

Ödem

Klassifikation nach ICD-10

R60	Ödem
I50.1	Linksherzinsuffizienz Akutes Lungenödem
G93.6	Hirnödem (exkl. Geburtsverletzung und traumatisch)

ICD-10 online (WHO-Version 2006) ^[1]

Das **Ödem** (v. griechisch οἰδημα, *oidema*, „Schwellung“) oder die „Wassersucht“ ist eine Schwellung des Gewebes aufgrund einer Einlagerung von Flüssigkeit aus dem Gefäßsystem.

Ist das Gleichgewicht zwischen Filtration einerseits und Resorption sowie Lymphabfluss andererseits zugunsten der Filtration verschoben, so bleibt vermehrt Flüssigkeit im Gewebe. Die Folge ist eine Wasseransammlung im Interstitium: ein Ödem.

Entstehung

Ödeme sind meist Folge einer zugrundeliegenden Erkrankung, also im engeren Sinne ein Symptom, z. B. bei einer Herz- oder Niereninsuffizienz, oder einer Leberzirrhose. Venöse Abflussstörungen können zu lokalisierten Ödemen führen, eine Beinvenen-Thrombose z. B. kann zu einer Schwellung des betroffenen Beins führen. Wird die Flüssigkeit im Gewebe nicht ausreichend über die Lymphbahnen abgeführt, so nennt man dieses ein Lymphödem. Weiterhin kommt es zum Ödem, wenn die Konzentration von Bluteiweißen (Albuminen) abnimmt (Hypoalbuminämie). Das geschieht z. B. beim Hungerödem (zu geringe Eiweißzufuhr) und beim nephrotischen Syndrom, das durch hohe Eiweißverluste über die Niere gekennzeichnet ist. Die austretende Flüssigkeit ist hierbei eiweißarm und wird Transsudat genannt.

Durch entzündliche oder allergische Prozesse kann es zu einer erhöhten Durchlässigkeit der Kapillaren kommen. Die hierbei ins Gewebe austretende Flüssigkeit ist eiweißreich und wird Exsudat genannt.

Auch Medikamente (z. B. Kortison, Antidepressiva oder Calciumantagonisten) können zur Ödementstehung beitragen. Ödeme können auch hormonell verursacht sein.



Lidödem

Pathophysiologie

Zwei grundlegende Schritte sind Voraussetzung zur Entstehung von Ödemen:

- Eine Änderung der Strömungsmechanik des Blutes in den Kapillargefäßen, welche den Übertritt von Flüssigkeit aus dem Gefäß in das umliegende Gewebe (Interstitium) begünstigt.
- Die Retention (verminderte Ausscheidung) von Natrium und Wasser durch die Nieren^[2].

Abgesehen von lokalen Ödemen wird ein Ödem erst sichtbar, wenn mindestens 2,5 bis 3 l Flüssigkeit in das Gewebe übergetreten sind. Da das Plasmavolumen normalerweise ca. 3,5 l beträgt, würde ein Ödem zum Volumenmangelschock führen, wenn die verloren gegangene Flüssigkeit nicht ersetzt würde. Der Verlust von Flüssigkeit in das Gewebe führt zu einer verminderten Füllung des arteriellen Gefäßsystems, welches jedoch nur etwa 15 % des gesamten Blutvolumens enthält^[3]. Das Gesamtblutvolumen kann daher beim Ödem durchaus erhöht sein, wenn davon nur das venöse Blutvolumen betroffen ist. Auch eine verminderte Herzleistung, wie z. B. bei einer Herzinsuffizienz, oder eine Erweiterung der Blutgefäße (Vasodilatation), wie z. B. bei einer Leberzirrhose oder in der Schwangerschaft, kann zu einer verminderten Füllung des arteriellen Gefäßsystems führen. Diese wird von arteriellen Dehnungsrezeptoren in Nierenkörperchen, Aortenbogen und Halsschlagader (Arteria carotis communis) registriert. Die Folge ist eine Aktivierung des Renin-Angiotensin-Aldosteron-System in der Niere, eine Stimulation des sympathischen Nervensystems und die Freisetzung von Vasopressin im Hypothalamus. Letztendlich führt dies in den Nierenkanälchen zur vermehrten Rückresorption von Wasser und Natrium.

Im Fall der Herzinsuffizienz kommt es zwar zur Überfüllung des venösen Systems, die von Dehnungsrezeptoren im linken Herzvorhof registriert wird, was zur Freisetzung von atrialem natriuretischen Peptid und damit eigentlich zu vermehrter Natrium- und Wasserausscheidung führt. Die Aktivierung der arteriellen Dehnungsrezeptoren überwiegt aber, so dass im Endeffekt Natrium und Wasser eingelagert werden.

Diagnose

Anamnese (Krankenvorgeschichte)

Bei der Erhebung der Krankenvorgeschichte (Anamnese) wird nach Vorerkrankungen (z. B. Koronare Herzkrankheit, Bluthochdruck, Alkoholmissbrauch) und Medikamenten gefragt, die zu Herz-, Nieren- oder Leberkrankheiten führen können.

Die Lokalisation der Wassereinlagerungen gibt weitere Hinweise: Bei Luftnot liegt möglicherweise ein Lungenödem bei Versagen der linken Herzkammer vor. Wassereinlagerungen im Bauch (Aszites) weisen auf eine Lebererkrankung hin. Schwellungen der Beine können durch Versagen der rechten Herzkammer, chronisches Nierenversagen, Venenerkrankungen oder Lymphabflussstörungen verursacht sein.

Wichtig ist auch der zeitliche Verlauf: Bei Frauen kann es im Rahmen des Menstruationszyklus zu periodischen Wassereinlagerungen kommen. Diese sollten jedoch nicht mit Diuretika behandelt werden, da durch die Behandlung die Symptome eher verschlimmert würden.

Körperliche Untersuchung

Beim Lungenödem sind beim Abhören mit dem Stethoskop (Auskultation) feuchte Rasselgeräusche zu hören. Zur sicheren Diagnose ist eine Lungen-Röntgen-Aufnahme erforderlich, da andere Lungenkrankheiten, wie z. B. eine Lungenembolie ähnliche Symptome verursachen können. Eine Schwellung des Leibes kann auf Wassereinlagerungen in der Leibeshöhle (Aszites) bei Leberzirrhose, nephrotischem Syndrom oder Rechtsherzversagen hinweisen. Beim Abklopfen ist ein gedämpfter Klopfeschall zu hören, unter Umständen kann beim Beklopfen der Bauchwand eine Flüssigkeitsschicht ausgelöst werden. Die Diagnose kann durch eine Ultraschalluntersuchung gesichert werden. Wassereinlagerungen in Bauchwand und Flanken (Anasarka) können bei schwerem Herzversagen oder fortgeschrittenem Nierenversagen auftreten. Eindrückbare Schwellungen der Beine

weisen auf venöse Abflussstörungen, eine Herzinsuffizienz oder eine Nierenerkrankung hin, eine einseitige Schwellung des Beines spricht dabei für eine Venenerkrankung. Nicht eindrückbare Schwellungen der Beine findet man dagegen bei Lymphabflussstörungen und beim Myxödem infolge einer Unterfunktion der Schilddrüse. Schwellungen der Augenlider können auf Eiweißverluste über die Nieren hinweisen^[4].

Labordiagnostik

Eine Erhöhung des Kreatinins im Serum weist auf eine Nierenerkrankung hin. Beträgt die Eiweißausscheidung im Urin über 2,5 g pro Tag, liegt am ehesten ein nephrotisches Syndrom vor.

Therapie

Behandelt wird in Abhängigkeit von der Ursache. Kardiale, renale und hepatische Ödeme werden mit Diuretika ausgeschwemmt. Meistens ist eine orale Gabe ausreichend, nur bei Resorptionsproblemen oder im Notfall (Lungenödem) wird eine intravenöse Gabe bevorzugt. Hierbei sollte eine Gewichtsreduktion nicht mehr als 1–1,5 kg betragen. Bei fehlender Ausscheidung bei Nierenversagen muss der Flüssigkeitshaushalt durch Dialyse kontrolliert werden. Lymphödeme werden im Allgemeinen mit Massagen (Lymphdrainage) behandelt, Ödeme auf dem Boden von Eiweißmangel durch Substitution. Allergische Prozesse werden symptomatisch mit Antihistaminika und Cortison behandelt.

Spezielle Ödemformen

- Angioödem
- Blutstauungsödem
- Dorsonuchales Ödem
- Hereditäres Angioödem (HAE)
- Hirnödem
- Höhenhirnödem (HACE), Höhenlungenödem (HAPE), siehe auch Todeszone
- Hungerödem oder Eiweißmangelödem
- Hydrops fetalis
- Hygroma colli
- Imbibition
- Knochenödem
- Nackentransparenz
- Nasopharynxödem
- Larynxödem
- Lungenödem
- Lymphödem
- Myxödem
- Makulaödem
- perifokales Ödem
- Reinke-Ödem
- Quincke-Ödem
- Schwangerschaftsödem

Siehe auch

- Lipödem (kein Ödem im Sinne der obigen Definition)

Referenzen

- [1] <http://www.dimdi.de/dynamic/de/klassi/diagnosen/icd10/htmlamtl2006/fr-icd.htm>
- [2] Burton D Rose: „*Pathophysiology and etiology of edema in adults.*“ UpToDate 2007; 15.2: S. Seiten Abstract (<http://patients.uptodate.com/topic.asp?file=fldlytes/17311>)
- [3] Robert W. Schrier: „*Decreased Effective Blood Volume in Edematous Disorders: What Does This Mean?*.“ J Am Soc Nephrol 2007; 18: S. 2028-2031 Abstract (http://www.ncbi.nlm.nih.gov/sites/entrez?cmd=retrieve&db=pubmed&list_uids=17568020&dopt=AbstractPlus)
- [4] Burton D Rose: „*Approach to the adult with edema.*“ UpToDate 2007; 15.2 Abstract (http://patients.uptodate.com/topic.asp?file=pc_neph/4943)

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