi dengan kecepatan 3000 rpm selama 5 menit. Selanjutnya supernatan dibuang dan endapan diperiksa dengan membuat sediaan basah eosin yang diperiksa di bawah mikroskop dengan pembesaran 10x40, dilanjutkan dengan pembesaran 10x45. Telur cacing dan kista protozoa usus yang ditemukan pada tiap sediaan diidentifikasi secara morfologis.

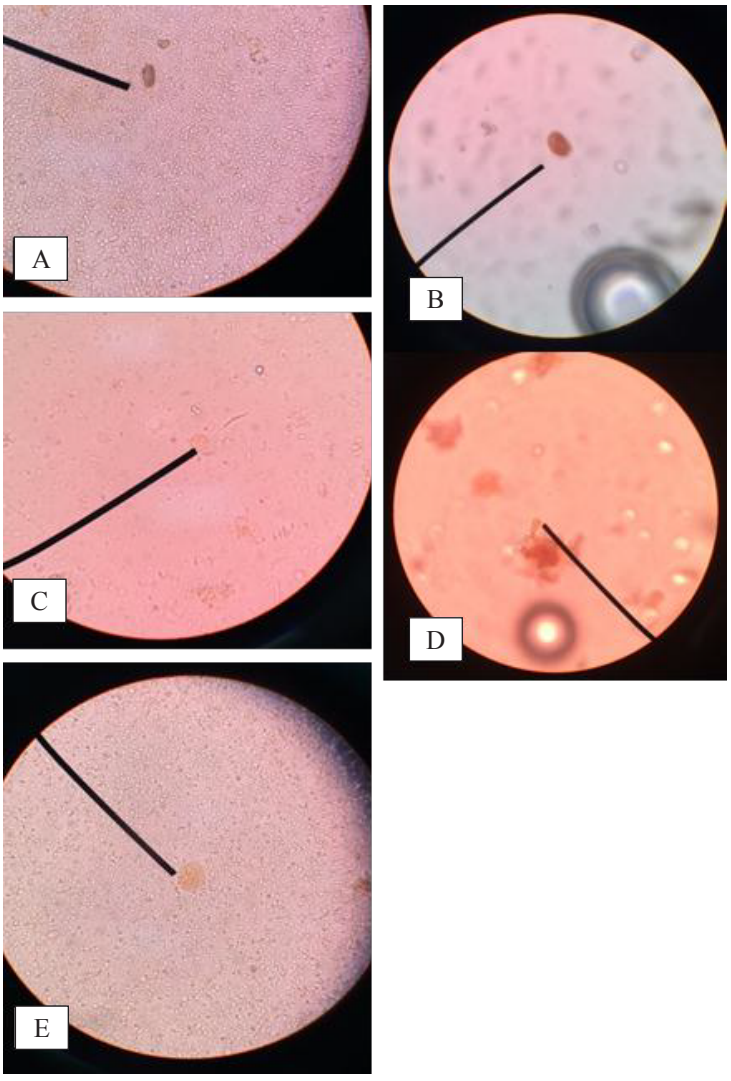
Hasil

Pada penelitian ini telah dilakukan pemeriksaan terhadap keberadaan parasit usus pada tubuh lalat yang dikumpulkan dari 38 warung makan di wilayah Tanjung Duren Timur Jakarta Barat. Dari 38 warung makan tersebut, sebanyak 380 lalat ditangkap untuk diteliti lebih lanjut. Lalat yang ditangkap terdiri atas *Musca domestica* dan *Chrisomya* sp. (Gambar 1).



Gambar 1. Lalat yang ditangkap menggunakan sedotan yang dilapisi lem di permukannya. *Chrysomya* sp.(A) dan *Musca domestica* (B).

Seluruh 38 warung (100%) yang diperiksa, lalatnya positif mengandung parasit usus yakni cacing dan protozoa. Cacing usus yang diidentifikasi adalah *Ascaris lumbricoide,* ditemukan pada lima (13%) warung makan, dan cacing tambang pada enam (15,7%) warung makan. Sementara itu protozoa usus yang ditemukan adalah kista *Entamoeba histolytica* pada 17 warung makan (44,7%), kista *Entamoeba coli* 32 warung makan (84%) dan *Giardia lamblia* pada 12 warung makan atau 31,5%. Tidak ditemukan lalat yang mengandung lebih dari satu jenis telur cacing, sementara pada golongan protozoa ditemukan lalat yang mengandung lebih dari satu jenis protozoa. yakni kista *E. histolytica, E. coli* dan *G. lamblia*.



Gambar 2. Sediaan eosin, pembesaran 100×, telur cacing tambang (A), telur *A. lumbricoides* tidak dibuahi (B); pembesaran 450 ×, kista *E. histolytic* (C), kista *G. lamblia* (D) kista *E. coli* (E)

Diskusi

Lalat yang ditangkap pada penelitian ini diidentifikasi sebagai *M. domestica* dan *Chrysomya. Musca domestica* adalah lalat rumah yang mudah ditemukan dimana-mana terutama di sekitar tempat tinggal manusia.[7] Demikian pula *Chrysomya*, yang biasa disebut sebagai *old world blow fly* dan mempunyai banyak genus, mudah ditemukan di sekitar tempat tinggal manusia. Keduanya dapat bertindak sebagai vektor mekanik beberapa patogen terhadap manusia termasuk parasit usus seperti cacing dan protozoa.[8] Warung makan yang diteliti berada di daerah pemukiman manusia yang padat, dengan sanitasi lingkungan buruk sebagaimana terlihat pada saluran pembuangan air kotor yang terbuka, dan airnya tergenang. Selain itu sebagian warung makan tersebut terletak dekat pasar tradisional yang berjualan daging, ikan, dan sayur-sayuran, serta dekat dengan jamban dan tempat sampah. Dapur tempat mengolah makanan kurang ventilasi udara serta cahaya yang memungkinkan pathogen berkembang biak dan kemudian terbawa oleh lalat sebagai vektor mekanik. Kondisi lingkungan seperti itu mengundang dan mendukung lalat untuk datang dan berkembang biak.

Chrysomya sp. mempunyai kebiasan berkumpul dan berkerumun di sekitar makanan, sampah, limbah yang membusuk, bangkai dan tinja, sehingga lalat tersebut dikenal sebagai serangga yang kehadirannya dianggap identik dengan kondisi kotor, jorok, dan tidak sehat. Sementara itu, *M. domestica* berkembang biak pada semua jenis tinja, seperti tinja kucing, anjing liar dan terutama tinja ayam. Selain itu, lalat juga dapat berkembang biak pada sisa makanan ternak dan berbagai bahan organik yang membusuk.[9,10]

Musca domestica dan *Chrysomya* sp., merupakan vektor mekanik yang memiliki kebiasaan berpindah. Kedua lalat tersebut dapat hinggap pada sampah/kotoran dan kemudian berpindah ke berbagai jenis bahan makanan sehingga dapat mencemari makanan dengan organisme patogen yang melekat di tubuhnya, seperti protozoa, cacing, bakteri, dan virus.[11-13] Lalat *M. domestica* dapat membawa telur cacing dan protozoa *(O. vermicularis*, *T. trichiura, G. lambia, E. histolytica* dan *B. coli)* di badanya. Selain sebagai vektor mekanik, *Chrysomya* sp. dapat menyebabkan *myasis* atau investasi larvanya pada mata, tulang dan organ lain melalui luka. Selain menjadi vektor parasit, lalat juga dapat menjadi vektor mekanik bagi patogen lain seperti bakteri, dan virus. bakteri usus (*Salmonella, Shigella* dan *Eschericia coli*).[11]

Dari kedua jenis lalat yang ditangkap di warung makan di wilayah Tanjung Duren Timur, didapat dua jenis telur nematoda usus, yaitu cacing tambang dan *A. lumbricoides*. Selain itu juga ditemukan kista protozoa usus seperti *E. histolytica, G. lamblia,* dan *E. coli.*

Tingginya prevalensi lalat yang positif parasit (38/38 warung makan) mengindikasikan rendahnya kebersihan di warung yang diteliti.

Cacing usus *A. lumbricoides* dan cacing tambang termasuk dalam golongan cacing yang ditularkan melalui tanah atau cacing berkembang menjadi infektif di tanah. *Ascaris lumbricoides* adalah parasit obligat pada manusia dan tidak dapat menginfeksi pejamu lain. Sehingga berdasarkan temuan ini, diduga di sekitar warung yang diteliti terdapat pencemaran tanah oleh tinja manusia. Selanjutnya, telur *A. lumbricoides* sulit dibedakan dengan telur *Ascaris suum* yang merupakan parasit pada babi. Sehingga perlu dicari keberadan peternakan babi disekitar tempat tersebut untuk memastikan bahwa telur cacing yang ditemukan memang telur *A. lumbricodes* dan bukan telur *A. suum*.[14]

Entamoeba histolytica dikenal sebagai protozoa penyebab kematian terbanyak setelah malaria. Protozoa tersebut mampu mnyebabkan kerusakan pada dinding usus dan menginfeksi jaringan ekstra intestinal lain seperti hati dan otak. Disamping amuba patogen *E. histolytica,* di dalam usus juga didapati amuba apatogen yakni *E dispar* yang secara morfologis sulit dibedakan dengan *E. histolytica*, namun memilki sifat-sifat biologis yang berbeda.[15] Pada penelitian dengan menggunakan *nested* PCR untuk membedakan *E. histolytica,* dengan dua ameba apatogen *E. dispar* dan *E moshkovskii*, hasilnya menunjukkan bahwa ketiga parasit tersebut dapat dapat ditemukan bersamaan di lumen usus manusia, sehingga identifikasi kista secara morfologis saja tidak cukup untuk membedakan ketiga parasit tersebut.[16]

Keterbatasan penelitian ini terletak pada cara identifikasi parasit secara morfologis yang tidak dapat membedakan parasit usus yang secara morfologis mirip. Diperlukan upaya lain misalnya identifikasi berbasis molekular untuk membedakan parasit tersebut. Di luar keterbatasan penelitian, hasil penelitian ini menunjukkan bahwa kondisi kebersihan berbagai warung di wilayah penelitian memerlukan perhatian serius, karena warung menyediakan makanan yang murah dan terjangkau. Keberadaan parasit pada lalat yang ditangkap di warung yang diteliti mengingatkan kita bahwa lalat merupakan vektor mekanik yang menularkan berbagai penyakit infeksi yang dapat bersifat letal atau mematikan.

Kesimpulan

Dari hasil penelitian ini, ditemukan telur cacing tambang, telur *A. lumbricoides*, dan kista *E. histolytica,* kista *E. coli* serta kista *G. lamblia* pada tubuh lalat yang ditangkap pada warung makan di wilayah Tanjung Duren Timur, Jakarta Barat.

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Laporan Kasus

Sindrom Wellens Tipe A

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Abstrak

Sindrom Wellens pertama kali ditemukan tahun 1980 oleh de Zwaan dan Wellens. Kriteria diagnostik yang penting adalah perubahan karakteristik gelombang T, riwayat nyeri dada, enzim jantung meningkat sedikit atau normal, EKG tanpa gelombang Q, tanpa peningkatan peningkatan ST elevasi signifikan dan progresi gelombang R yang normal. Sindroma Wellens merupakan salah satu indikasi bahwa ada sumbatan kritikal arteri koroner terutama LAD yang lebih dari 50%. Tulisan ini akan melaporkan kasus seorang laki laki berusia 57 tahun dengan keluhan nyeri dada tipikal angina yang telah berlangsung selama tiga hari. Pasien dirujuk ke RS POLRI untuk *primary percutaneous coronary intervention* (PCI), ditemukan stenosis 90% di bagian proksimal *left anterior descending artery* (LAD) dan dilakukan pemasangan stent dengan hasil baik dan stent paten.

Kata Kunci : Wellens, LAD, PCI

Wellens Syndrome Type A

Abstract

Wellens syndrome was first discovered in 1980 by de Zwaan and Wellens. The diagnostic criteria for Wellens syndrome are changes in the characteristics of T waves, a history of chest pain, a slight or normal increase in cardiac enzymes, and ECG without Q waves, without a significant increase in ST segment elevation and normal R wave progression. Wellens syndrome indicates that there is a critical blockage of coronary arteries, especially *left anterior descending artery* (LAD) that is more than 50%. This paper will report a case of a 57-year-old man with typical chest pain with three days onset of chest pain. The patient was referred to POLRI Hospital for primary percutaneous coronary intervention (PCI), and 90% stenosis was found in the mid proximal of left anterior descending artery (LAD) and stenting was performed, with good results and patent stents.

Keywords: Wellens, LAD, PCI

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Pendahuluan

Setiap tahun secara global penyebab kematian nomor satu adalah penyakit kardiovaskular. Penyakit kardiovaskular adalah penyakit yang disebabkan gangguan fungsi jantung dan pembuluh darah, seperti: penyakit jantung koroner, penyakit gagal jantung atau payah jantung, hipertensi dan stroke.[1]

Di Indonesia tahun 2013 prevalensi penyakit jantung koroner sebesar 0,5% atau diperkirakan sekitar 883 447 orang, sedangkan berdasarkan diagnosis dokter/ gejala sebesar 1,5% atau diperkirakan sekitar 2 650 340 orang.[1] Penyakit jantung koroner, pada praktik dokter sehari hari sering menimbulkan masalah dalam hal penegakan diagnosis, utamanya penegakan diagnosis berdasarkan rekaman aktivitas listrik jantung atau elektrokardiografi (EKG). Kesulitan menegakan diagnosis berdasarkan EKG melibatkan banyak faktor, diantaranya adalah banyaknya kriteria gambaran EKG yang harus dipenuhi untuk menegakan diagnosis sindrom koroner akut (SKA).[2] Interpretasi EKG dalam diagnosis sindrom koroner akut sangat penting di instalasi gawat darurat untuk mengevaluasi pasien dengan nyeri dada akut. Kesulitan menegakan diagnosis jenis serangan jantung terutama berdasarkan EKG, menimbulkan masalah tersendiri dalam praktik sehari hari.[2]

Sindrom Wellens, merupakan tanda terjadinya SKA dengan gambaran EKG yang berbeda dari kriteria EKG untuk serangan jantung biasa. Serangan Jantung hanya diklasifikasikan berdasarkan peningkatan segmen ST, peningkatan enzim jantung (cardiac biomarkers), *ST segment elevation myocardial infarct* (STEMI) atau *non segment elevation myocardial infarct* (NSTEMI).[2] Gambaran EKG pada sindrom Wellens adalah absensi gelombang Q, progresi gelombang R, dan perubahan karakteristik gelombang T (bifasik / inversi).[2] Pola gambaran EKG yang berbeda pada sindrom Wellens, menunjukkan penyakit iskemik miokard yang disebabkan oleh stenosis anterior proksimal (LAD) yang kritis atau hampir total. Hal itu seringkali diabaikan karena tidak ada peningkatan segmen ST seperti pada STEMI umumnya.[2]

Menurut penelitian Wellens,[2] sebanyak 75%-100% pasien yang memiliki karakteristik EKG seperti ini memiliki stenosis LAD yang signifikan dan berpotensi berkembang menjadi infark dinding anterior yang luas dan lebih lanjut dalam beberapa minggu. Ada dua pola EKG dalam sindrom Wellens. Tipe A ditandai dengan inversi gelombang T yang sangat simetris dalam sadapan V2 dan V3, sering termasuk sadapan V1 dan V4 dan kadang-kadang menyebabkan perubahan pada V5 dan V6. Sementara tipe B ditandai oleh gelombang-T bifasik pada sadapan V2 dan V3. Nilai prediksi positif dari tanda sindrom Wellens adalah sekitar 86% pada setiap kasus.[3]

Oleh karena itu, amat penting untuk mempertimbangkan angiogram koroner sebagai modalitas diagnostik awal dibandingkan pemeriksaan konservatif lainnya pada pasien dengan pola EKG yang menunjukkan kemungkinan sindrom Wellens.[1]

Pada tulisan ini dilaporkan kejadian sindrom Wellens pada seorang laki-laki dewasa yang semula tampak sehat.

Laporan Kasus

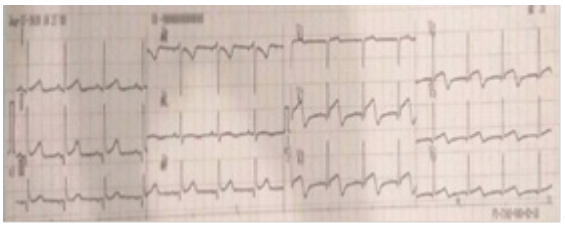
Seorang laki – laki usia 57 tahun datang ke IGD RSU UKI dengan keluhan nyeri dada kiri sejak 40 menit sebelumnya, merasa seperti ditimpa beban berat terus – menerus, nyeri yang makin berat dan menjalar menembus ke punggung serta disertai keringat dingin. Gejala tersebut timbul saat istirahat dan tidak hilang dengan isosorbide dinitrate (ISDN) tablet 5mg yang diberikan ublingual. Pasien tidak mengeluh sesak,

mual, maupun muntah. Tiga hari sebelumnya pasien mengeluh nyeri dada dengan pola yang sama, namun tidak langsung ke IGD karena dirasakan tidak terlalu berat. Latar belakang pasien; pasien memiliki riwayat hipertensi dan seorang perokok aktif.

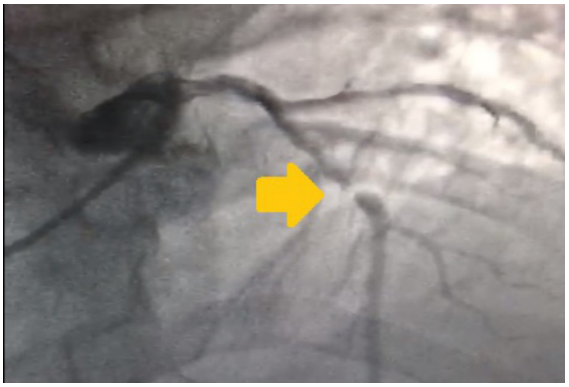
Pada pemeriksaan fisik didapatkan keadaan umum tampak sakit sedang, kesadaran kompos mentis, dan pasien tampak kesakitan karena nyeri dada dengan *visual analog score* (VAS) sekitar 6. Tekanan darah 140/100 mmHg, frekuensi nadi 100 kali/menit reguler, frekuensi nafas 24 kali/menit. *Jugular vein pressure* (JVP) tidak distensi. Pada pemeriksaan paru tidak didapatkan ronki maupun wheezing. Pemeriksaan jantung tidak terdapat murmur maupun gallop, dan tidak ditemukan edema tungkai pada kedua ekstremitas bawah.

Dilakukan pemeriksaan EKG untuk konfirmasi, dan didapatkan gambaran elevasi ST segmen dengan gambaran Wellens Tipe A di sadapan V2, V3, V4, V5, V6 dan tidak disertai gambaran resiprokal serta gelombang T bifasik. Pemeriksaan dilanjutkan dengan pemeriksaan enzim jantung yaitu Troponin I dan didapatkan hasil kualitatif positif (+). Pasien kemudian didiagnosis sebagai STEMI anterior onset 72 jam Killip I TIMI 2. Kemudian, dilakukan pemeriksaan lanjut dengan ekhokardiografi dan didapatkan fungsi sistolik ventrikel menurun, LVEF 50% dan pergerakan dinding jantung hipokinetik apeks ventrikel kiri (VK). Pasien kemudian direncanakan dirujuk ke RS POLRI untuk dilakukan terapi reperfusi koroner dengan *percutaneus coronary intervention* (PCI). Saat tindakan PCI dilakukan, ditemukan stenosis 90% di bagian tengah proksimal arteri LAD, dan dilakukan pemasangan stent dengan hasil baik, dan paten.

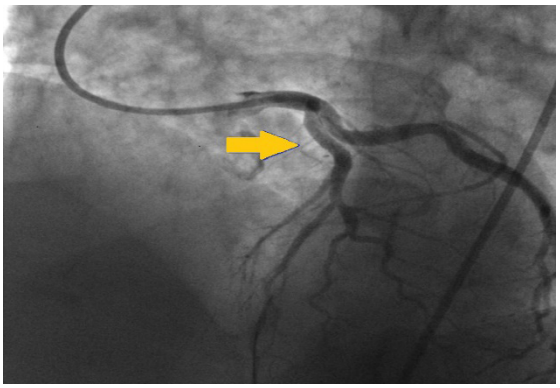
Pasien kemudian dievaluasi selama 24 jam di ICCU RS POLRI dan tidak didapatkan perburukan maupun adanya serangan sindrom koroner akut berulang pada pasien, sehingga keesokan harinya pasien sudah diperbolehkan untuk pulang dari RS POLRI.



Gambar 1. Hasil EKG pasien dengan Sindroma Wellen Tipe A, tampak ST elevasi tidak signifikan dan gelombang T bifasik sadapan V2-V6



Gambar 2. Gambaran ekokardiografi saat dilakukan intervensi koroner perkutaneus. Terlihat sumbatan pada arteri desendens anterior kiri sebesar 90%



Gambar 3. Gambaran ekokardiografi pasien sindroma wellen tipe A saat dilakukan intervensi koroner kerkutaneus dan setelah pemasangan ring jantung. Gambaran sumbatan pada LAD menghilang.

Diskusi

Infark miokard, yang merupakan bagian SKA yang terjadi akibat ketidakseimbangan antara pasokan oksigen dan substrat dengan kebutuhan oksigen di miokard,

yang kemudian akan mengarah ke iskemia dan akhirnya kematian sel otot jantung.[4] Sindrome koroner akut dapat sangat merugikan dan mengancam jiwa jika tidak terdiagnosis dan ditangani dengan strategi terapi yang tepat.[5]

Prognosis pasien SKA tergantung pada pengenalan gejala awal dan petunjuk samar pada EKG yang mengarahkan dokter untuk menggolongkan pasien ke dalam kategori berisiko rendah, risiko menengah, atau berisiko tinggi sehingga dapat ditentukan strategi penanganan selanjutnya.[5]

Inversi gelombang T pada sadapan prekordial bagian anterior memiliki banyak variasi bentuk, yang dapat dipengaruhi oleh berbagai penyebab, dan kadang kala merupakan varian normal dalam pola gelombang T remaja yang persisten.[5] Dalam praktik sehari hari, kadang kala dapat menjadi sangat sulit untuk menegakkan diagnosis SKA terlebih bila keluhan yang dikeluhkan pasien nampak tidak khas atau sesuai dengan gejala SKA pada umumnya. Pola variasi EKG yang beragam, menjadi tantangan tersendiri bagi klinisi untuk menegakan diagnosa didukung dengan data klinis yang kadang kurang lengkap.[6]

Pada kasus ini, pasien datang dengan keluhan yang sudah dirasakannya selama tiga hari, dan datang dalam kondisi yang tampak sakit sedang, dan nyeri dada yang menurut pengakuan pasien, masih dapat ditahan. Tentunya gejala yang timbul pada pasien ini tidak sesuai dengan gejala STE-ACS pada umumnya, sehingga apabila tidak dikonfirmasi dengan pemeriksaan EKG, kemungkinan pasien ini hanya akan didiagnosis dengan angina pektoris tidak stabil, atau Non STE-SKA/NSTEMI.[6]

Setelah dilakukan pemeriksaan konfirmasi dengan EKG, kembali didapatkan gambaran EKG yang tidak spesifik yang umumnya dijumpai pada NSTEMI maupun STEMI, yaitu ditemukan adanya pola gelombang T bifasik pada sadapan prekordial, di sadapan V2, V3, V4,V5 dan V6 atau sadapan anterolateral yang diperdarahi oleh pembuluh darah LAD.[6]

Pola gelombang T bifasik ini, sesuai dengan teori yang dikemukakan pertama kali oleh de Zwaan dan Wellens.[5,6] Bila ditemukan pola seperti itu pada sadapan prekordial, kemungkinan besar, pasien tersebut mengalami oklusi hampir total pada LAD. Hal itu sesuai dengan teori mengenai sindrom wellens yang nilai prediksi positifnya sekitar 86% untuk setiap kasus.[2,3]

Dilakukan ekokardiografi untuk mempelajari struktur jantung pasien dan segmental analisis pergerakan dinding jantung untuk memeriksa bagian mana yang mengalami hipokinetik akibat serangan iskemia. Ditemukan hasil LVEF menurun 50% dan hipokinetik apeks ventrikal kiri (VK), yaitu daerah yang diperdarahi oleh LAD.[6,7]

Pasien dirujuk untuk dilakukan reperfusi koroner dengan PCI sebagai pilihan terapi, karena kasus ini dianggap sebagai STEMI. Didapatkan hasil yang sesuai dengan teori yaitu ditemukan sumbatan pada *mid proximal* LAD sekitar 90% dan dilakukan pemasangan stent. Setelah satu hari pasca PCI, pasien dievaluasi kembali dan tidak ditemukan gejala komplikasi, kemudian pasien berobat jalan.[8]

Kesimpulan

Sidroma Wellen, merupakan pertanda terjadinya infark miokard akut yang sedang berlangsung, yang diakibatkan oleh oklusi total atau hampir total pada arteri koroner khususnya LAD. Apabila sindrom ini tidak dikenali dan dianggap sebagai angina pektoris tidak stabil atau NSTE-ACS, dapat berakibat fatal akibat keterlambatan reperfusi. Pada kasus ini, Wellens syndrome dapat dikenali dan telah dilakukan terapi reperfusi dan pemasangan stent sesuai indikasi dengan hasil baik.

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Obesitas pada Anak: Ada Kaitan dengan Asupan Air?

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Abstrak

Obesitas merupakan masalah kompleks pada anak dan telah menjadi masalah kesehatan masyarakat di dunia terlebih pada negara maju dan berkembang.Penyebab obesitas bersifat multifaktorial, namun penyebab dasarnya adalah ketidak seimbangan antara kalori yang dikonsumsi dan yang digunakan atau dikeluarkan.Salah satu penyebab obesitas adalah asupan kalori yang berlebih yang diperoleh dari makanan atau minuman seperti minuman mengandung gula. Penambahan gula dan zat pewarna pada air minum merupakan upaya meningkatkan asupan air, karena air mempunyaibanyak fungsi dalam tubuh makhluk hidup yang berperan menjaga kesehatan dan hidupJika asupan air berkurang, dapat terjadi kurang air tubuh atau dehidrasi. Beberapa penelitian melaporkan volume air yang dikonsumsi sebagian masyarakat berada di bawah standar yang dianjurkan, dan lebih dari 20% anak dan remaja mengonsumsi air di bawah standar.Salah satu upaya meningkatkan konsumsi air per hari adalah menyediakan minuman dalam kemasan dengan memberi warna dan rasa seperti menambahkan gula.Penambahan gula atau kalori ke dalam minuman berperan terhadap kejadian obesitas terutama pada anak. Berbagai upaya dilakukan untuk tata laksana obesitas, namun konsumsi air putih sebagai salah satu faktor yang dapat dipertimbangkan dalam tata laksana obesitas belum banyak diperbincangkan. Dalam kepustakaan disebutkan bahwa minum air putih dapat menurunkan berat badan karena air putih tidak mengandung kalori sehingga asupan kalori total berkurangdan meningkatnya oksidasi lemak melalui peran insulin karena minum air non kalori tidak menstimulasi insulin.

Kata kunci: air, anak, minuman, obesitas

Obesity in Children : Is It Related to Water Intake?

Abstract

Obesity is a complex problem in children and has become a worldwide health problem especially in developing and developed countries. Although obesity has multifactorial etiologies, its basic etiology is imbalance between caloric intake and expenditure. One cause of obesity is excessive caloric intake from food or glucose containing beverages. The addition of glucose and food coloring in drinking water is an attempt to increase water intake since water has many functions in living organisms and plays a key role in maintaining health and life. Decreased water intake may cause dehydration. Studies reported that the volume of water consumed by a portion of people was below the recommended standard and more than 20% of children and adolescents had substandard water consumption. An attempt to increase daily water consumption is to provide packed-beverages with the addition of color, flavour and glucose. The addition of glucose or calorie in beverages contributes to increased incidence of obesity in children. Several attempts are addressed to manage obesity; however, clear-drinking water consumption as a considerable factor in obesity management still has been not much of a discussion. Literatures stated that consuming clear-drinking water could decrease body weight because drinking water didn't contain calorie and therefore led to decreased total caloric intake and increased insulin-related fat oxidation since consumption of non-caloric water didn't stimulate insulin secretion.

Keywords : water, children, beverages, obesity

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Pendahuluan

Obesitas merupakan masalah kompleks pada anak dan telah menjadi masalah kesehatan masyarakat di dunia terlebih pada negara maju dan berkembang,[1] termasuk di Indonesia yang mengalami peningkatan dari tahun ketahun. Berdasarkan laporan WHO, pada tahun 2010 didapatkan sekitar 43 juta anak dengan *overweight* dan obesitas. Di berbagai negara dilaporkan peningkatan prevalensi obesitas pada anak. Di Amerika Serikat, obesitas meningkat dari 5% pada tahun 1960an menjadi 19% pada tahun 1990an.[2] Di Turki, obesitas pada anak sebesar 26,3%,[3] dan di Afrika Selatan obesitas pada anak 13-17 tahun sebesar 20,4%.[4]

Di Indonesia, berdasarkan data Riset Kesehatan Dasar (Riskesdas), pada tahun 2010, kejadian obesitas pada anak 13-15 tahun sebesar 2,5%, dan pada tahun 2013, obesitas sebesar 10,8%.[5]

Masalah Obesitas pada Anak

Obesitas pada anak mempunyai konsekuensi jangka panjang, oleh karena itu deteksi dan penanganan terhadap obesitas perlu dilakukan secara dini dan komprehensif pada semua jenjang pendidikan dan melibatkan semua pihak terkait seperti keluarga, guru, lembaga pendidikan, masyarakat dan pusat pelayanan kesehatan.[6] Konsekuensi jangka panjang akibat obesitas pada anak dipengaruhi oleh faktor genetik, epigenetik, perilaku, faktor lingkungan dan perilaku dapat dimodifikasi sejak masa kanak-kanak sehingga menjadi fokus intervensi klinis.[7]

Obesitas pada anak berisiko tinggi menjadi obesitas pada masa dewasa. Meningkatnya prevalensi obesitas menyebabkan meningkatnya prevalensi penyakit terkait obesitas seperti diabetes melitus tipe 2, *obstructive sleep apneu,* penyakit kardiovaskular, dan hipertensi.[8] Selain itu obesitas berperan pada gangguan ginjal, baik secara langsung maupun tidak langsung.

Penyebab obesitas bersifat multifaktorial, namun penyebab dasarnya adalah ketidak seimbangan antara kalori yang dikonsumsi dan yang digunakan atau dikeluarkan.Salah satu penyebabnya adalah asupan kalori yang berlebih yang diperoleh dari makanan atau minuman.Minuman yang berperan terhadap kejadian obesitas adalah minuman mengandung gula atau berkalori atau *sugar-sweetened beverages*.

Meskipun tujuan utama penanganan obesitas adalah preventif, namun sekarang sudah mulai bergeser ke identifikasi dan pengobatan komplikasi obesitas.[6] Berbagai upaya dilakukan untuk menanggulangi obesitas, namun peran konsumsi air putih belum banyak diperbincangkan.

Kebutuhan Air

Air mempunyai banyak fungsi dalam tubuh makhluk hidup, dan berperan penting dalam menjaga kesehatan dan hidup. Air tubuh tidak hanya penting untuk pencernaan, penyerapan makanan, metabolisme, pengeluaran zat sisa metabolisme, tetapi juga untuk mengatur suhu dan mempertahankan bentuk dan stuktur sel, jaringandanorgan tubuh. Salah satu masalah yang berkaitan dengan kebutuhan air adalah kurang air tubuh atau dehidrasi yang dapat mengancam jiwa.[9] Kehilangan air tubuh sebesar 1-2% berat badan telah menimbulkan dampak pada kesehatan berupa gangguan fungsi kognitif, kesadaran, atau kapasitas latihan fisik.[10] Untuk mempertahankan dan memelihara keseimbangan cairan tubuh, direkomendasikan asupan air dengan jumlah tertentu yang berbeda-beda untuk setiap individu dan daerah atau negara. Asupan total air sehari-hari diperoleh dari makanan, air minum, dan berbagai jenis minuman.[9]

Kebutuhan air bervariasi pada setiap individu tergantung pada umur, berat badan, lingkungan, aktivitas. Pada tabel berikut ini, terlihat kebutuhan air yang tidak sama. Asupan air adekuat bervariasi berdasarkan umur, mulai dari 0,7 L air total pada bayi hingga 3,7 Liter pada laki-laki dewasa dan 2,7 Liter pada perempuan dewasa.

Tabel 1. *Dietary Reference Intake* (Asupan Adekuat) untuk Air

Usia	Laki-laki			Perempuan		
	Air total	Dari minuman	Dari makanan	Air total	Dari minuman	Dari makanan
1-6 bulan	0,7	0,7	0,0	0,7	0,7	0,0
7-12 bulan	0,8	0,6	0,2	0,8	0,6	0,2
1-3 tahun	1,3	0,9	0,4	1,3	0,9	0,4
4-8 tahun	1,7	1,2	0,5	1,7	1,2	0,5
9 -13 tahun	2,4	1,8	0,6	2,1	1,6	0,5
14-18 tahun	3,3	2,6	0,7	2,3	1,8	0,5
≥19 tahun	3,7	3,0	0,7	2,7	2,2	0,5
Ibu hamil				3,0	2,3	0,7
Ibu menyusui				3,8	3,1	0,7

Keterangan: kebutuhan Liter per hari. Data diperoleh dati *Institute of Medicine*[10]

Tabel 2.Angka Kecukupan Air Untuk Orang Indonesia (Berdasarkan Permenkes RI nomor 75 tahun 2013)[11]

Kelompok umur	**Angka kecukupan air (mL/hari)**
Bayi	
0-6 bulan	Air susu ibu
7-12 bulan	800 mL
Anak	
1-3 tahun	1.200
4-6 tahun	1.500
7-9 tahun	1.900
Pria	
10-12 tahun	1.800
13-15 tahun	2.000
16-18 tahun	2.200
19-29 tahun	2.500
30-49 tahun	2.600
50-64 tahun	2.600
65-80 tahun	1.900
80+ tahun	1.600
Wanita	
10-12 tahun	1.800
13-15 tahun	2.000
16-18 tahun	2.100
19-29 tahun	2.300
30-49 tahun	2.300
50-64 tahun	2.300
65-80 tahun	1.600
80+ tahun	1.500

Berdasarkan konsensus kebutuhan air pada anak sehat oleh Ikatan Dokter Anak Indonesia, kebutuhan air pada anak ditentukan berdasarkan 1).usia atau 2). berat badan menggunakan formula Holliday-Segar.

Di United Kingdom, tidak ada rekomendasi asupan air per hari yang telah disepakati. Kebutuhan air digunakan berdasarkan rekomendasi *US National Academies Food and Nutrition Board*yang menganjurkan untuk usia 1-3 tahun harus minum air 0,9 L per hari, untuk usia 4-8 tahun harus minum air 1,2 L per hari, untuk usia 9-13 tahun dibedakan berdasarkan jenis kelamin yaitu untuk perempuan: 1,6 L per hari dan laki-laki: 1,8 L per hari, sedangkan untuk usia 14-18 tahun perempuan memerlukan 1,8 L per hari dan laki-laki memerlukan sebanyak 2,6 L per hari. Pada keadaan cuaca panas dan aktivitas fisik, asupan air harus lebih tinggi.[12]

Asupan air di Indonesia dapat dilihat pada peraturan Menteri Kesehatan Republik Indonesia dan konsensus kebutuhan air pada anak sehat oleh Ikatan Dokter Anak Indonesia. Berdasarkan Peraturan Menteri Kesehatan Republik Indonesia nomor 75 tahun 2013 tentang angka kecukupan gizi yang dianjurkan bagi bangsa Indonesia, di dalamnya termasuk kebutuhan air.

Tabel 3. Kebutuhan Air Minimal Berdasarkan Usia[13]

Kelompok usia	Kecukupan asupan untuk laki-laki			Kecukupan asupan untuk perempuan		
	Dari makanan	Dari minuman	Total air	Dari makanan	Dari minuman	Total air
0-6 bulan*	0	700	700	0	700	700
7-12 bulan	200	600	800	200	600	800
1-3 tahun	400	900	1.300	400	900	1.300
4-8 tahun	500	1.200	1.700	500	1.200	1.700
9-13 tahun	600	1.800	2.400	500	1.600	2.100
14-18 thn	700	2.600	3.300	500	1.800	2.300

Keterangan:*: kebutuhan air dipenuhi dengan ASI eksklusif

Tabel 4. Kebutuhan Air pada Anak Berdasarkan Berat Badan dengan Formula Holliday-Segar[13]

Berat badan (Kg)	Kebutuhan air dalam 24 jam
< 10 Kg	100 mL/kgbb
10-20 Kg	1.000 + 50 mL/kgbb untuk setiap kilogram kenaikan berat badan di atas 10 kg
>20 Kg	1.500 + 20 mL/kgbb untuk setiap kilogram kenaikan berat badan di atas 20 Kg

Asupan Air Tidak Adekuat

Berbagai penelitian menunjukkan bahwa asupan air umumnya masih di bawah kebutuhan air yang dianjurkan. Penelitian DONALD di Jerman menunjukkan hidrasi tidak adekuat pada anak usia 4-11 tahun, laki- laki: 49% dan perempuan: 29%,[14] dan 75% anak usia 9-11 tahunberangkat ke sekolah tanpa minum.[15]

Data *National Health and Nutrition Examination Survey* (NHANES) 2005-2010 terhadap 4.766 anak usia 4-13 tahun, mereka mengonsumsi air sekitar 70-75% dari kebutuhan harian dan 25-30% diperoleh dari

makanan. Sekitar 75% anak usia 4-8 tahun, 87% anak perempuan usia 9-13 tahun, dan 85% anak perempuan usia 13 tahun tidak memenuhi kebutuhan air total harian.[16]

Penelitian lainmelaporkan bahwa 20% anak dan remaja mengonsumsi air di bawah standar *European Food Safety Authority* (EFSA). Pada beberapanegara didapatkan bahwa sebagian besar anak mengonsumsi air minum kurang dari setengah asupan yang dianjurkan, dan hanya 40% laki-laki dan 60% perempuan mengonsumsi air minum sesuai dengan standar EFSA.[17,18]

Penelitian di Indonesia oleh *The Indonesian Hydration Regional Study*: mendapatkan bahwa kurang air atau dehidrasi didapatkan pada 45% dari 605 remaja di 6 kota besar. Hardinsyah *et al*. (2012) melaporkan bahwa asupan air adekuat pada anak usia 6-9 tahun, laki-laki sebesar 92,6% dan perempuan: 95,4%, sedangkan pada anak remaja, laki-laki: 55,6% ± 23,6%, dan perempuan: 64,7% ± 25,4%.[19]

Institute of Medicine dan EFSA menyebutkan bahwa dehidrasi 2% dapat menurunkan konsentrasi waktu belajar menurun. Pada penelitian Benton dan Davis (2011), yang dilakukan terhadap anak berusia 9 tahun yang dibagi menjadi dua kelompok, yakni yang minum air dan tidak minum air selama 2 jam. Siswa disuruh menjawab soal matematika dalam kelas dengan suhu ruangan 24 $\pm$ 1 ^{0}C. Hasil penelitian menunjukkan bahwa persentase siswa yang dapat menjawab soal matematika lebih tinggi pada kelompok yang mengonsumsi air dibandingkan dengan yang tidak mengonsumsi air (78,8 vs. 53,0% dengan $p < 0,0001$). Pada 5 menit pertama, pada kelompok yang mengonsumsi air terdapat 85,4 $\pm$ 1,8 % siswa dapat menyelesaikan soal dan 57,2 $\pm$ 3,8% pada kelompok tidak mengonsumsi air, dan menit ke 30, sebanyak 72,7 $\pm$ 2,3% kelompok minum air dan 47,6 $\pm$ 3,2 % kelompok tidak minum air yang dapat menyelesaikan soal.[20]

Konsumsi Minuman dan Kaitannya dengan Obesitas

Untuk meningkatkan konsumsi air per hari, berbagai upaya dilakukan antara lain dengan edukasi tentang pentingnya hidrasi sehat baik melalui media cetak, media elektronik, atau sarana komunikasi lain yang tersedia, atau dengan menyediakan sarana air minum. Upaya lain untuk meningkatkan konsumsi air adalah dengan menyediakan air untuk diminum dengan berbagai kemasan, dengan memberikan warna atau rasa. Hal ini menyebabkan konsumsi air per hari meningkat, namun di sisi lain dapat berdampak pada peningkatan asupan kalori karena beberapa air dalam kemasan yang tersedia juga mengandung kalori dengan kadar yang berbeda-beda.

Belakangan ini, konsumsi *sugar sweetened beverages* atau minuman mengandung gula sebagai asupan air semakin meningkat. Berbagai jenis *sugar sweetened beverages* terdapat di pasaran yang mengindikasikan bahwa masyarakat semakin banyak yang mengonsumsi minuman tersebut, termasuk anak-anak. Berbagai penelitian membuktikan terdapat kaitan erat antara konsumsi *sugar sweetened beverages* dengan peningkatan kejadian obesitas.

Meta-analisis *long-term prospective cohort studies* oleh Te Morenga *et al*.,(2015) menunjukkan bahwa *overweight* atau obesitas: lebih banyak pada anak yang mengonsumsi banyak minuman bergula (*sugar-sweetened beverages*) dibandingkan dengan yang lebih sedikit minum *sugar-sweetened beverages*. Selain itu, reduksi asupan energi dapat menurunkan berat badan yang dapat terlihat jika mengganti minuman *sugar-sweetened beverages* dengan air putih.[21]

Konsumsi minuman berkalori terbukti meningkatkan laju obesitas secara global, yang menyebabkan peningkatan jumlah penyakit komorbid, penurunan kualitas

hidup, dan meningkatkan pengeluaran untuk pemeliharaan kesehatan, sehingga upaya mencegah obesitas merupakan prioritas di berbagai negara. Asupan minuman berkalori berkontribusi terhadap meningkatnya kejadian diabetes melitus tipe 2 dan penyakit kardiovaskular. [22]

Penelitian De Ruyter *et al*. pada anak membandingkan pemberian minuman bebas gula dengan minuman mengandung gula dikaitkan dengan penambahan berat badan. Penelitian dilakukan terhadap 641 anak dengan berat badan normal berusia 4 tahun 10 bulan hingga 11 tahun 11 bulan. Penelitian dilakukan dengan membagi subjek menjadi dua kelompok yakni kelompok yang mendapat *artificial sweetened beverages* (0 kcal) dan *sugar containing beverages* (104 kcal) selama 18 bulan. Hasil penelitian menunjukkan terdapat kenaikan berat padan sebesar 6,35 kg pada kelompok bebas gula vs. 7,37 kg pada kelompok yang mendapat gula. Penelitian ini juga meyimpulkan bahwa penggantian minuman mengandung gula dengan minuman tidak berkalori menurunkan berat badan dan timbunan lemak.[23]

Pada tahun 2012, Ebbelin *et al*. melakukan penelitian *randomized* mengenai pemberian minuman mengandung gula dan berat badan pada 224 anak remaja (124 orang laki-laki dan 100 permpuan) *overweight* dan obesitas yang rutin mengonsumsi *sugar sweetened beverages*. Subjek dibagi menjadi dua kelompok yaitu kelompok eksperimen dan kelompok kontrol. Pada kelompok eksperimen dilakukan intervensi dengan mengganti konsumsi minuman bergula dengan minuman non kalori setiap dua minggu selama 1 tahun dan pada tahun kedua tidak dilakukan intervensi. Pada kelompok kontrol tidak dilakukan intervensi. Hasil penelitian menunjukkan bahwa pada 1 tahun: pertama, peningkatan index massa tubuh pada kelompok eksperimen lebih rendah dibandingkan kelompok kontrol, sedangkan pada 2 tahun tidak ada perbedaan peningkatan index massa tubuh pada kedua kelompok.[24]

The Choose Healthy Options Conciously Everyday (CHOICE) di North Carolina, USA, melakukan penelitian *randomized clinical trial* terhadap orang dewasa pada Mei 2008 – Januari 2010 untuk mengetahui peran minuman berkalori terhadap berat badan, yaitu dengan mengganti minuman berkalori dengan air atau minuman non kalori dan dievaluasi penurunan berat badan. Penelitian dilakukan pada 318 subjek *overweight* dan obesitas berumur 18-65 tahun, yang dibagi menjadi tiga kelompok yaitu 105 kelompok kontrol dan 213 kelompok intervensi yang dibagi menjadi dua kelompok yaitu 108 kelompok minum air putih (non kalori) dan 105 kelompok minum minuman berkalori. Dilakukan *follow-up* selama 3 bulan. Kemudian pada kelompok intervensi, dilakukan penggantian minuman berkalori dengan air putih pada minuman non kalori diberikan minuman berkalori. Hasil penelitian ini menyimpulkan dengan penggantian minuman berkalori dengan minuman non kalori terdapat penurunan berat badan sebanyak 2% -2,5%.[25]

Pada penelitian *randomized, controlled cluster* tentang promosi minum air terhadap siswa sekolah untuk mencegah *overweight* pada 32 sekolah dasar di Dortmund dan Essen, Jerman pada Agustus 2006-Juli 2007. Pada 17 sekolah dilakukan intervensi dengan menyediakan tempat minum dan promosi konsumsi air, sedangkan pada 15 sekolah tidak dilakukan intervensi. Terdapat 2950 siswa yang terdiri atas 1641 kelompok intervensi dan 1309 kelompok kontrol. Setelah satu tahun ajaran sekolah, dilakukan penilaian. Hasil menunjukkan bahwa setelah intervensi, risiko *overweight* turun 31% dibanding kontrol. Disimpulkan bahwa edukasi dan lingkungan efektif untuk meningkatkan konsumsi air minum untuk mencegah *overweight* pada anak.[26,27].

Pada orang dewasa, mengonsumsi diet hipokalori dan minum air putih berhubungan dengan penurunan berat badan. Dennis *et al* melakukan penelitian *randomized* terhadap 48 pasien dewasadengan *overweight* atau obesitas yang dibagi menjadi dua kelompok, yaitu kelompok dengan diet hipokalori plus 500 mL air putih setiap makan dan kelompok dengan diet hipokalori (kontrol). Diet hipokalori: 1200 Kkal/hari untukperempuan dan 1500 Kkal/hari untuk laki-laki. Setelah 2 minggu, terlihat bahwa berat badan kelompok dengan diet hipokalori plus 500 mL air turun 2 Kg lebih banyak dibandingkan kelompok kontrol.[28]

Meta-analisis *long-term prospective cohort studies* oleh Te Morenga *et al*., 2015 menunjukkan bahwa *overweight* atau obesitas lebih banyak pada anak yang mengonsumsi banyak *sugar-sweetened beverages* dibandingkan dengan yang lebih sedikit minuman *sugar-sweetened beverages* Selain itu, reduksi atau pengurangan asupan energi menurunkan berat badan yang terbukti dengan mengganti minuman *sugar-sweetened beverages* dengan air putih. [21]

Tata Laksana Obesitas

Perubahan gaya hidup untuk menurunkan berat badan akan menurunkan insiden diabetes dan hipertensi, yang dapat dilakukan dengan perubahan sosial yang mendasar.[29] Mencegah obesitas dimulai sejak dini yaitu sejak masa kehamilan (penambahan berat badan ibu harus dipantau), pemberian air susu ibu, faktor psikososial (makan bersama keluarga, mengatur pola makan sehat sejak dini), modifikasi pola diet (membatasi konsumsi minuman yang mengandung banyak gula, perbanyak buah dan sayuran, mengurangi makan di restauran, membatasi porsi makanan), dan aktivitas fisik seperti berolah raga setiap hari, meminimalkan jam menonton televisi atau gadget lainnya.[6,29]

Upaya lain yang dapat membantu menanggulangi obesitas adalah asupan air minum yang tidak mengandung gula atau kalori (*non-sugar-sweetened beverages.* Dalam kepustakaan disebutkan bahwa minum air putih dapat menurunkan berat badannamun belum banyak dibicarakan sebagai tata laksana obesitas.

Bagaimana Minum Air Putih Menurunkan Berat Badan?

Penelitian melaporkan bahwa mengonsumsi air putih dapat menurunkan berat badan. Hal ini dapat diterangkan melalui asupan kalori yang berkurang dan meningkatnya oksidasi lemak. Asupan kalori total berkurang karena minum air putih menyebabkan rasa kenyang tetapi tidak mengandung kalori, yang dapatmenyebabkan konsumsi makanan atau kalori berkurang.[16] Kandungan kalori pada air minum adalah 0 kalori, sedangkan minuman berkalori mengandung sekitar 200 kalori per Liter. Minum air putih non kalori meningkatkan oksidasi lemak melalui peran insulin karena minum air non kalori tidak menstimulasi insulin.[15,30].

Bagaimana minum air putih meningkatkan oksidasi lemak? Oksidasi lemak akan maksimal jika kadar insulin dalam darah rendah. Sebagaimana diketahui, insulin menghambat enzim (*hormone-sensitive lipase, acylcarnitine transferase, pyruvate carboxylase*) yang memecah trigliserida menjadi *free fatty acid*, menghambat transpor *free fatty acid* ke dalam mitokondria dan oksidasi melalui *tricarboxylic acid* atau *Krebs cycle.*

Penelitian menunjukkan bahwa oksidasi lemak lebih tinggi 40% setelah air minum dibanding dengan minuman berkalori, dan menurun setelah asupan makanan karena insulin meningkat. Selain itu, makan plus air minum tidak berkalori dibandingkan dengan makan plus minuman berkalori

menyebabkan kadar insulin darah dan oksidasi lemak lebih cepat 2 jam ke keadaan sebelum makan pada yang minum air tidak berkalori dibandingkan dengan minuman berkalori.[30]

Ada penelitian lain yang membanding-kan kadar insulin dan oksidasi lemak pada subjek yang makan dan minum air putih dibandingkan dengan makan dan minum minuman berkalori. Hasilnya menunjukkan bahwa kadar insulin darah dan oksidasi lemak lebih cepat 2 jam kembali ke keadaan sebelum makan pada yang subjek yang makan dan minum air putih dibandingkan dengan subjek yang makan dan minuman berkalori. Asupan 500 - 600 Kkal karbohidrat dapat menekan oksidasi lemak dalam 6 jam setelah makan.[30]

Kesimpulan

Obesitas merupakan masalah kesehatan global. Konsumsi minuman bergula atau berkalori berperan terhadap kejadian obesitas pada anak. Minum air putih berperan menurunkan berat badan melalui pengurangan asupan kalori dan oksidasi lemak.

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Tinjauan Pustaka

Malaria in Pregnancy: A Holistic Review and Approach to Laboratory Findings, Management and Outcomes

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Abstract

Malaria is an infectious disease caused by protozoa of the genus *Plasmodium*, transmitted by the bite of female mosquitoes Anopheles. Malaria can infect various populations including pregnant women. The incidence of malaria in pregnancy is quite high especially in tropical - endemic regions such as Indonesia, Papua New Guinea, Nigeria, West Africa and Sudan. It happens because there is no enhanced control activities such as two prevention approaches advocated by the WHO, as well prevention intermittently with sulfadoxine- pyrimethamine (SP) and the treatment of malaria during pregnancy. There are several factors that influence the high prevalence of malaria such as mosquito vectors, malaria parasites, human hosts and environment. As a result of the presence of malarial parasites in the placenta appear serious adverse outcomes to the mother, fetus and newborn. Malaria can be diagnosed with microscopy, rapid diagnostic test (RDT), and polymerase chain reaction (PCR).The first-line treatments recommended by the WHO in the second and third trimester of pregnancy are artemisinin-based combination treatments (ACT).

Keywords: Malaria, pregnancy, laboratory findings, management, outcomes

Malaria dalam Kehamilan: Telaah Holistik, Pendekatan Laboratorum, Tata Laksana dan Luarannya

Abstrak

Penyakit malaria merupakan infeksi yang disebabkan oleh genus *Plasmodium*, yang ditularkan melalui gigitan nyamuk Anopheles betina. Malaria dapat menginfeksi berbagai populasi termasuk perempuan hamil. Insidens malaria dalam kehamilan cukup tinggi terutama di wilayah tropis - endemik seperti Indonesia, Papua Nugini, Nigeria, Afrika Barat, dan Sudan. Hal itu terjadi karena ketiadaan penanggulangan/pencegahan seperti yang dianjurkan WHO yaitu pemberian sulfadoxine- pyrimethamine (SP) intermiten dan pengobatan malaria dalam kehamilan. Ada banyak faktor yang berperan terhadap kejadian malaria yakni nyamuk sebagai vektor, parasit malaria, manusia sebagai pejamu dan lingkungan yang juga berperan terhadap kejadian malaria dalam kehamilan. Malaria dalam kehamilan berakibat serius trehadap ibu, janin dan neonatus. Diagnosis dapat ditegakkan secara mikroskopi, *rapid diagnostic test* (RDT), dan polymerase chain reaction (PCR). Pengobatan lini pertama yang direkomendasikan oleh WHO pada trimester ke dua dan ketiga kehamilan adalah kombinasi artemisinin (*artemisinin-based combination treatments* - ACT).

Kata kunci: Malaria, kehamilan, pemeriksaan laboratorium, tatalaksana, luaran

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Introduction

Malaria is an infection caused by protozoa of the genus Plasmodium. The species of plasmodium include *Plasmodium falciparum*, *Plasmodium vivax*, *Plasmodium ovale* and *Plasmodium malariae.*[1]According to World Health Organization (WHO), most of the cases in 2016 were in the African (90%), followed by South-East Asia (7%) and Eastern Mediterranean (2%).[2] Malaria is also a cause of mortality and morbidity in pregnant women globally.[1,3] It is estimated 125 million women each year are at risk of malaria in pregnancy.[4-5] The incidence of malaria in pregnancy is still high, especially in endemic tropical countries such as Indonesia (Papua 10%, Nusa Tenggara Timur 5%)[6], Papua New Guinea 18,5%, Nigeria (Igbo-Eze North 50.7%)[7], and West Africa 40.2%[8] , and Sudan (Blue nile state 58.9%)[9]. No enhanced control activities as recommended by the WHO, as well prevention intermittently with sulfadoxine-pyrimethamine (SP) and the treatment of malaria during pregnancy, it can be a potential cause the prevalence remain high.[9]

Malaria infections could occur in blood of mother or in the placenta.The presence of infected erythrocytes or malaria pigment (haemozoin) in the placenta intervillous referred to as placental malaria. The occurrence of placental malaria is influenced by many factors such as primipara, multipara, live in rural areas, young age ≤ 23.2 year, did not undergo the ante natal care (ANC), do not use the nets when sleeping, and the absence of clinical symptoms malaria.[10-12]

As a result of the presence of malarial parasites in the placenta serious adverse outcomes occurs to the mother, fetus and newborn such as maternal anemia, low birth weight (LBW) baby, intrauterine growth restriction, preterm birth (PTB), miscarriage, stillbirth, and congenital malaria.[6,7,13-15]This article will discuss about the laboratory findings, management and adverse outcomes of malaria in pregnancy. The data taken were from the year 2014 to 2017 research articles.

Laboratory Findings

The crucial issue in the management of malaria is consideration of the possibility of this diagnosis. Malaria should be suspected in anyone with a fever or a history of fever who has returned from or previously visited a malaria endemic area. There are no specific symptoms of malaria: most patients complain of fever, headache and general malaise, gastrointestinal disturbances, jaundice. Malaria infection during pregnancy is often asymptomatic.[16] The effects of malaria infection in the first trimester are largely unknown.[17] Because of the low parasitemia found in all age groups pregnant women may be the reason why they do not show malaria symptoms even though they are infected. This suggests that pregnant women can become carriers of malarial parasites without showing any symptoms of infection.[18] Malarial parasites in pregnancy can be found with microscopy, rapid diagnostic test (RDT), and polymerase chain reaction (PCR).

Microscopy examination was made using thick and thin smears on the same slide and stained with 5 % Giemsa for 30 minutes.[19] A smear was declared negative if examination of 100 high power fields did not reveal asexual parasites or gametocytes. For positive smears, parasite density was calculated by counting the number of asexual parasites per 200 leukocytes (or per 500, if the count is <10 asexual parasites/200 leukocytes), assuming a leukocyte count of 8,000/µl. When a thick smear was positive, the corresponding thin blood smear was read to determine the parasite species.[20]

Malaria rapid diagnostic tests (RDTs) assist in the diagnosis of malaria by detecting evidence of malaria parasites in human blood.

The test principle underlying malaria RDTs is the detection of parasite antigens, most commonly histidine-rich protein 2 (HRP2), parasite lactate dehydrogenase (pLDH), and/or parasite aldolase through lateral flow immunochromatography. Whereas HRP2 is specific to *Plasmodium falciparum*, pLDH and parasite aldolase are common to all *Plasmodium* species.[21]

RDT-HRP2 tests have a higher sensitivity compared with RDT-pLDH.[22] Based on this study, the HRP2 assay showed superior sensitivity but inferior specificity compared with the pLDH assay. The difference in sensitivity between the tests was due mostly to better detection with HRP2 at low parasite densities. Non-falciparum infections contributed to false negative results for both RDTs. In particular, in two-thirds of cases in which the HRP2 test was negative although microscopy detected parasites, the infection was caused by non-falciparum species. The higher specificity and positive predictive value of the pLDH assay was due to the fact that pLDH antigenemia closely mirrors parasitemia, while HRP2 commonly persists in the bloodstream weeks after successful treatment of malaria.[23]

When RDT-HRP2 performed on peripheral blood samples for pregnancy malaria diagnoses, the sensitivity was more than 90% when it compared with peripheral blood smears, and 80–95% when it compared with placental BSwith specificity between 61%- 94%. The sensitivity of RDT-HRP2 using peripheral blood samples was much lower when compared with PCR detection of parasite nucleic acids in peripheral or placental blood.[22]

Other alternatives for malaria diagnosis are DNA/RNA-based detection techniques, of which the PCR is the most widely used.[24] PCR methods to detect malaria infection were described more than two decades ago, and generally more sensitive for detecting parasite DNA than thick blood smears for detecting parasites. Whether applied to peripheral blood or placental blood samples, PCR methods yield positivity rates 20% or more above those of blood smears microscopy. Infections identified by PCR, or RDT when microscopy fails to detect parasites on blood smears are defined as submicroscopic infections. PCR methods have been applied to detect submicroscopic pregnancy malaria. Although these tools are more sensitive for parasite detection compared with microscopy, the test format and the time to obtain results are not suitable for use in a primary care setting.[22]

Management

Therapy

According to the WHO, seven days of quinine (10 mg) plus clindamycin (5 mg) twice a day is the preferred regimen to treat malaria in the first trimester of pregnancy.[25] If clindamycin is unavailable or unaffordable, quinine monotherapy should be given.[26] Although this regimen thought to be safe during pregnancy, we should be aware of the side effects. Clindamycin does pass through the placenta and may be accumulated in the fetal liver.[27] The use of quinine is limited by its disadvantages, including a long treatment course and an increased risk of hypoglycaemia.[28]

The first-line treatments now recommended by the WHO for malaria in the second and third trimester of pregnancy are artemisinin-based combination treatments (ACT); these are combinations of fast acting artemisinin-based compounds that combined with a drug from a different class. Artemisinin derivatives include dihydroartemisinin, artesunate and artemether.[29] They are eliminated rapidly and well tolerated; none of the studies reported serious adverse event attributed to the use of artemisinin.[30] Four ACTs are currently recommended:

artesunate–mefloquine, dihydroartemisinin-piperaquine, artesunate–amodiaquine, and artemether–lumefantrine.[31] The benefits of ACTs are their high efficacy, fast action, and the reduced likelihood of resistance developing.[32]

The combination of artesunate-mefloquine is currently one of the most effective treatments against malaria in pregnancy.[33] Typical dosage are 100 mg artesunate and 220 mg mefloquine every 8 hours for 3 days.[34] Mefloquine has many characteristics such as long half-life, single dose administration, a well-characterized pharmacokinetic profile in pregnant women, and infrequent mefloquine resistance. It often gives rise to side effects such as asthenia, loss of appetite, dizziness, nausea, and vomiting.[33,35]

New infections can be prevented by long-acting artemisin such as dihydroartemisin-piperaquine especially for areas with high malaria transmission.[36] The fixed dose combination formulated in tablets containing dihydroartemisinin (40 mg) and piperaquine (320 mg) is commercially available in many countries in Asia, and also more recently in Africa. The fixed combination given once daily for 3 days was effective and well tolerated. The most common adverse effects are gastrointestinal (nausea, vomiting, abdominal pain, and diarrhea), but they are usually mild and self limiting.[37]

Artesunate-amodiaquine has the lowest side effects on the frequency of pulse and blood tension.[38] Some studies have shown that amodiaquine or combinations of amodiaquine with artesunate were better at clearing parasitaemia compared with chloroquine given alone. The total recommended treatment is 50 mg of artesunate and 153 mg of amodiaquine once a day for 3 days.[39]

Artemether-lumefantrine is a fixed-dose antimalarial drug combination in pregnancy containing 40 mg artemether and 240 mg lumefantrine per tablet as a 6-dose regimen given twice a day for 3 days. The first two doses should, ideally, be given 8 hours apart.[40] It is tolerated very well in the majority of cases, but it has the highest grade of reinfection because lumefantrine possesses short effect that it can be eliminated rapidly.[41]

Prevention

The prevention of malaria is important to reduce adverse outcomes of malaria in pregnancy. Centers for Disease Control and Prevention (CDC) recommends pregnant women who do travel to malaria countries to: I) begin a prophylactic antimalarial medication such as mefloquine (228 mg) or chloroquine (310 mg) before travelling begin i.e. two weeks before travel, during travelling once a week, on the same day of the week and continue post-travel for 4 weeks,II) [42] wear long-sleeved shirts and long pants to prevent mosquito bites, III) remain outdoors during peak time, IV) use insect repellent with N,N-Diethyl-m-toluamide (DEET) or Picaridin, and V) sleep in a well-screened or air-conditioned room or under a mosquito net treated with permethrin.[43] While the inhabitants of endemic areas, WHO recommends interventions such as intermittent preventive treatment in pregnancy (IPTp) and insecticides-treated nets (ITNs) to prevent and control malaria during pregnancy.[44]

Intermittent preventive treatment in pregnancy is the administration of antimalarial medications at predefined intervals with the intent to protect a pregnant woman against malaria.[45] It is advised not to be given to pregnant women in the first trimester because they are teratogenic and may cause fetal death.[46] Intermittent preventive treatment in pregnancy administration of SP, sulphadoxine (500 mg) and pyrimethamine (25 mg) starting as early as possible in the second trimester until the

time of delivery, with doses given at least 1 month apart, so that women receive at least three doses of SP during pregnancy.[47]

The usage of treated nets offer a form of personal protection and have repeatedly been shown to reduce severe diseases and death due to malaria for both the pregnant women and children in endemic regions.[48] The insecticides used for treating the nets kill mosquitoes and other insects. This insecticide also has repellent properties that reduce the number of mosquitoes that enter the house and attempt to feed thereby offering protection not only for the person under the net but also for those in the same room with the net owner.[49] In addition, other similar protection such as indoor insecticide residual sprays (IRS) and mosquito coils (MC) can also be used.[50]

Several studies have also mentioned some alternatives that can prevent malaria in pregnancy such as the consumption of azithromycin-chloroquine (250 mg azithromycin, 155 mg chloroquine) in combination doses, or supplementation with 25 mg zinc and 2500 IU vitamin A everyday.[51-53] However, there has not been much research to discuss this.

Outcomes

Malaria infections during pregnancy are often asymptomatic.[16] The effects of malaria infection in the first trimester are mostly unknown.[17] Low parasitemia found in all pregnant women age group may be the reason why malaria infection did not show any symptoms. This suggests that pregnant women can become carriers of malarial parasites without showing any symptoms of infection.[18]

Women primigravida uniquely vulnerable because they do not have a specific malaria immunity during pregnancy in the form of antibodies and memory B cells against a specific parasite variant surface antigens VAR2CSA. This protective antibody responses develop during subsequent pregnancies, providing protection against placental malaria. Live in rural area increase heterogeneous malaria transmission in Papua New Guinean.[11] Other data showed higher risk of placental malaria in women who experienced 0-1 episodes of malaria symptoms and <50% of the samples LAMP +; and women who have ≥ 2 episodes of malaria symptoms or ≥ 50% of the samples LAMP + compared to pregnant women without exposure to malaria.[12]

Complication of malaria in pregnancy include anemia, low birth weight, IUGR, premature birth, abortus, stillbirth, and congenital malaria. Infection in first or second trimester raise the risk of LBW, wherein acute infection of malaria is associated with LBW, with an average birth weight 199 gram lower. Acute malaria infection with high parasitemia is associated with premature birth. Chronic infection also associated with premature birth, while chronic placental malaria infection with chronic massive intervillositis is associated with anemia and LBW in pregnancy, specifically moderate anemia (7.0-8.9 g/dL) and severe anemia (<7.0 g/dL). Pregnant women with malaria are 33% more likely to develop anemia. Low birth weight is also associated with peripheral malaria infection during labor. [9,11,43]

Infection of *P. falciparum* in early pregnancy can cause intra uterine growth restriction (IUGR) between 212-253 days of gestational age, and the gestational age becomes shorter. Birth weight and weight of the placenta also found reduced. Small placental volumes in early pregnancy may be used as IUGR predictor at birth. Small placental size is associated with disorder of trophoblast invasive, late complication, and premature birth due to exposure to the VAR2CSA protein. The process underlining VAR2CSA cause IUGR is not

fully understood. *Plasmodium falciparum* divide itself in erythrocyte, makes the infected erythrocytes are more prone to be discharged into the spleen. To prevent it, the parasite express *P. falciparum* erythrocyte membrane protein 1 (PfEMP1) in the surface of infected cells. PfEMP1 is a multiprotein and each of the parasite's genome encoded around 60 different variant of protein, which one of it is VAR2CSA. This protein binds to most of tumor cells, the bound cells show decreased migration, invasion, and growth. VAR2CSA also binds infected erythrocyte to syncytiotrophoblast expressed glycosaminoglycan and present in the intervillous section. The infected erythrocyte triggers the inflammation response that negatively affected the fetal growth. VAR2CSA also may affect trophoblast and cause permanent damage to proper uteroplacental circulation.[13]

Plasmodium falciparum detected during labor in peripheral blood flow and in the placental blood flow increase the risk of stillbirth, meanwhile *Plasmodium vivax* increase the risk of stillbirth if detected during labor but not when detected and treated during pregnancy. Association between *P. falciparum* infection during pregnancy and stillbirth is two times higher in low-medium endemic area compared to high-endemic area. Increased CLCX9 in peripheral blood flow during pregnancy linked with increased risk of born premature and abortus. Increased exosome or change of microparticles composition activated peripheral mononuclear cells has been speculated to cause proinflammation immune response which associated with born premature and abortus. Increased of IL-1β di placental blood circulation also associated with miscariage and born premature.[4,54]

Congenital malaria defined as presence of the malaria parasites in asexual form either in placental blood flow or in peripheral blood flow during the first week of life, caused by the transmission of the parasite through the placental before or during the labor. In Burundi, prevalence of congenital malaria shows 0% even though the survey was carried in meso-endemic area while in the wet, high transmission season. It is assumed that during the high transmission season, mothers acquired a high immunity level and their malaria specific antibody rise when the mothers exposed to the infection. None of the newborns born from microscopy or RDT positive mothers found to be qPCR-positive might can confirm that placenta play an effective role as a barrier. Two newborns born from mothers with high parasitemia found to be HRP-2 RDT positive, but negative with qPCR, suggest that the HRP-2 antigen can pass through the placenta, while the DNA isn't transmitted.[55]

Compared to Burundi, in Maumere, Indonesia, the prevalence of congenital malaria in selected subgroup of newborns with an increased risk of congenital malaria was 42,4%, while the prevalence in all newborns was 14,7%. The congenital infection of the parasite can occur at different time, either it is *in utero* or during labor. The mechanism might be done by direct penetration through the chorionic villi or might be through premature separation of the placenta. The infected infants are at 4,7 times higher risk to develop anemia. Congenital malaria mostly found in first or second child, similar to placental malaria.[56]

Infants born to mothers that has been exposed to malarial infection more than once, or experienced placental malaria before, have higher risk of impaired growth in the first year of life. Infants born to mothers infected with malaria in their first year of life may experience at least one episode of malaria (5%), acute respiratory infection (51%), or diarrhea (22%). The risk of malaria infection in infants increased three-folds in infants born to mothers with peripheral malaria infections, dan increase

more than ten-folds in infants born to mother with placental malaria.[57]

Conclusion

Malaria in pregnancy is common in everyday life, especially in endemic areas. Therefore, with prevention as early as possible, the definitive diagnosis and the best therapy should make pregnant women free from adverse outcomes such as maternal anemia, miscarriage, intrauterine growth restriction, low birth weight, preterm birth, stillbirth, and congenital malaria.

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