AUSTRALIA

Communicable Diseases Intelligence

Bulletin number 81/2
Issue date: 30 January 1981

Contents:

TOXIC-SHOCK SYNDROME

(Contributed by L.E. Bolitho, N. Bhardwaj, and R. Sloman, Western General Hospital, Footscray, Victoria.)

Two days after commencing a normal menstrual period using tampons, a 30 year old woman experienced rigors and felt generally unwell. She later developed vomiting, diarrhoea and conjunctival irritation. Her local doctor diagnosed gastroenteritis and prescribed Lomotil and Stemetil tablets. The next day she was found semi-conscious and in shock. She was admitted to Western General Hospital after she was examined by another general practitioner.

On arrival she was semicomatose, hypotensive (BP 70/40 mm Hg) and had generalised erythroderma and conjunctival injection. Temperature ranged from 39° to 41°C, pulse rate was 160/min, respiratory rate 30/min. A vaginal discharge was noted and a purulent bloodstained tampon was removed. Vaginal examination revealed a soft oedematous cervix. The uterus and adnexae were normal, and there were no other abnormalities.

The patient was transferred to intensive care, and was resuscitated with intravenous fluids. Her condition improved rapidly, but a dopamine infusion was required for 12 hours to maintain blood pressure. A wide spectrum of antibiotics was administered intravenously (penicillin, ampicillin, gentamicin, metronidazole) until sensitivities were available.

On the second day in hospital the patient developed a scarlatiniform rash on her trunk. There was ECG evidence, but no clinical signs or symptoms, of pericarditis. Fine desquamation of the skin on her face and trunk was noted on the third day.

Staphylococcus aureus, sensitive to all antibiotics, was cultured from swabs of cervical pus and from the tampon, so the antibiotic regimen was changed to cloxacillin. Cultures from blood, urine, faeces and cerebrospinal fluid showed no pathogens. Blood specimens revealed anaemia, and an elevated white cell count with the neutrophils showing toxic changes. Metabolic acidosis was also evident, and a clotting profile indicated a mild coagulopathy.

Editorial Comment - The hospital has commented that one of three unopened tampons left in the pack used by the patient was contaminated with a strain of S. aureus sensitive to all antibiotics. The remaining two tampons

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β-LACTAMASE PRODUCING N. GONORRHOEAE - 1980

Gonorrhoea infections are notifiable in all States, although analysis of the incidence of the disease is influenced by the variations in reporting and availability of microbiological testing. Interpretation of the available data shows no increase in the incidence of gonorrhoea in 1980, e.g. 9781 reports received to October 1980, compared with 11647 and 12352 reports for the whole of 1978 and 1979 respectively. However, the increase in the number of laboratory reports of post-pubertal β-lactamase producing strains of N. gonorrhoeae (PPNG) from 42 and 36 in 1978 and 1979 respectively to 144 in 1980, is of concern.

Of the 144 infections, 116 were in males, and 28 in females. The age distribution is shown in Table 1.

TABLE 1 Age distribution of PPNG infections - 1980

	15-20	21-30	31-40	41-50	51+	Unknown
Males	12	59	25	9	1	10
Females	10	13	3	-	-	2
Total	22	72	28	9	1	12

Although the majority of PPNG infections were contracted overseas (87 cases), 46 cases were traced to Australian sources, and several 'chains of infection' were recognised in 1980[1]. Six of the locally acquired PPNG infections were in homosexuals in Adelaide. Five of the isolates were from urethral swabs and one from a rectal swab. Table 2 is an analysis of the State in which each PPNG was recognized together with the suspected source of infection. The figures for infections contracted overseas include infections contracted in Australia from persons who had recently arrived from overseas.

TABLE 2 PPNG Infections by probable source of contact - 1980

Probable Source	ACT	NSW	VIC	QLD	SA	WA	NT	TAS	TOTAL
Philippines	1	8	4	7	7	4	1		32
Thailand	2	2	9		2	5		2	21
Malaysia				3		1			4
Bali			1			1			2
Hong Kong		2	1						3
Fiji			1						1
Singapore			4			2			6
Indonesia			1		2				3
S.E. Asia (unspec.)			1		2	8			11
USA					2	1			3
England						1			1
Australia		5	1	2	28	9			46
Unknown			3		2	6			11
TOTAL	3	17	26	12	45	38	1	2	144

Most of the imported PPNG infections were contracted in S.E. Asia. No cases originated from West Africa.

Table 3 is a collation of the available data on PPNG infections in Canada and the United Kingdom, and indicates that these countries have also experienced similar local PPNG foci.

TABLE 3 PPNG Infections in United Kingdom and Canada

Country	Total	Probable Source			
		Asia	Africa	Other & unknown	Local
England, Wales, Ireland - 1979[2]	104	33	28	20	23
England, Wales, Ireland - June 1980[3]	84	30	19	16	19
Scotland - Aug. 1980[4]	5	2	2		1
Canada - 1979[5]	34	9	2	15	8
Canada - 15 Sept. 1980[5]	20	9	0	6	5

β-lactamase producing strains of N. gonorrhoeae were first isolated in 1976 in the USA from two servicemen who had returned from the Philippines. The incidence of penicillin resistant gonorrhoea in the Philippines has now risen from 30% in 1975 to more than 60% in 1979[6], and the problem has reached global proportions with increasing exportation from the two endemic foci, the Far East and West Africa.

The appearance of two recent cases in Singapore and England of ophthalmia neonatorum caused by PPNG infection has increased this concern[7,8]. Both cases were successfully treated, one with kanamycin, and the other with erythromycin and cefuroxime.

Rigorous contact tracing and early use of effective treatment will minimise the spread of and complications from PPNG infections. The Centers for Disease Control, Atlanta, recently published a number of recommendations concerning PPNG or suspect PPNG infections in MMWR (1980) 29:381 and MMWR (1980) 29:541

"Spectinomycin 2g should be given as the initial treatment for uncomplicated anogenital gonorrhoea in patients who have recently returned from countries such as Philippines, Singapore, Thailand, and other areas of high prevalence of PPNG infections.

The same dosage is recommended for:

- The initial treatment of patients with proven PPNG infection.
- The treatment of sexual partners of these patients.
- The retreatment of patients who have persistent infections after initial therapy with another antibiotic.

Isolates of N. gonorrhoeae obtained from patients treated with spectinomycin should be tested for penicillinase production. All patients treated for gonorrhoea should have a post-treatment culture taken three to seven days after treatment.

There are, as yet, no published studies on the treatment of PPNG-associated salpingitis and PPNG pharyngeal infections. Spectinomycin and cefoxitin appear effective in the treatment of salpingitis caused by penicillin-sensitive gonococci[9,10], and are definitely effective in urethritis caused by PPNG[11,12]. However, these two drugs may be relatively ineffective for pharyngeal gonococcal infection[13,14]. The fixed-combination antimicrobial sulfamethoxazole/trimethoprim has been used to treat pharyngitis caused by penicillin-sensitive gonococci and may be effective for PPNG urethritis[16].

Pending definitive studies, the CDC recommends the following regimens:

. For salpingitis associated with endocervical PPNG infection:-

1. Outpatients - spectinomycin 2g IM daily for 5-10 days.
2. Inpatients - cefoxitin 2g IM or IV every 8 hours for 5-10 days.

Because experience with treatment of this infection is very limited, hospitalisation of most patients may be advisable.

. For PPNG pharyngeal infection:-

Sulfamethoxazole/trimethoprim, 9 tablets (400mg sulfamethoxazole/80 mg trimethoprim per tablet) daily for 5 days; these tablets should be taken as a single daily dose. Sulfamethoxazole/trimethoprim should be avoided by pregnant or nursing women[17]."

The recognition of endemic foci of PPNG in Los Angeles, Pierce County, Washington and New York City emphasised the need for continuing surveillance[18]. As a result the CDC has recommended the expansion of spectinomycin use to include the initial treatment of all patients with uncomplicated gonococcal infection from these areas, and that the practice should be considered when more than 5% of gonococcal isolates in an area are penicillin resistant.

With this resurgence of gonorrhoea the prospects of developing an effective vaccine have been reconsidered.[19] However, the antigenic heterogeneity of the organism is the major obstacle in establishing artificial immunity. The outer surface structures of the gonococcus include a lipopolysaccharide complex, an outer membrane protein complex and attachment pili. Studies have shown a single determinant on the lipopolysaccharide that evokes a bactericidal antibody, and sixteen different serotype specificities on the outer membrane protein complex. The pili also exhibit antigenic variation. However, the pili structures, which are associated with the virulence of the organism, and responsible for the attachment to the epithelial cells on mucosal surfaces, are the most hopeful ingredient for a successful vaccine. Preliminary trials in human volunteers have shown a protective effect with antibody to pili both in blood and in urogenital tract secretions[20].

Nevertheless, the ethical and practical problems that would be encountered in the distribution of a gonococcal vaccine would also be considerable. For example, where could the vaccine be best used in practice, and should

it be available on request or restricted to selected patients in high risk groups?

References

1. CDI (1980) 80/14:2
2. CDR (1980) 80/46:6
3. CDR (1980) 80/40:4
4. CDS (1980) 80/35:v
5. CDWR (1980) 6-40:198
6. JAMA (1980) 244:1884
7. BMJ (1979) 1:380
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10. Obstet Gynecol (1979) 54:193
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12. NEJM (1979) 301:509
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14. Sex Transm. Dis. (1979) 6:239
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17. NEJM (1980) 303:426
18. MMWR (1980) 29:541
19. BMJ (1977) 2:917
20. Science (1980) 209:1103

NOSOCOMIAL RUBELLA INFECTION - USA

(Based on MMWR (1981) 29:629)

Although a record low of 11795 rubella cases were reported to the CDC in 1979, more than 70% were in person aged 15 years and older. This age group includes women of childbearing age, approximately 20% of whom are thought to be susceptible to rubella, and thus at risk of having infants with congenital rubella syndrome (CRS).

Infants with CRS shed large quantities of virus from body secretions up to 12 months of age, and are a known transmission source. This risk is demonstrated in the reports following:

. On 3 December 1979 a two month old infant with CRS was admitted to a hospital in Fargo, North Dakota, for two weeks. The infant had cataracts, hearing loss, congenital heart disease, microcephaly and congenital glaucoma. On 26 December, a 27 year old male doctor who had attended the infant became ill with rubella. The infant was readmitted to the same hospital on 19 January 1980. Sixteen days later a 29 year old nurse who had cared for the infant presented with rubella.

  The infant had been promptly placed in strict isolation on both admissions, and neither of the contacts infected had a history of rubella vaccination.

- On 4 December 1979, a newborn male infant, whose mother had had laboratory confirmed first-trimester rubella infection, was admitted to a newborn intensive care unit in Birmingham, Alabama. Only known seropositive personnel were assigned to his care. However, a staff shortage resulted in a 39 year old nurse (with a rubella HI titre of <8) being detailed for a single eight hour shift. Twenty-three days after the single exposure to the infant, the nurse developed rubella.

- In a six week period in December 1979 to January 1980, four infants with CRS were born at a Cleveland hospital, Ohio. Despite the placing of the infants in strict isolation, three nurses who had had direct contact with one or more of the infants developed rubella.

As a result of these nosocomial infections, the three hospitals introduced stringent policies; either all staff members, or all female employees of childbearing age were required to show proof of rubella vaccination or serological immunity to rubella.

In Australia, the National Health and Medical Research Council considers that the objective of vaccination is not to eliminate the disease, but to induce adequate immunity in females to eliminate the risk of infection during pregnancy. Consequently, it is recommended that hospitals institute policies whereby all female employees of child-bearing age undergo rubella screening before they are employed, and be advised if vaccination is necessary.

AMOEBIC MENINGITIS - SOUTH AUSTRALIA

Primary amoebic meningitis was clinically diagnosed in a ten year old boy from Whyalla who was admitted to Adelaide Children's Hospital on 24 January 1981. Protozoa morphologically similar to Naegleria fowleri were seen in CSF specimens, although species confirmation will involve further culture and serological testing. The patient was given amphotericin B chemotherapy, but died on 28 January. The source of infection has not yet been determined, since samples from municipal swimming pools in Whyalla have shown satisfactory water quality.

The last cases seen in South Australia involved the fatal infection of a seven year old girl and five year old boy from Port Augusta in 1972. Over a 25 year period, 17 cases have been recognized in Australia, 14 from South Australia, one from Queensland, and two from Western Australia (see CDI 80/3, 80/4).

(continued from page 1)
are now being examined. The manufacturers were being contacted at the time of going to press.

This appears to be the first case of toxic-shock syndrome in which pericarditis has been reported.

Dr. Margaret Peel of the Microbiological Diagnostic Unit, University of Melbourne, has also advised preliminary findings on growth of S. aureus in tampons under simulated menstrual conditions. There is no significant growth up till 3 hours, then rapid multiplication up to a maximum at 7 hours. A full report will follow.

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

1

REPORTING PERIOD - 8-1-81 - 21-1-81 BULLETIN NUMBER - 81/2.

VIRAL IDENTIFICATIONS FROM CONTRIBUTING LABORATORIES

VIRUS OR VIRAL ANTIGEN	ICPMR (NSW)/ WVH (ACT)	RAHC (NSW)	PHH/ POW (NSW)	FAIR-FIELD (VIC)	RCH (VIC)	IMVS (SA)	STATE LAB (QLD)	STATE LAB (WA)	Total
0100 ADENOVIRUS NOT TYPED	19	1			1	4	6		31
0101 ADENOVIRUS TYPE 1			2		2				4
0102 ADENOVIRUS TYPE 2			1	1	7				9
0103 ADENOVIRUS TYPE 3	1							2	3
0104 ADENOVIRUS TYPE 4			1						1
0106 ADENOVIRUS TYPE 6						1			1
0107 ADENOVIRUS TYPE 7	1	2			1				4
0119 ADENOVIRUS TYPE 19								5	5
0199 ADENOVIRUS TYPING PENDING		2	3		1				6
0201 INFLUENZA A VIRUS						6			6
0203 INFLUENZA B VIRUS	1			2		8	2		13
0301 PARAINFLUENZA VIRUS TYPE 1						4	1		5
0302 PARAINFLUENZA VIRUS TYPE 2						1			1
0303 PARAINFLUENZA VIRUS TYPE 3	1				2	1	2		6
0399 PARAINFLUENZA VIRUS TYPING PENDING						2			2
0400 RESPIRATORY SYNCYTIAL VIRUS (RS)							2		2
0500 RHINOVIRUS (ALL TYPES)	3				4	3	3	2	15
0600 MYCOPLASMA PNEUMONIAE	2	1				3	13	1	20
0700 ORNITHOSIS-PSITTACOSIS	2			1		3			6
0809 COXSACKIEVIRUS A9				1	3		1	3	8
0902 COXSACKIEVIRUS B2	1				3	2			6
0903 COXSACKIEVIRUS B3	1								1
0904 COXSACKIEVIRUS B4		1					1		2
1006 ECHOVIRUS TYPE 6			1					1	2
1009 ECHOVIRUS TYPE 9					3	1			4
1011 ECHOVIRUS TYPE 11	1		2						3
1014 ECHOVIRUS TYPE 14	1						2		3
1022 ECHOVIRUS TYPE 22			2		4				6
1030 ECHOVIRUS TYPE 30	3			5	2				10
1101 POLIOVIRUS TYPE 1	1	1					1		3
1102 POLIOVIRUS TYPE 2	1			1		1			3
1103 POLIOVIRUS TYPE 3	1	2	2						5

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE 2.

REPORTING PERIOD - 8 - 1 - 81 - 21 - 1 - 81 BULLETIN NUMBER - 81/2.

VIRAL IDENTIFICATIONS FROM CONTRIBUTING LABORATORIES-CONTINUED

VIRUS OR VIRAL ANTIGEN	ICPMR (NSW)/ WVH (ACT)	RAHC (NSW)	PHH/ POW (NSW)	FAIR-FIELD (VIC)	RCH (VIC)	IMVS (SA)	STATE LAB (QLD)	STATE LAB (WA)	Total
1104 POLIOVIRUS-VACCINAL STRAIN					2				2
1200 MUMPS VIRUS	2	1		9		4	2		18
1300 HERPES VIRUS GROUP-NOT TYPED	3			1		3		4	11
1301 HERPES SIMPLEX VIRUS NOT-TYPED	16	2		7	1			52	78
1302 EPSTEIN-BARR VIRUS (EB VIRUS)						5			5
1303 VARICELLA-ZOSTER VIRUS	4					1			5
1306 HERPES SIMPLEX TYPE 1	7			18		9	4		38
1307 HERPES SIMPLEX TYPE 2	69			17		8	13		107
1399 HERPES VIRUS TYPING PENDING			10			2			12
1401 COXIELLA BURNETI	5			2		16	14		37
1514 MOLLUSCUM CONTAGIOSUM								2	2
1515 CONTAGIOUS PUSTULAR DERMATITIS (ORF VIRUS)								1	1
1521 MEASLES VIRUS				1			1		2
1522 RUBELLA VIRUS	1			2		3	1	3	10
1532 HEPATITIS B ANTIGEN	9		5	19		41	5	2	81
1535 HEPATITIS A ANTIBODY	4		3	12		12		3	34
1541 CHLAMYDIA A - TRIC TYPE	24					1		49	74
1556 CMV - CYTOMEGALOVIRUS	6		1	21	2	1	5	3	39
1563 CORONAVIRUS				1					1
1564 ROTAVIRUS	8		5		2	3			18
1599 ENTEROVIRUS TYPING PENDING		3	3		12	1	4		23
ASTROVIRUS	4								4
Total	202	16	41	121	52	150	83	133	798

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PERIOD : 8/1/81 to 21/1/81 .... 81/2.

Viral Identifications by Clinical Information Table 1.
Code 00,99 -No ill or data; 01,02,11,12 -Respiratory; E3 -Encephalitis; M3 -Meningitis; 04 -Paralysis; 05,13 -CNS other unspec.; 07,49 -GI; 17,47 -Hepatic; 19 -CVS; 89 -Urinary; 06 -Skin/mucous.

VIRUS OR VIRAL ANTIGEN	No-ill or data	Respiratory	Encephalitis	Meningitis	Paralysis	CNS other unspec	GI	Hepatic	CVS	Urinary	Skin/mucs memb
0100 ADENOVIRUS NOT TYPED.....	1	4					3				1
0101 ADENOVIRUS TYPE 1.....		1					3				
0102 ADENOVIRUS TYPE 2.....	1	1					4				
0103 ADENOVIRUS TYPE 3.....		1					1				
0104 ADENOVIRUS TYPE 4.....							1				
0106 ADENOVIRUS TYPE 6.....							1				
0107 ADENOVIRUS TYPE 7.....		2					1				
0119 ADENOVIRUS TYPE 19.....	4										
0201 INFLUENZA A VIRUS.....		6									
0203 INFLUENZA B VIRUS.....		8							1		
0301 PARAINFLUENZA VIRUS TYPE 1....		5									
0302 PARAINFLUENZA VIRUS TYPE 2....		1									
0303 PARAINFLUENZA VIRUS TYPE 3....		6		1							
0400 RESPIRATORY SYNCYTIAL VIRUS (RS).....		2									
0500 RHINOVIRUS (ALL TYPES).....		11									
0600 MYCOPLASMA PNEUMONIAE.....	5	9									
0700 ORNITHOSIS-PSITTACOSIS.....		6									
0809 COXSACKIEVIRUS A9.....	1	1					1				1
0902 COXSACKIEVIRUS B2.....	1	1					2				1
0903 COXSACKIEVIRUS B3.....							1				
0904 COXSACKIEVIRUS B4.....		1		1							
1006 ECHOVIRUS TYPE 6.....							1				
1009 ECHOVIRUS TYPE 9.....	1	1					2				
1011 ECHOVIRUS TYPE 11.....				1		1	1				
1014 ECHOVIRUS TYPE 14.....		1		1							
1022 ECHOVIRUS TYPE 22.....	4						2				
1030 ECHOVIRUS TYPE 30.....	1			8							

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE 4

PERIOD : 8/1/81 to 21/1/81 .... 81/2

Viral Identifications by Clinical Information Table 1.
Code 00,99 -No ill or data; 01,02,11,12 -Respiratory; E3 -Enceph-alitis; M3 -Meningitis; 04 -Paralysis; 05,13 -CNS other unspec.; 07,49 -GI; 17,47 -Hepatic; 19 -CVS; 89 -Urinary; 06 -Skin/mucous.-CONTINUED

VIRUS OR VIRAL ANTIGEN	No-ill or data	Respir atory	Enceph alitis	Mening -itis	Para- lysis	CNS other unspec	GI	Hepa -tic	CVS	Urin -ary	Skin/ mucs memb
1101 POLIOVIRUS TYPE 1.....		1									
1102 POLIOVIRUS TYPE 2.....		1					1				
1103 POLIOVIRUS TYPE 3.....							2				
1104 POLIOVIRUS-VACCINAL STRAIN....							2				
1200 MUMPS VIRUS.....	2	2		8							
1300 HERPES VIRUS GROUP-NOT TYPED..			2								4
1301 HERPES SIMPLEX VIRUS NOT-TYPED	17	3	1			1				1	51
1302 EPSTEIN-BARR VIRUS (EB VIRUS).		1						1			
1303 VARICELLA-ZOSTER VIRUS.....	1										4
1306 HERPES SIMPLEX TYPE 1.....	1	1	1	1						3	18
1307 HERPES SIMPLEX TYPE 2.....	2										5
1401 COXIELLA BURNETI.....	6	4							1		
1514 MOLLUSCUM CONTAGIOSUM.....											2
1515 CONTAGIOUS PUSTULAR DERMATITIS (ORF VIRUS).....											1
1521 MEASLES VIRUS.....											2
1522 RUBELLA VIRUS.....	2										5
1532 HEPATITIS B ANTIGEN.....	38						1	39			
1535 HEPATITIS A ANTIBODY.....	2							32			
1541 CHLAMYDIA A - TRIC TYPE.....	48										
1556 CMV - CYTOMEGALOVIRUS.....	13	7			1	2				4	1
1563 CORONAVIRUS.....							1				
1564 ROTAVIRUS.....	1						17				
ASTROVIRUS							4				
Total.....	152	88	4	21	1	4	52	72	2	8	96

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

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81/2

PERIOD : 8/1/81 to 21/1/81 ...

Viral Identifications by Clinical Information Table 2.
Code 10 -Eye; 59 -Genital; 39 -Endo/sal gland;
38 -RES; 29 -Muscle/joint; 69 -Congenital; P8 -PUO;
G8 -Fever/malaise; 09 -Other; A1 -SIDS ...

VIRUS OR VIRAL ANTIGEN	Eye	Genital	Endo/sal gland	RES	Muscle/joint	Congenital	PUO	Fever/malaise	Other	SIDS
0100 ADENOVIRUS NOT TYPED	1								1	
0102 ADENOVIRUS TYPE 2	1						1		1	1
0103 ADENOVIRUS TYPE 3	2							1		
0107 ADENOVIRUS TYPE 7	1									
0119 ADENOVIRUS TYPE 19	1									
0203 INFLUENZA B VIRUS			1					3		
0500 RHINOVIRUS (ALL TYPES)			1							
0600 MYCOPLASMA PNEUMONIAE					1			7		
0700 ORNITHOSIS-PSITTACOSIS								1		
0809 COXSACKIEVIRUS A9							1	4		
0902 COXSACKIEVIRUS B2							1			
1006 ECHOVIRUS TYPE 6							1			
1014 ECHOVIRUS TYPE 14								1		1
1030 ECHOVIRUS TYPE 30								1		
1101 POLIOVIRUS TYPE 1										2
1102 POLIOVIRUS TYPE 2										1
1103 POLIOVIRUS TYPE 3										3
1200 MUMPS VIRUS			8					1		
1301 HERPES SIMPLEX VIRUS NOT-TYPED	2	1							1	
1302 EPSTEIN-BARR VIRUS (EB VIRUS)				3						
1306 HERPES SIMPLEX TYPE 1	5	6			1		2		2	
1307 HERPES SIMPLEX TYPE 2		98								
1401 COXIELLA BURNETI							13	17		
1522 RUBELLA VIRUS			1					1		
1532 HEPATITIS B ANTIGEN									2	
1541 CHLAMYDIA A - TRIC TYPE	1	24								
1556 CMV - CYTOMEGALOVIRUS			1	1		2	3	3	3	1
Total	14	129	12	4	2	2	22	40	10	9