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Effects of 24-hour unilateral ureteral obstruction on glomerular hemodynamics in rat kidney

ANTONIO DAL CANTON, ARIBERTO CORRADI, RODOLFO STANZIALE, GIANFRANCO MARUCCIO, and LUIGI MIGONE

Seconda Clinica Medica, University of Parma, Parma, Italy

Effects of 24-hour unilateral ureteral obstruction on glomerular hemodynamics in rat kidney. Glomerular hemodynamics were studied, by micropuncture, in Munich-Wistar rats submitted to 24-hour unilateral ureteral ligation (UUL). Glomerular capillary pressure (P_G), intratubular pressure (P_T) and pressure in the first-order peritubular capillaries (EAP) were measured with a servo-nulling device. Single nephron filtration fraction (SNFF) was calculated from arterial and peritubular blood protein concentration. SNGFR was both measured by conventional micropuncture techniques and calculated from efferent arteriole blood flow (EABF) and SNFF. Afferent arteriole blood flow (AABF) and resistance of afferent (R_a) and efferent (R_e) arterioles were calculated. Measurements were repeated 1 to 2 hours after the release of the ureter. Sham-operated rats were used as control. UUL caused a marked increase in R_a (from 4.9 ± [SD] 2.4 to 12.7 ± 5.1 dynes/sec/cm^{-5}). The fall in SNGFR (from 111.9 ± [SD] 23.9 to 34.4 ± 23.1 nl/min/kg body wt) was secondary to a decrease in both P_G and AABF. A further increase in R_a (16.0 ± 6.7 dynes·sec·cm^{-5}) occurred after releasing the ureter. SNGFR, however, was unaltered (33.7 ± 16.6 nl/min/kg body wt) since P_G decreased parallel to P_T, but AABF did not significantly change. *Conclusion.* Ureteral obstruction determines, in 24 hours, a marked cortical ischemia that is not promptly reversed by ureteral release.

Effets d'une obstruction urétérale unilatérale sur l'hémodynamique glomérulaire dans le rein de rat. L'hémodynamique glomérulaire a été étudiée par microponctions, chez des rats Munich-Wistar soumis à 24 heures de ligature unilatérale de l'uretère (UUL). La pression capillaire glomérulaire (P_G), la pression intratubulaire (P_T), et la pression dans les capillaires péritubulaires de premier ordre (EAP) ont été mesurées au moyen d'un dispositif à zéro asservi. La fraction de filtration des néphrons individuels (SNFF) a été calculée à partir des concentrations de protéines dans le sang artériel et péritubulaire. SNGFR a été mesuré à la fois par les techniques usuelles de microponction et calculé à partir du débit artériolaire efférent (EABF) et de SNFF. Le débit artériolaire afférent (AABF) et les résistances des artérioles afférente (R_a) et efférente (R_e ont été calculés. Les mesures ont été répétées une à deux heures après la libération de l'uretère. Des rats ayant subi un simulacre d'opération ont été utilisés comme contrôles. UUL a déterminé une augmentation importante de R_a (de 4,9 ± [SD] 2,4 à 12,7 ± 5,1 dynes/sec/cm^{-5}). La diminution de SNGFR (de 119,9 ± [SD] 23,9 à 34,4 ± 23,1 nl/min/kg poids corporel) a été secondaire à la diminution à la fois de P_G et de AABF. Une augmentation supplémentaire de R_a (16,0 ± 6,7 dynes·sec·cm^{-5}) est survenue après la libération de l'uretère. SNGFR, toutefois, n'a pas été affecté (33,7 ± 16,6 nl/min/kg poids corporel) du fait que P_G a diminué parallèlement à P_T alors que AABF n'a pas changé significativement. En conclusion l'obstruction urétérale détermine, en 24 heures, une ischémie corticale importante qui n'est pas rapidement réversible après la libération de l'uretère.

Soon after ureteral obstruction, afferent arterioles dilate, raising both plasma flow and hemodynamic pressure in glomerular capillaries, so that GFR is almost normally maintained in spite of a marked increase in intratubular pressure. Lessening of the obstruction is followed rapidly by restoration of preobstructive conditions [1].

On the contrary, that afferent arterioles constrict 24 hours after ureteral ligation was suggested [2, 3]. No measurement, however, of GFR in the whole kidney nor in the single nephron (SNGFR) has been reported as yet in this condition. More extensive studies were carried out after the release of the ureter, showing a decrease in filtration rate as well as in renal plasma and blood flow [4–10]. The details of glomerular dynamics were not clarified, however, authors' efforts being mainly directed toward elucidation of tubular function(s).

In this work our attention was focused on the glomerulus, with the purpose of contributing to a more comprehensive knowledge of renal function, both during and after a sustained ureteral obstruction.

Methods

The experiments were carried out in a mutant strain of Munich-Wistar rats having glomeruli on the kidney surface [11]. Eighteen nonfasted rats,

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each weighing 160 to 290 g, were lightly anesthetized with ether. The left ureter was ligated in 9 rats, about 1 cm above the bladder, and the other 9 were sham-operated and used for control. Twenty hours after these preliminary procedures, the rats were again anesthetized with sodium pentobarbital (Nembutal, 60 mg/kg body weight, i.p.) and were prepared for micropuncture as previously described [12]. After the surgical preparation, an i.v. infusion of bicarbonate-saline solution (sodium chloride, 110 mEq/liter; sodium bicarbonate, 28 mEq/liter; potassium chloride, 5 mEq/liter) containing inulin (5%) was begun at an infusion rate of 0.02 ml/min, and was maintained at that rate thereafter. One hour was allowed for equilibration before micropuncture measurements were started.

Ureteral obstruction studies. In 9 rats with 20-hour unilateral ureteral ligation (UUL), glomerular capillary pressure (P_G), intratubular pressure (P_T), and pressure in first-order peritubular capillaries (EAP) were measured with a servo nulling device (Instrumentation for Physiology and Medicine, San Diego, California) and were recorded simultaneously with arterial blood pressure (BP) on a dual channel recorder (Hewlett-Packard, model 7702 B). Timed, complete collections of tubular fluid were performed together with arterial blood samples from a femoral artery for SNGFR measurement [11]. After the insertion of the pipette tip into the tubule, an oil block (3 to 4 tubular diameters) was injected, and collection was started by gentle aspiration and spontaneously continued thereafter. Generally, collection was very slow, 5 to 15 min being required to obtain adequate volumes of tubular fluid. Moreover, the oil block sometimes did not move downstream in the tubule, so that collection of fluid was impossible. The number of collections (and of SNGFR measurements) being limited by these difficulties, SNGFR was calculated also from the efferent arteriole plasma flow (EAPF) and single nephron filtration fraction (SNFF) according to the equation

$$\text{SNGFR} = \frac{\text{EAPF}}{1 - \text{SNFF}} - \text{EAPF} \qquad (1)$$

This method, recently validated in our laboratory [1], does not require tubular collection, efferent arteriole blood flow (EABF) (from which EAPF is calculated) being measured by timed complete collection of blood from the efferent arteriole at its welling point on the kidney surface, and SNFF being calculated from protein concentration in blood samples collected from the efferent arteriole and from a femoral artery.

Postobstructive studies. In 6 rats, when the measurements of the obstructive period were completed, the left ureter was clamped just below the pelvis and was cannulated proximally to the ligation with a PE-50 tubing connected to a pressure transducer. The clamp was then released and ureteral pressure (P_U) recorded. The obstruction was relieved by cutting the PE-50 catheter used for P_U measurement. One hour was allowed before starting the following measurements: *(a) GFRs and sodium excretion rates ($U_{Na}V$) of both kidneys.* Urine was collected under mineral oil from the left ureter and from the bladder (for the right kidney) through PE-50 catheters. Arterial blood samples were obtained from the femoral artery at the beginning and at the end of clearance periods. *(b) Micropuncture measurements.* Timed, complete collections of tubular fluid and of peritubular blood were carried out, and micropressures were recorded, as previously described.

Control studies. Measurements, as described above, were performed also on 9 sham-operated rats, in whom the left ureter was handled in the same way as in the experimental group, except for ligation.

Analytical determinations. Urine volume was obtained by weight. The volumes of fluid and blood collected from proximal tubules and efferent arterioles, respectively, were estimated from the length of the fluid column in a calibrated constant-bore quartz tubing of approximately 100 μ I.D. (Friedrick and Dimmock, Millville, New Jersey, USA). The concentration of chemical inulin in tubular fluid was measured by the microfluorescence method of Vurek and Pegram; chemical inulin concentrations in plasma and urine were determined by the diphenylamine method; plasma protein concentration was measured by Lowry's method; a microadaptation of the same method was used to measure protein concentration in blood collected from efferent arterioles; sodium concentration in urine was measured by flame photometry [11].

Calculations. SNGFR was calculated from Equation 1 or measured according to

$$\text{SNGFR} = (\text{TF/P})\text{InV} \qquad (2)$$

where (TF/P)InV refers to tubular fluid/plasma inulin concentration ratio and V to tubular flow rate. SNFF was calculated as

$$\text{SNFF} = 1 - P_a/P_e \qquad (3)$$

where P_a and P_e equal protein concentration in blood samples collected from the femoral artery and from efferent arterioles respectively. Efferent arteriole blood flow (EABF) was obtained by timed complete collection of blood according to

$$EABF = \frac{\text{total collected blood (nl)}}{\text{collection time (min)}} \quad (4)$$

The microhematocrit in the efferent arteriole of superficial nephrons (Hct_e) was calculated according to the expression

$$Hct_e = \frac{1}{1 + (1 - SNFF) \times \left[\frac{1}{Hct_a} - 1\right]} \quad (5)$$

where Hct_a was the hematocrit measured in blood samples collected from the femoral artery. Efferent arteriole plasma flow (EAPF) was calculated according to the expression

$$EAPF = EABF\ (1 - Hct_e) \quad (6)$$

Afferent arteriole blood flow (AABF), that is, the initial blood flow rate per glomerulus, was calculated as

$$AABF = EABF + SNGFR \quad (7)$$

Afferent arteriole plasma flow (AAPF) or glomerular plasma flow (GPF) was derived from the expression

$$AAPF = GPF = AABF\ (1 - Hct_a) \quad (8)$$

The hydrostatic pressure gradient across the glomerular capillary (ΔP_G) was defined as

$$\Delta P_G = P_G - P_T \quad (9)$$

Oncotic pressure at the afferent end (π_a) and at the efferent end of the glomerulus (π_e) was derived from P_a and P_e, respectively, according to the equation of Landis and Pappenheimer [11]. Effective filtration pressure at the afferent end of the glomerulus (EFP_a) was calculated according to the expression

$$EFP_a = P_G - P_T - \pi_a \quad (10)$$

Vascular resistance across single afferent arteriole (R_a) was calculated from systemic blood pressure (BP), P_G, and AABF as

$$R_a = (BP - P_G)/AABF \times 7.962 \times 10^{10} \quad (11)$$

whereas efferent arteriolar resistance (R_e) was calculated as

$$R_e = (P_G - EAP)/EABF \times 7.962 \times 10^{10} \quad (12)$$

(the factor 7.962×10^{10} was used to express R in units of dynes · sec · cm^{-5}, when pressures were expressed in mm Hg and flows in nl/min). Total arteriolar resistance (R_{TA}) was obtained by the sum

$$R_{TA} = R_a + R_e \quad (13)$$

Transtubular pressure gradient (ΔP_T) was calculated as

$$\Delta P_T = (P_T + \pi_e) - EAP \quad (14)$$

Results

Ureteral obstruction studies. The results of these studies are summarized in Table 1. P_G fell from 44.8 ± (SD) 3.6 to 36.3 ± 4.1 mm Hg ($P < 0.001$). P_T and π_a being unchanged, the decrease in P_G was responsible for the reduction in both ΔP_G (from 31.4 ± 4.6 to 20.7 ± 3.3 mm Hg, $P < 0.001$) and EFP_a (from 17.2 ± 5.6 to 7.8 ± 3.4 mm Hg, $P < 0.001$). A marked increase in R_a (from 4.9 ± 2.4 to 12.7 ± 5.1 dyne · sec · cm^{-5}) determined a fall in both AABF (from 643.5 ± 241.3 to 241.2 ± 50.5 nl/min/kg body wt) and AAPF. Hence SNGFR was also markedly lowered, from 111.9 ± 23.9 to 34.4 ± 23.1 nl/min/kg body wt[1] ($P < 0.001$).

Ureteral obstruction caused a rise in R_e and a decrease in both EAP and EABF. At the efferent end of the glomerulus, glomerular pressure equilibrium was taking place in control conditions and was maintained also during UUL, the ratio $\pi_e/\Delta P_G$ not being different from 1 ($P > 0.1$).

P_U rose from 5.1 ± 2.2 to 15.4 ± 4.1 mm Hg ($P < 0.005$), the latter value not being statistically different from P_T, during UUL. Finally, no difference was found in SNFF and ΔP_T, even though P_e and π_e were moderately lower during obstruction.

Postobstructive studies. The results of the postobstructive studies are summarized in Tables 2 and 3. A further reduction of P_G was caused by the release of the ureter. P_T, however, decreased parallel to P_G, leaving unaltered both ΔP_G and EFP_a. Even though R_a increased from 10.6 ± 2.2 to 16.0 ± 6.7 dyne · sec · cm^{-5}, a value bordering statistical significance ($0.01 < P < 0.05$), AAPF was maintained, measuring 142.5 ± 21.7 during UUL vs. 122.2 ±

[1]Calculation of SNGFR from Equation 1 gave a mean value of 36.6 nl/min/kg body wt ($N = 32$), whereas measurement of SNGFR from Equation 2 gave a mean value of 31.7 nl/min/kg body wt ($N = 25$). These results were similar. Therefore, the values of SNGFR derived from the two methods were cumulated, to calculate the final mean.

Table 1. Pressures, flows, protein concentrations, hematocrits, and resistances in control conditions and after 24 hours of ureteral obstruction[a]

	Body wt	BP	P_G	P_T	EAP	ΔP_G	AABF	AAPF	EABF	EAPF	SNGFR
	g	*mm Hg*					*nl/min/kg body wt*				
Control	192.0 ±44.9	109.3 ± 8.5	44.8 ±3.6	13.4 ±1.7	17.1 ±2.8	31.4 ±4.6	643.5 ±241.3	329.3 ±126.1	531.5 ±228.2	218.0 ±112.9	111.9 ±23.9
Ureteral obstruction	230.0 ±37.4	118.1 ±10.2	36.3 ±4.1	15.6 ±3.4	10.2 ±2.3	20.7 ±3.3	241.2 ± 50.5	129.7 ± 28.9	206.6 ± 49.8	95.3 ± 26.0	34.4 ±23.1
P	>0.05	>0.05	<0.001	>0.05	<0.001	<0.001	<0.001	<0.001	<0.001	< 0.01	<0.001

[a] Abbreviations used are: BP, arterial blood pressure; P_G, glomerular capillary pressure (hemodynamic); P_T, intratubular pressure; EAP, pressure in the first-order peritubular capillaries; ΔP_G, hydrostatic pressure gradient across glomerular capillaries; SNGFR, single nephron glomerular filtration rate; AABF, afferent arteriole blood flow; AAPF, afferent arteriole plasma flow; EABF, efferent arteriole blood flow; EAPF, efferent arteriole plasma flow; P_a, systemic arterial protein concentration; P_e, efferent (peritubular capillary) protein concentration; π_a and π_e oncotic pressure in afferent and efferent arteriole, respectively; EFP_a, effective filtration pressure at the afferent end of the glomerulus; R_a and R_e, resistance of single afferent and efferent arterioles, respectively; SNFF, single nephron filtration fraction; Hct_a and Hct_e, hematocrit in afferent and efferent arteriole, respectively. All values are mean ± SD. The results are given as mean values of the averaged measurements in single rats.

Table 2. Summary of pressures, flows, and resistances during ureteral obstruction and in postobstructive period[a]

	P_G	P_T	ΔP_G	EFP_a	SNGFR	AAPF	SNFF	π_e	ΔP_T	R_a	R_e
	mm Hg				*nl/min/kg body wt*			*mm Hg*		*dynes · sec · cm*$^{-5}$	
Obstructive period	35.2 ±4.5	13.6 ±1.9	21.5 ±3.2	8.3 ±2.0	41.5 ±25.8	142.5 ±21.7	0.29 ±0.17	21.8 ±6.7	26.1 ±6.9	10.6 ±2.2	4.1 ±1.4
Postobstructive period	28.2 ±5.4	9.5 ±3.4	18.7 ±2.8	7.8 ±1.9	33.7 ±16.6	122.2 ±48.2	0.28 ±0.12	17.8 ±2.9	20.6 ±5.8	16.0 ±6.7	4.7 ±2.4
P	<0.01	<0.01	>0.05	>0.1	>0.2	>0.1	>0.1	>0.05	>0.05	<0.05	>0.1

[a] Abbreviations are defined in Table 1. All values are mean ± SD. The results are given as the mean values of the averaged measurements in single rats.

Table 3. Urine flow, sodium excretion, and inulin clearance from the right and the left (previously obstructed) kidney in postobstructive period[a]

	Inulin clearance *ml/min*	V *μl/min*	$U_{Na}V$ *μEq/min*
Right kidney	0.910 ±0.125	3.82 ±1.26	0.72 ±0.11
Left kidney	0.155 ±0.092	2.94 ±1.12	0.59 ±0.14
P	<0.001	<0.05	>0.05

[a] Abbreviations used are: V, urine flow rate; $U_{Na}V$, urine sodium excretion. All values are mean ± SD.

48.2 nl/min/kg body wt in the postobstructive condition (Table 2).

After releasing the ureter, GFR was markedly lower in the obstructed (left) kidney than it was in the contralateral one. This striking reduction in GFR (−83%) was accompanied only by a moderate decrease in urinary volume (−23%), the slight decrease in sodium excretion rate being not statistically significant (Table 3).

Discussion

Previous studies have given some evidence that renal vasoconstriction takes place in response to prolonged ureteral obstruction and is maintained after the release of the ureter. This site of vasoconstriction was placed by some authors at the afferent arteriole, because of a decrease in stop-flow intratubular pressure [2, 3]. The results of our study clearly demonstrate that 20 hours after complete ureteral ligation an increase in afferent arteriole resistance takes place, accounted for by a constriction of the afferent arterioles. According to Poiseuille's law, in fact, the resistance of a vessel is directly related to blood viscosity and to the length of the vessel and is inversely related to the fourth power of its radius. Although blood is a non-Newtonian fluid, its viscosity remains constant at physiologic flow rates for a given hematocrit and protein concentration [13]. Thus, since the latter values were unchanged during ureteral obstruction, and assuming also that the length of the renal arterioles was not modified, the increase in R_a is accounted for by a reduction of the radius of the afferent arteriole, probably caused by a contraction of the muscle fibers of the wall. During ureteral obstruction, a rise was observed also in efferent arteriole resistance, although of minor extent. This cannot be fully explained by an increase in the muscular activity of

Table 1. (*continued*)

P_a	π_a	P_e	π_e	EFP_a		R_a	R_e		Hct_a	Hct_e	ΔP_T
g/100 ml	*mm Hg*	*g/100 ml*	*mm Hg*		$\pi_e/\Delta P_G$	*dynes · sec · cm*$^{-5}$		SNFF	*volume fraction*		*mm Hg*
4.56	13.7	7.50	29.4	17.2	0.95	4.98	2.73	0.37	0.485	0.604	24.5
±0.22	±0.9	±1.53	±9.5	±5.6	±0.34	±2.39	±1.35	±0.11	±0.03	±0.05	±9.9
4.37	13.1	6.01	20.7	7.8	1.02	12.70	4.68	0.26	0.460	0.539	25.9
±0.74	±2.7	±1.10	±5.7	±3.4	±0.29	±5.10	±1.50	±0.14	±0.04	±0.05	±7.1
>0.1	>0.1	<0.05	<0.05	<0.001	>0.1	<0.001	<0.025	>0.05	≤0.1	<0.025	>0.1

the wall, since anatomic studies, as well as physiologic data, have given evidence that the efferent arterioles of superficial nephrons behave as thin-walled venules [12, 14–17]. On the other hand, glomerular capillary pressure, that is, the hemodynamic pressure of blood entering efferent arterioles, was markedly reduced during ureteral obstruction, whereas intratubular pressure, on which interstitial pressure mainly depends, was unchanged; therefore, we can assume that the transmural pressure gradient was reduced and, according to Laplace's law, that it contributed to decrease the radius, that is, to raise the resistance of efferent arterioles.

SNGFR was markedly lowered during ureteral obstruction. This is accounted for, in part, by the decrease in P_G, and consequently, in effective filtration pressure. Another main factor responsible for the reduction in glomerular filtration was the fall in glomerular plasma flow, the importance of the latter being increased by the occurrence of filtration pressure equilibrium [18, 19]. Since filtration pressure equilibrium was maintained before and after relief of obstruction, we cannot state whether any change in glomerular ultrafiltration coefficient plays a role in the decreased GFR [20].

The release of the ureter was followed by a further increase in afferent arteriole resistance, causing a reduction in glomerular capillary pressure. This, however, was compensated by a decrease in intratubular pressure, so that ΔP_G and EFP_a remained unchanged. The other major mediator of change in nephron filtration rate, that is, glomerular plasma flow, was unmodified too, allowing SNGFR to be maintained at previous (obstructive) levels.

No further modification in R_e was taking place in the postobstructive phase—according to the concept that efferent arterioles are almost devoid of muscular activity—and because the transmural pressure gradient was unaltered, since both P_T and P_G were decreasing in a parallel way.

A much greater decrease in GFR than in urine flow rate and sodium excretion was observed after ureteral release. As the reduction in GFR overcame that in SNGFR, a redistribution to superficial nephrons, having short, salt-losing Henle's loops, may have contributed to decrease fractional sodium and water reabsorption. Proximal tubular reabsorption was not measured in our micropuncture experiments. The transtubular pressure gradient, however, was not different from control conditions. Furthermore, others have given evidence that after 24 hours of ureteral obstruction, proximal fractional reabsorption, measured by micropuncture technique, was increased rather than reduced [6]. We may assume, therefore, that in our experiments the fractional sodium and water reabsorption was decreased at more distal tubular sites.

Our study does not clarify the mechanism(s) responsible for vasoconstriction occurring after ureteral obstruction. Some persistent decrease in cortical blood flow can be determined by surgical handling of the ureter [21]. These alterations, however, are admittedly of minor importance and cannot justify the striking increase in arteriolar resistance taking place in rats with ureteral ligation, as compared to sham-operated controls.

The increase in some peripheral vasoconstrictor substance can be excluded as well, the control (the right kidney) being not affected by contralateral ureteral obstruction. On the other hand, a reduction in stop-flow intratubular pressure was demonstrated in single tubules blocked with mineral oil for 24 hours, suggesting that the response to tubular obstruction occurs on an individual nephron basis [3].

Single nephron hemodynamics seems to be regulated by at least two hormonal systems having antagonist effects: the renin-angiotensin system and the renal prostaglandins.

According to the tubuloglomerular feedback hypothesis, an increased sodium load to the macula densa results in local renin release and afferent arteriole constriction; on the contrary, reduction of distal sodium delivery below normal values does not

measurably alter glomerular hemodynamics [22]. The latter situation was probably taking place in our experiments, in which GFR was markedly reduced and a decrease in proximal fractional reabsorption could be reasonably excluded, as previously discussed. Therefore, it is logical to conclude that the tubuloglomerular feedback was inactive during ureteral obstruction.

The renal prostaglandins are synthetized in the medullary interstitium, and there is evidence that they enter Henle's loop and move via the distal tubular fluid to the cortex, where they exert vasodilating effects [23, 24]. A reduction in distal tubular flow rate may occur relatively early after ureteral obstruction and lead to diminished prostaglandin delivery and prevalence of normal vasoconstrictive stimuli. A decrease in glomerular filtration would occur, further reducing tubular flow rate and creating a self-perpetuating vasoconstrictor mechanism. This vicious circle is not interrupted by ureteral release, whose primary effect is to allow emptying of hydronephrotic urinary tract and to reduce intrapelvic, intratubular, and interstitial pressure. A clue to how these changes might affect afferent arteriole resistance comes from recent experiments giving evidence that the sensitivity of the tubuloglomerular feedback mechanism is variable in response to changes in interstitial pressure, with decreases in the latter raising feedback responsiveness [25]. As a mere hypothesis, a decrease in interstitial pressure, taking place after release of the ureter, might lower the tubular threshold, above which a feedback vasoconstrictive response is elicited, increasing afferent arteriole resistance.

Reprint requests to Dr. A. Dal Canton, Divisione di Nefrologia Medica, Seconda Facoltà di Medicina Università, via S. Pansini, 80131 Napoli, Italia

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