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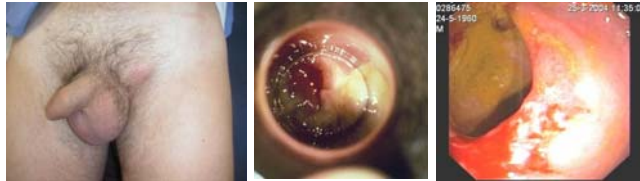
Introduction

LGV is a systemic disease caused by the *Chlamydia trachomatis* (CT) serovars L1, L2, L2a and L3.

Clinical manifestations:

- 1) an inguinal syndrome, with genital ulceration and inguinal lymphadenopathia (buboes) and subsequent suppuration, and
- 2) an anogenito-rectal syndrome, with procto-colitis and hyperplasia of intestinal and perirectal lymphatic tissue.

In 2003-2004 an unusual outbreak of LGV was identified in Rotterdam among MSM with proctitis and constipation.



1: Inguinal bubo

2: Purulent proctitis

3: Ulcerative colitis

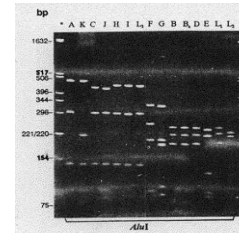
Study design

symptomatic (n=42 pt) when mucopurulent anal discharge and/or bloody ulcerative rectal processes were reported by the examining MD

asymptomatic (n=30 pt), in the absence of clinical inflammatory signs of the rectal mucosa.

The presence of LGV was determined by two methods:

1. RFLP of the *omp* – 1 gene¹
2. Sequencing²

PCR based *omp* 1 – RFLP genotyping

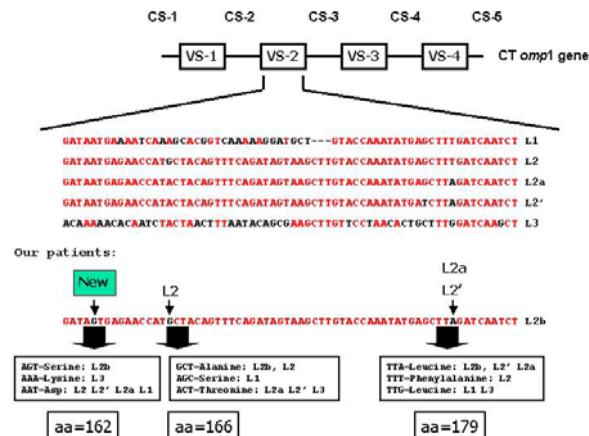
Aim

Did LGV proctitis already occur in 2002/2003 in A'dam?
If so, what are the characteristics of the LGV patients?

Study design

We selected 82 samples of 72 different MSM presenting at our STD clinic (\pm 20.000 new consultations/year) in 2002 and 2003 with CT proctitis based on a positive PCR test (COBAS AMPLICOR) from Roche (Hoffman – La Roche, Basel, Switzerland). Upon proctoscopic examination patients were designated into two groups:

Results



Results

	<u>LGV: L2b</u>	<u>non-LGV</u>
• symptomatic		
Nr. of samples	39 (76%)	12
Anal discharge reported	32	4
Genital ulcers	4 (L2b)	1 (<i>T.pall.</i>)
Lymph. pathy	5	0
Known HIV+	31	6

	<u>LGV: L2b</u>	<u>non-LGV</u>
• asymptomatic		
Nr. of samples	13 (41%)	18
Anal discharge reported	10	5
Genital ulcers	0	1 (HSV1)
Lymph. pathy	2	2
Known HIV+	7	7

Conclusions

- both symp. and asympt. mostly HIV positive MSM are infected
- caused by a newly identified L2b variant
- LGV outbreak expands beyond Rotterdam and can be traced back to at least 2002

References

- ¹ J Clin Microb 1993 31(5), 1060-1065
- ² J Clin Microb 1998 36(2), 345-351